Encyclopedia of Drugs, Alcohol & Addictive Behavior

SECOND EDITION

Volume 1

Encyclopedia *of* Drugs, Alcohol & Addictive Behavior

Editorial Board

ENCYCLOPEDIA *of* DRUGS, ALCOHOL & ADDICTIVE BEHAVIOR

SECOND EDITION

VOLUME 1

A – D

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Preface

Why a second edition of the *Encyclopedia of Drugs and Alcohol*? And why change the name to the *Encyclopedia of Drugs, Alcohol, and Addictive Behavior*?

Consider a smattering of statistics:

- In 1999, 10.3 million people (4.7% of the American population) ages 12 years or older were dependent on illicit drugs or alcohol (National Household Survey on Drug Abuse, 1999)
- In 1998, it was estimated that approximately 19,000 alcohol-induced deaths occur annually in the United States (excluding those deaths due to motor vehicle accidents involving alcohol) (National Vital Statistics Reports, Murphy, 2000)
- In 1993, it was reported that 13,984 people were killed in alcohol-related motor vehicle accidents; 3,765 of these individuals had not been drinking themselves (Heien, 1996)
- In 1998, 16,926 deaths were attributed to drug-induced causes (National Vital Statistics Reports, Murphy, 2000)
- In 1998, chronic liver disease and cirrhosis ranked as the tenth leading cause of death in the United States (National Vital Statistics Reports, Murphy, 2000)
- Fetal alcohol syndrome affects one in every 600–750 births, and is currently the leading cause of mental retardation, beating out previous contenders such as Down syndrome and spina bifida (Abel, 1986)
- Economic costs to society related to alcohol and drug abuse were projected at nearly $246 million in 1992; projections for 1995 suggested that economic costs related to alcohol and drug abuse would reach over $276 million (National Institute on Drug Abuse and National Institute on Alcohol Abuse and Alcoholism)
- Smokers ages 18–25 have a fourfold increased likelihood of illicit drug use compared to their nonsmoking peers; smokers ages 12–17 have a sevenfold increased likelihood of illicit drug use compared to their nonsmoking peers (1999 National Household Survey on Drug Abuse)
- In 2000, the United States put together an emergency aid package to Colombia, totaling $1.3 billion, intended in part to address the issues of drug trafficking between Colombia and the United States (White House, Office of the Press Secretary, 2000)
- In 1999, the annual cost for problem and pathological gambling reached $5 billion, with lifetime costs projected at $40 billion associated with decreased productivity, social service costs, and creditor losses (Gerstein et al., 1999)

Drugs, alcohol, and addictive behaviors have enormous ramifications on a global scale, and include political, economic, legal, social, and public health issues, as well as family well-being and physical and psychological health. These ramifications can be studied as a macrocosm of the way in which the issues attendant to addictions affect every stratum of society, or collapsed down into the microcosm of misery visited on a single individual struggling to loosen the stranglehold of addiction.

The first edition of Macmillan's *Encyclopedia of Drugs and Alcohol* is an amazing compendium of

information on the effects of addictions at every level. Drs. Jaffe, Anthony, Johanson, Kuhar, Moore, and Sellers and their team of experts put together an intelligently organized, complete survey of drug and alcohol addictions. Our task, then, was to respectfully update these articles to reflect the impressive amount of research performed over the five years since the publication of the original edition.

The newly renamed second edition, now called the *Encyclopedia of Drugs, Alcohol, and Addictive Behavior*, retains the first edition's organizational format, but includes new and updated articles that address the exciting frontiers that have opened up in the field of addiction studies. New brain imaging techniques, an explosion of information about the human genome and its relevance to health, excellent efforts at data collection to define the scope and nature of the problem of addictions, and carefully designed studies aimed at uncovering the statistical relevance of various prevention, diagnosis, and treatment programs are all illuminating and igniting the process of understanding addictions. The encyclopedia's name change reflects acknowledgment that behaviors not involving chemical substances (such as pathological gambling) are being seriously and thoughtfully studied, and appear to involve some physiological and psychological pathways in common with those addictions involving chemical substances.

Far from being an esoteric consideration of theoretical concepts, research within the field of addiction studies quickly doubles back to benefit those individuals who are trapped in the mundane, nitty-gritty ugliness of an addiction. For this reason, and because issues within the field of addictions surely touch the lives of essentially every member of society, this second edition preserves the first edition's mission of providing information that will be useful, interesting, and accessible to nonspecialists seeking an understanding of addiction topics.

The second edition's editorial board (Drs. Kathleen Carroll, Jeffrey Fagan, Henry Kranzler, and Michael Kuhar) has been impressively dedicated in their efforts to produce this encyclopedia. Their expertise and guidance have been invaluable, as have the expertise and knowledge of the various writers who have contributed to this work. All of us who have worked on this encyclopedia have appreciated the forbearance of publisher Elly Dickason, senior editor Anne Davidson, and editor Amanda Quick. I wish to thank my husband, Toby, and my daughters, Anna, Emma, and Isabelle, for their patience as "the encyclopedia project" took its place in our family life.

Last, we wish to acknowledge with appreciation all of the researchers and academicians who continue to illuminate issues relevant to addiction, the professionals who work compassionately with those affected by addiction, and the many individuals who continue to reach for a life free of addiction's grip.

Rosalyn Carson-Dewitt, M.D.
Editor in Chief
October 2000

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AA *See* Alcoholics Anonymous; Treatment: Twelve Step Facilitation

ABRAXAS *See* Treatment Programs/Centers/Organizations: An Historical Perspective

ABSTINENCE *See* Addiction: Concepts and Definitions; Alcoholism; Sobriety; Withdrawal

ABSTINENCE VIOLATION EFFECT (AVE) The abstinence violation effect (AVE) occurs when an individual, having made a personal commitment to abstain from using a substance or to cease engaging in some other unwanted behavior, has an initial lapse whereby the substance or behavior is engaged in at least once. Some individuals may then proceed to uncontrolled use. The AVE occurs when the person attributes the cause of the initial lapse (the first violation of abstinence) to internal, stable, and global factors within (e.g., lack of willpower or the underlying addiction or disease).

In RELAPSE PREVENTION, the aim is to teach people how to minimize the size of the relapse (i.e., to counter the AVE) by directing attention to the more controllable external or situational factors that triggered the lapse (e.g., high-risk situations, coping skills, and outcome expectancies), so that the person can quickly return to the goal of abstinence and not "lose control" of the behavior. Specific intervention strategies include helping the person identify and cope with high-risk situations, eliminating myths regarding a drug's effects, managing lapses, and addressing misperceptions about the relapse process. Other more general strategies include helping the person develop positive addictions and employing stimulus-control and urge-management techniques. Researchers continue to evaluate the AVE and the efficacy of relapse prevention strategies.

(SEE ALSO: *Treatment*)

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ABUSE *See* Addiction: Concepts and Definitions

ABUSE LIABILITY OF THERAPEUTIC DRUGS: TESTING IN ANIMALS Determining the probability that a new drug will be abused is an important step in reducing the overall abuse of therapeutic drugs. Since the likelihood that a drug will be abused by a patient must be carefully weighed against the benefit provided by the drug, it is important that research outline any and all reinforcing effects a drug may have which could lead to subsequent abuse. Prediction of the likelihood of abuse has historically been based upon human experiments and observation. This method, however, is increasingly being replaced with experimentation on animals.

Research conducted since the early 1960s has shown that animals such as monkeys and rats will, with very few exceptions, repeatedly self-administer the same drugs that human beings are likely to abuse. Moreover, test animals do not self-administer drugs that human beings do not abuse.

Research based on animal testing is conducted in a slightly different manner and often requires laboratory procedures not needed for research based on human test subjects. Provisions must be made to allow the animal a means by which to self-administer the drug. Since animals frequently are not physically able to administer a drug in the same way a human would, alternate methods are employed. Animals may be taught to push levers or do similar actions in order to get a dose of a drug. The results of these drug self-administration studies in animals play a critical role in the prediction of the likelihood of abuse of new drugs in human beings.

The liability that a drug will be abused is often evaluated by what has been termed a "substitution procedure." Such research begins with the administration of a known drug, which is then substituted with a new drug under investigation. The first phase in the substitution procedure is designed to establish a baseline of how much effort an animal is willing to make to obtain a drug dose in general. Each day an animal is allowed to give itself a drug of known potential for abuse. The researcher notes how frequently the animal takes a dose and how much effort it is willing to make to get a dose of the drug. The researcher can make a lever harder to push, make the animal push it repeatedly, or make the animal follow a complicated set of actions to get a dose. This provides a baseline against which to compare the effects of the new drug which will be studied.

For example, a monkey may give itself COCAINE or CODEINE via intravenous injections during sessions that last several hours. When session-to-session intake of the known drug is stable (that is, stays about the same, thus showing the dosage which is sufficient to satisfy the animal and reduce its drive to obtain more), the liquid in which it was dissolved is substituted for the baseline drug for several consecutive sessions. Since this liquid is usually neutral, with no positive or negative effects, the animal gives itself fewer and fewer injections until it hardly bothers pushing the lever at all. The animal is briefly returned to baseline conditions, followed by a substitution period during which a dose of the test drug is made available. This continues for at least as many sessions as were required for the animal to stop bothering with pushing the lever for the neutral liquid. This process is repeated with different concentrations of the new drug until the experimenter has tested a range of possible doses of the new medicine.

The rates at which the animal gives itself the test drug, neutral liquid, and known addictive drug are then compared. A new drug that the animal prefers to the neutral liquid is considered to be a substance that reinforces the desire for itself (a "positive rein-

forcer") and would thus be predicted to have abuse liability.

Such substitution procedures provide information which indicates whether or not a drug is liable to be abused, but do not allow a comparative estimate as to whether or not a new drug is more addictive or less addictive than other known drugs. These procedures measure how frequently the animal gives itself a dose, a measure that reflects both the direct effects of the drug and the effects of the drug's reinforcement of the desire for itself. Another method must be used to measure the reinforcing effect of a drug separately from its other effects. To compare drugs, it is useful to know how big the maximum reinforcing effect is—termed its reinforcing efficacy. Several procedures have been developed to measure reinforcing efficacy. Most either allow an animal to choose between the new drug and another drug or non-drug reinforcer(choice procedures), or they measure how hard an animal will work to obtain an injection (progressive-ratio procedures).

In choice procedures, the measure of reinforcing efficacy is how often the new drug is chosen in preference to the other drug (or non-drug). In progressive-ratio procedures, the number of times the animal must push the lever in order to get a drug injection is increased until the animal no longer bothers to push the lever. At some point the animal determines that it is not worth the extra effort to get another dose. This point is called the break point and is a measure of the reinforcing efficacy of the drug.

The fact that animals given a choice between different strengths of the same drug show a propensity to choose the higher dose most often is evidence that these procedures provide a valid measure of reinforcing efficacy. In addition, break points are higher in progressive-ratio experiments involving higher stable doses and lower for experiments involving lower doses. Results of both the choice and the progressive-ratio procedures in animal research are consistent with what is known about abuse of drugs in human beings—that is, drugs such as COCAINE, a highly preferred drug in choice studies, maintain higher break points in progressive-ratio studies than other drugs, and are frequently abused.

These experiments show how animals discriminate among drugs, and the extent to which they prefer certain drugs over other drugs. The results may be used to predict potential subjective effects in human beings. Since subjective effects play a major role in drug abuse, such experiments are an important tool used in the evaluation of the likelihood of abuse in new drugs. A new drug with subjective effects similar to those of a known, addictive, and often abused drug is likely to be abused itself. Additionally, drug-discrimination experiments not only identify the potential for abuse but also provide important information which allows researchers to classify new drugs based on their predicted subjective effects, something that drug self-administration experiments cannot do. Thus, drug discrimination provides additional information relevant to the comparison between the new drug and drugs that we already know are addictive and frequently abused. For example, a monkey shows a similar discrimination pattern using a new drug as it has shown previously using a known drug such as cocaine. This new drug is likely to be abused and to have subjective effects similar to those produced by cocaine.

CONCLUSION

Researchers have improved methods for predicting the likelihood that a new drug will be abused. Using animals in substitution, choice, and progressive-ratio procedures has greatly enhanced researchers' understanding of factors involved in determining the liability that a new drug or chemical compound will be abused. Current research techniques allow the evaluation of likely preference and the reinforcing efficacy of a new compound based on experiments with animals such as monkeys and rats. This information is then used to reliably predict whether a drug is likely to be abused and to which known drugs it is likely to be similar, both in terms of how addictive it is and what its subjective effects will be. Such information is clearly valuable in deciding how much to restrict a new drug and is a critical tool in the effort to reduce the abuse of therapeutic drugs.

(SEE ALSO: *Abuse Liability of Drugs: Testing in Humans; Controlled Substances Act of 1970; Reinforcement; Research: Animal Model*)

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ABUSE LIABILITY OF DRUGS: TESTING IN HUMANS There is probably no drug used to treat illness that does not also pose certain risks. One such risk, generally limited to drugs that have actions on the central nervous system, is that the drug will be misused or abused because of these effects. Drugs such as these are said to have abuse potential or abuse liability. If the drugs have important therapeutic use, they may still be made available, but they will be subject to certain legal controls under various federal and state laws (see Controlled Substances Act). Over the past fifty years, a number of methods have been developed to test new drugs to determine their abuse liability, so that both the public and the medical profession can be warned about the need for appropriate caution when using certain drugs. These methods involve both testing in animals (preclinical) and testing in humans (clinical).

Several important reasons exist for why testing with humans is useful and necessary in the development of safer and more effective pharmacological agents. The research on laboratory animals demonstrating greater or lesser degrees of the abuse liability of drugs must be validated with humans; this reduces the likelihood of error in assessing potential risks. Moreover, certain self-reported changes associated with the subjective effects of medicinal drugs can be more readily evaluated in the humans for whom they were developed. Human clinical studies are also important in determining appropriate dose levels and dosage forms to ensure safety and efficacy while minimizing unwanted side effects. Finally, comprehensive and effective testing with humans helps to reduce the availability of abusable drugs to those who are likely to misuse them and to provide for the legitimate medical and scientific needs for such pharmacological agents.

HUMAN VOLUNTEER SELECTION

One of the most important factors in drug-abuse-liability testing with humans is the way the volunteer subjects are chosen to participate in the assessment procedures. In most studies, the human volunteer subjects are experienced drug users, but wide variations exist in the nature and extent of their drug use and abuse. Some studies, for example, use students and other volunteers whose misuse and abuse of drugs has been mostly "recreational"; other studies involve people with histories of more intensive drug use and abuse over extended periods. Also, the settings in which the tests are conducted vary widely, from residential laboratory environments, where the subjects live for several days or weeks at a time, to laboratories, where the subjects do not remain in residence but continue their daily routine after drug ingestion. Variations also occur in the age of the subjects tested and the time of day that the drug is administered. Often subjects have been selected for certain human drug-abuse-liability tests on the basis of some special features (e.g., anxiety levels, level of alcohol consumption) to determine the extent to which such factors influence the outcome of the tests.

Convincing evidence now exists that many of these factors—particularly the prior experience of the subject with respect to drug and alcohol use and misuse—play an important role in the assessment of abuse liability. The obvious value in using such subjects lies in the fact that these individuals are similar to those most likely to abuse drugs with abuse liability—for example, drug abusers who help determine whether a new drug has a greater or lesser chance for abuse than the one they already know. It is also important to carry out abuse-liability testing with people who, for example, do not usually abuse drugs but are light social drinkers—to assess the likelihood of abuse of certain generally available medications, such as sleeping pills or appetite suppressants.

DRUG COMBINATIONS

The prediction of a drug's abuse liability, based on a wide variety of testing procedures with humans, is further complicated by the fact that drugs of abuse are often used in combination and simultaneously with other pharmacological agents. This creates some very difficult problems for the testing

of abuse liability, because of the large number of possible drug combinations that need to be tested and their unknown, potentially toxic, effects. While it has long been known that drugs such as COCAINE and HEROIN or MARIJUANA and ALCOHOL are used simultaneously by drug abusers, few testing procedures have been developed for assessing their interactions. Even more puzzling is that some drugs with opposite effects (e.g., stimulants like the AMPHETAMINES and depressants like the BARBITURATES) are known to be used simultaneously by POLYDRUG ABUSERS, suggesting that unique subjective-effect changes may be important factors in such abuse patterns.

PRINCIPLES OF ABUSE-LIABILITY TESTING

Based on extensive research undertaken over the past several decades, some important general principles governing abuse-liability testing with humans have been established. In the first instance, for example, a meaningful assessment requires that the test drug be compared with a drug of known abuse liability to provide a standard for evaluation. Second, the assessment procedure must involve the indicated comparison over a range of doses of both the test drug and the standard drug of abuse. This permits both a quantitative and a qualitative comparison of the drugs, while guarding against the possibility of overlooking some unique high- or low-dose effects. Third, the testing procedures should include measures of drug effects in addition to those like drug taking in the lab, which directly predict the likelihood of abuse. With these additional measures, it is often possible to obtain reliable estimates of abuse liability by comparing test drugs with a drug of abuse across a range of effects as a standard for evaluation. Fourth, confidence in conclusions regarding the abuse liability of a drug compound can be enhanced by utilizing a multiplicity of measures and experimental procedures. This is the case because our present level of knowledge in this area does not permit a firm determination of the best or most valid predictor of the likelihood of abuse. Finally, a population of test subjects with histories of drug use appears to be the most appropriate selection for predicting the likelihood of abuse of a new test drug, since this is the population who might use such a drug in that way.

DEVELOPMENT OF ABUSE-LIABILITY TESTING PROCEDURES

The origins of assessing drug-abuse liability with humans can be found in some of the earliest writings of civilization, describing the subjective effects of naturally occurring substances, such as wine. Since the mid-nineteenth century, literary accounts of the use and misuse of opium, marijuana, and cocaine, among other substances, have emphasized their mood-altering effects and their potential for abuse. Only in recent years, however, have systematic methods for measuring such subjective effects been refined through the use of standardized questionnaires. Volunteers who are experienced drug users complete the questionnaires after they have taken a drug; their answers to the subjective-effects questions—how they feel, their likes and dislikes—readily distinguish between the various drugs and doses, as well as between drug presence or absence (i.e., placebo).

This basic subjective-effects methodology has been further refined in recent years by using a training procedure to ensure that the human volunteer can differentiate a given drug (e.g., morphine) from a placebo (i.e., nondrug). Then new drugs are tested to evaluate their similarity to the trained reference drug of abuse. This behavioral drug discrimination method permits the volunteer to compare a wide range of subjective and objective effects of abused drugs with those of new drugs. These highly reliable procedures have proven very useful in identifying drugs that may have abuse liability.

Among the most important factors in assessing abuse liability is the determination of whether humans will take the drug when it is offered to them and whether such drug taking is injurious to the individual or society. These cardinal signs of drug abuse have provided an important focus for laboratory animal self-injection experiments, but systematic studies in which humans self-administer drugs of abuse have been less common. Methods have been developed with humans, however, for comparing the behavioral and physiological changes produced by self-administration of a known drug of abuse with the changes produced by other self-administered drugs.

The measure that has proven most useful in this approach to human drug-abuse-liability assessment is the ability of a drug to reinforce and maintain self-administration behaviors much like

the behaviors used to obtain food and water. Such reinforcing effects of drugs are an important determinant influencing the likelihood that a particular drug will be abused. Laboratory studies with volunteers who are experienced drug users, for example, have shown that they will perform bicycle-riding exercises to obtain doses of abused drugs (e.g., pentobarbital). There is a systematic relationship between the amount of exercise performed and the amount of drug available (i.e., higher doses and shorter intervals between doses produce more exercise behavior than lower doses and longer interdose intervals). When a placebo or a drug that is not abused (e.g., Thorazine) is made available for bicycle riding, on the other hand, the rate of self-administration declines to near zero.

Differences between drugs in abuse liability can also be assessed by determining whether humans prefer one drug of abuse to another. During a training period, for example, experienced drug users sample coded capsules containing different drugs or different doses of a drug. Then, during subsequent test sessions, they are presented with the coded capsules and allowed to choose the one containing the drug or drug dose they prefer. This "blind" procedure (i.e., the volunteers are not told what drugs the capsules contain) prevents biases that might be introduced by using the drug names. When neither the volunteer subject nor the person conducting the test knows what drug the capsules contain, the procedure is referred to as "double blind."

Not surprisingly, it has also been shown that the preference for one drug over another or one drug dose over another agrees well with ratings of "drug liking" made independently of the choice tests just described. In self-administration studies in which volunteers show a preference for one drug of abuse over another, subjective ratings of "liking" and positive mood changes were clearly more frequent for the preferred drug than for the drug chosen less often. Such self-reports have inherent limitations, however, because of variations in individual verbal skills, which make it necessary to confirm such findings with other measures.

In addition to the self-administration and subjective-effects measures of obvious value in testing the abuse liability of drugs in humans, other quantitative drug-effect measurements have proven useful. When, for example, observer ratings (e.g., nurses watching the patients) and performance tests (e.g., speed of movement) are measured after different drugs are administered to volunteers, the results can be compared to determine whether the behavioral changes produced by a test drug are the same as or different from those of a known drug of abuse. When a number of different performance tests (e.g., arithmetic calculations, memory for numbers and letters, speed of reaction) are given following such drug administration, it is possible to construct a behavioral profile showing the performance effects of different drugs. Comparisons between different drugs and test drugs with regard to the similarity of such profiles across their respective dose ranges increase confidence in assessments made of the abuse liability of unknown drugs.

EFFECTIVENESS OF ABUSE-LIABILITY TESTING

Because of the availability of procedures for abuse-liability testing in humans, it seems reasonable to ask how well they work. That is, has it been possible to predict from the results of these tests whether a new drug will be abused when it becomes generally available? The two major sources available for checking the effectiveness of human abuse-liability testing procedures are case reports by clinicians of patient drug abuse, and EPIDEMIOLOGICAL surveys of large numbers of individuals as well as of specific target sites (e.g., hospital emergency rooms). Both of these approaches have many shortcomings, since they lack the precision and focus that human laboratory testing can provide. But despite the draw-backs, they can detect abuse-liability problems in both specific groups of individuals and the population at large, in a manner that has generally validated the results of human laboratory-testing procedures.

ETHICAL CONSIDERATIONS

A number of codes and regulations agreed on by scientists and the lay public provide norms for the conduct of research and testing with human volunteers. In general, they require a clear statement, understandable to the volunteer, of the risks and benefits of the testing procedure, as well as an explicit consent document in written form. After it is clear that the participant thoroughly appreciates all that is involved and the potential consequences of participation, the volunteer signs the consent

form in the presence of a witness who is not associated with the research. These required procedures ensure both the autonomy and the protection of volunteers for drug-abuse-liability testing.

(SEE ALSO: *Abuse Liability of Drugs: Testing in Animals; Research: Animal Model*)

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JOSEPH V. BRADY

ACCIDENTS AND INJURIES FROM ALCOHOL Trauma (bodily injury) is a major social and medical problem in both developed and developing countries. Injuries are among the leading cause of death and disability in the world, and affect all populations, regardless of age, sex, income, or geographic region. In 1998 about 5.8 million people died of injuries worldwide, and injuries caused 16 percent of the global burden of disease (Krug, et al, 2000). In developed countries injuries are the leading cause of death between the ages of one and forty, and in the U.S. population it is the fourth leading cause of death (exceeded only by heart disease, stroke, and cancer). Of all deaths from injury in the United States, about 65 percent are classified as unintentional (which excludes deaths from suicide, homicide, and other criminal offenses); of these, about half result from motor vehicle accidents. Trauma also accounts for high rates of morbidity (number of sick to well). In the United States, the rate of serious injury is estimated to be at least three hundred times the death rate.

The first documentation of alcohol's involvement in injury dates to 1500 B.C.E., with an Egyptian papyrus warning that excessive drinking leads to falls and broken bones. The scientific study of alcohol and injuries has been the subject of much investigation throughout the twentieth century. Data from both coroner and emergency room studies indicate that a large proportion of victims of both fatal and nonfatal injuries test positive for blood alcohol—this proportion is greater than one would expect to find in the general population on any given day. The consumption of alcohol (ethanol) has been highly associated with fatalities and serious injuries, but this may be the result of other high-risk behaviors on the part of the drinking accident victim, such as not using seat belts or motorcycle helmets. Studies of alcohol, injury, and risk-taking dispositions in the general population have found risk-taking, impulsivity, and sensation-seeking to be associated with both injury occurrence and alcohol consumption. Those who scored high on risk-taking and sensation-seeking were twice as likely to report an injury for which treatment was obtained during the last year, and were also more likely to report heavier drinking

Roslyn Cappiello, president of the Omaha, Nebraska, chapter of Mothers Against Drunk Driving, was paralyzed from the neck down in an accident caused by a drunk driver. (AP Photo/ Nati Harnik)

(Cherpitel, 1999). Although alcohol cannot be said to actually cause the accident in most cases, alcohol consumption is thought to contribute to both fatal and nonfatal injury occurrence, primarily because it is known to diminish motor coordination and balance and to impair attention, perception, and judgment with regard to behavior, placing the drinker at a higher risk of accidental injury than the nondrinker. The residual or hangover effects of alcohol consumption may also contribute to injury occurrence.

ESTIMATES OF ALCOHOL'S INVOLVEMENT

In emergency room (ER) studies, such as those conducted by Cherpitel (1988), patients testing positive for alcohol have had levels that ranged from 6 to 32 percent—established either directly or from a breath sample taken at the time of admission to the ER. In a review of ER studies, Cherpitel (1993a) determined that this variation in blood alcohol level (BAL) or BLOOD ALCOHOL CONCENTRATION (BAC) is due to differences in the time that passed between the injury and arrival in the ER, to individual characteristics of the particular ER populations studied (such as age, sex, and socioeconomic status—all known to be associated with alcohol consumption in the general population) and to the mix of various types of injury in the ER caseload. For example, alcohol has been found to have a higher prevalence in injuries resulting from violence than from any other cause. In studies that have been restricted to weekend evenings, when one would expect a large proportion of the population to be consuming alcohol, the proportion of those testing positive for alcohol at the time of ER admission has been found to be close to 50 percent. In coroner studies, such as those conducted by Haberman and Baden (1978), alcohol-related fatalities were estimated to account for about 43 percent of all unintentional injuries. (However, the distinction between intentional and unintentional injury is not always readily apparent among victims of fatal injuries.) Studies that have compared estimated BAC between fatal and nonfatal injuries in the same geographic locality have found higher rates of positive BACs among fatal injuries (57%) compared to nonfatal injuries (15%) (Cherpitel, 1996). It is well known that many who drink also consume psychoactive drugs so the independent effect of alcohol on both fatal and nonfatal accidents is not possible to ascertain.

TRAUMA RELATED TO MOTOR VEHICLES

Motor vehicle crashes are the leading cause of death from injury—and the greatest single cause of all deaths for those between the ages of one and thirty-four in the United States. It has been estimated that 7 percent of all crashes and 44 percent of fatal crashes involve alcohol use, and alcohol's involvement is greater for drivers in single-vehicle nighttime fatal crashes (U.S. Department of Health and Human Services, 1997). The risk of a fatal crash is estimated to be from three to fifteen times higher for a drunk driver (one with a BAL of at least .10 to 100 milligrams of alcohol for each 100 milliliters of blood—the legal limit in most U.S. states) than for a nondrinking driver, according to Roizen (1982). Alcohol is more frequently present in fatal than in nonfatal crashes. It is estimated that about 25 to 35 percent of those drivers requiring ER care for injuries resulting from such crashes have a BAL of .10 or greater. The number of alcohol-related crashes has declined in recent years, particularly among younger and older drivers, but has increased among women.

Motorcyclists are at a greater risk of death than automobile occupants, and it has been estimated that up to 50 percent of fatally injured motorcyclists have a BAL of at least .10. Pedestrians killed or injured by motor vehicles have also been found more likely to have been drinking than those not involved in such accidents. Estimates of 31 to 44 percent of fatally injured pedestrians were drinking at the time of the accident. According to Giesbrecht et al. (1989), 14 percent of fatal pedestrian accidents involved an intoxicated driver, but 24 percent involved an intoxicated pedestrian.

HOME ACCIDENTS

Among all nonfatal injuries occurring in the home, an estimated 22 to 30 percent involve alcohol, with 10 percent of those injured having a BAL at the legally intoxicated level at the time of the accident. Coroner data suggest that alcohol consumption immediately before a fatal accident occurs more often in deaths from falls and fires than in motor vehicle deaths.

Falls. Falls are the most common cause of nonfatal injuries in the United States (accounting for over 60%) and the second leading cause of fatal accidents, according to Baker, et al (1992). Alcohol's involvement in fatal falls has been found to range from 21 to 48 percent (with an average of 33%) according to Roizen; for nonfatal falls, alcohol's involvement has been estimated from 17 to 53 percent (with an average of 30%). Alcohol may increase the likelihood of a fall as much as sixty times in those well over the legal limit for intoxication, compared with those having no alcohol exposure.

Fires and Burns. Fires and burns are the fourth leading cause of accidental death in the United States, according to Baker and co-workers. Alcohol involvement has been estimated in 12 to 83 percent of these fatalities (with a median value of 46%), and between 0 and 50 percent among nonfatal burn injuries (with a median value of 17%). In a review of studies of burn victims, Hingson and Howland (1993) estimated that about 50 percent of burn fatalities were intoxicated and that alcohol exposure is most frequent among victims of fires caused by cigarettes.

RECREATIONAL ACCIDENTS

Drownings. Drownings rank as the third leading cause of accidental death in the United States. Haberman and Baden (1978) reported that 68 percent of drowning victims had been drinking, but other estimates have ranged from 30 to 54 percent (with an average of 38%) (Hingson and Howland, 1993). Alcohol is consumed in relatively large quantities by many of those involved in water-recreation (especially boating) activities, and studies suggest that those involved in aquatic accidents are more likely to be intoxicated than those not involved in such accidents. In a review of the literature on those who came close to drowning, Roizen (1982) found that about 35 percent had been drinking at the time.

WORK-RELATED ACCIDENTS

Alcohol's involvement in work-related accidents varies greatly by type of industry, but the proportion of those testing positive for blood alcohol following a work-related accident is considerably lower than for other kinds of injuries, particularly in the United States, since drinking on the job is not a widespread or regular activity. Among work-related fatalities, an estimated 15 percent has been found positive for blood alcohol, and a range of 1 to 16 percent has been estimated for nonfatal injuries, according to Giesbrecht et al. (1989).

VIOLENCE-RELATED TRAUMA

Both fatal and nonfatal injuries commonly result from violence, and these injuries are more likely to be alcohol-related than injuries from any other cause, for men and for women, regardless of age. Such injuries are considered intentional and include those nonfatal injuries resulting from assaults and fights, as well as fatal injuries from homicides and suicides. Alcohol is more likely to be involved in fatal injuries from violence than in nonfatal injuries treated in an ER in the same geographic locality, and a positive BAC in nonfatal injuries among ER patients has been found to range from 17 to 70 percent (Cherpitel, 1993b). These figures refer to alcohol involvement among the victims of violence-related events, and little is known about the alcohol involvement of the perpetrator of such events, but the correlation is thought to also be high. ER patients with violence-related injuries are also more likely to be heavier drinkers and to report alcohol-related problems than those with injuries from other causes.

ALCOHOLISM VERSUS UNWISE DRINKING

The available literature on the role of alcoholism as opposed to unwise drinking in injury occurrence suggests that problem drinkers and those diagnosed as alcoholics are at a greater risk of both fatal and nonfatal injuries than those in the general population who may drink prior to an accident. Alcoholics and problem drinkers are significantly more likely to be drinking and to be drinking heavily prior to an accident than others. Haberman and Baden (1978) found among fatalities from all causes that alcoholics and heavy drinkers were more than twice as likely as nonproblem drinkers to have a BAL at the legal limit. Alcoholics have also been found to experience higher rates of both fatal and nonfatal accidents, even when sober. Analysis of national mortality data found that those who died of injury drank more frequently and more

heavily than those who died of disease, and that daily drinking, binge drinking (consuming 5 or more drinks per occasion), and heavier drinking (14 or more drinks per week) increased the likelihood of injury as the underlying cause of death (Li et al., 1994).

Data from the general U.S. population found the risk of injury increased with an average of one drink a day for both men and women, regardless of age. The risk of injury also increased with the frequency of consuming five or more drinks a day more often than twice a year (Cherpitel et al., 1995). This suggests that the risk for injury may be increased at relatively low levels of consumption, in which case preventive efforts aimed at more moderate drinkers, who are greater in number, may have a larger impact on the reduction of alcohol-related accidents than efforts focused on heavier drinkers, who are fewer in number.

Chronic alcohol abuse has long-term physiologic and neurological effects that may increase the risk of accidents. Chronic drinking also impairs liver function, which plays an important part in injury recovery. A damaged liver compromises the immune system, predisposing the alcoholic to bacterial infections following injury. The risk of accidental death has been estimated to be from three to sixteen times greater for alcoholics than for nonalcoholics, with the highest risk being death from fires and burns. Haberman and Baden (1978) found that among all fatalities from fires, 34 percent were alcoholics.

(SEE ALSO: *Alcohol; Driving, Alcohol, and Drugs; Driving under the Influence; Industry and Workplace, Drug Use in; Social Costs of Alcohol and Drug Abuse*)

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CHERYL J. CHERPITEL

ACCIDENTS AND INJURIES FROM DRUGS Throughout history, humans have taken substances other than food into their bodies in ways that usually were socially accepted. The most common form has been as medicine, in attempts to change some feeling of ill-being or disease, such as PAIN, fatigue, or tension. Some cultures distinguish between socially approved and disapproved uses of substances by labeling those approved as "medicine" and those disapproved as

"drugs." Although the word *medicine* derives from a Latin word meaning "of a physician," throughout much of recorded history and even today, "folk" medicine and "home remedies" are widely practiced. Early medicines were taken exclusively from nature, and PLANTS are still an important source of medicinal products (e.g., foxglove for heart problems, bread mold for penicillin).

Organic HALLUCINOGENIC substances (plants with perception-altering properties, such as peyote and mescaline, and fungi, such as psilocybin mushrooms) have been used in various cultures in the context of religious rituals, with the dramatic visual and aural hallucinations induced being interpreted in spiritual terms. Intoxicating beverages (with alcohol and/or other drugs) also have a long history of use, usually recreationally (i.e., for their relaxing and disinhibiting effects in social situations), but sometimes also for supposed medicinal purposes, as in elixirs and tonics that were marketed as patent medicines in the United States in the late 1800s and early 1900s.

With the development of modern chemistry and scientific growing methods, there has been an increase in the number, types, and strength of both organically derived (principally MARIJUANA) and chemically synthesized (laboratory-created) substances, which has prompted the implementation of measures to regulate their processing or manufacture, distribution, and dispensing (by pharmacists and physicians). In the United States, this regulatory scheme is called the Federal CONTROLLED SUBSTANCES ACT (Public Law 91-513, H.R. 18583, October 27, 1970). This act, which is regularly updated, classifies substances into five categories according to the potential for abuse, accepted medical effectiveness and use, and potential for creating physiological or psychological dependence. The Controlled Substances Act is the basis for federal and state drug laws that specify the conditions making specific substances illicit (illegal) drugs and define differential criminal penalties for their manufacture, distribution, sale, and possession. Substance abuse, for the purposes of this discussion, is defined as the use of illicit drugs, the misuse of medicines (particularly those which must be prescribed by physicians but also those which are available OVER THE COUNTER). Accidents and injuries from the excessive or prohibited (e.g., underage) use of other legal drugs, such as alcoholic beverages, are not included here.

An ambulance crew unloads a patient at the emergency room entrance at Beth Israel Hospital, New York City. In 1995, there were 531,800 drug-related visits to U.S. hospital emergency rooms; more than half were due to drug overdoses. (© Ed Eckstein/CORBIS)

There have been increases since the mid-1960s in both substance abuse and public awareness of it. Here the emphasis will be on an aspect of substance abuse—the unintended, negative consequences—that is relatively well known to researchers but less familiar to the general public and their representatives in government.

UNINTENDED AND NEGATIVE CONSEQUENCES

Data do not exist to document in a comprehensive or detailed manner the extent to which the negative consequences of substance abuse exist worldwide, for specific nations (including the

United States), or for given states or cities. Consequently, I have chosen to present mainly summary information that is based on the best evidence available rather than partial statistical data of questionable accuracy, which would soon be out of date.

The following discussion is divided into four categories of drug problems; acute, chronic, drug-caused, and drug-related. Acute problems are defined as those which usually occur suddenly and often can be remedied in a relatively short time. Chronic problems typically have a relatively gradual onset and tend to persist, sometimes indefinitely. Drug-caused problems, for the purposes of this discussion, are defined as those which have an obvious and/or demonstrated direct causal connection between the use of a substance and a negative effect. In drug-related problems, negative outcomes result from drug-diminished capacities and their effects on user behaviors.

Acute Drug-Caused Problems. The National Institute on Drug Abuse's DRUG ABUSE WARNING NETWORK (DAWN) estimates that *alcohol-in-combination* (alcohol used with another drug, the criterion for reporting this substance to DAWN) represents the most frequently reported category in drug-related hospital emergency department visits.

"Other drugs" (illicit drugs, accidentally misused, and intentionally abused legal medications) collectively represent an acute drug-caused problem that may equal or surpass ALCOHOL in this category. The DAWN system is the best source of documentation of these problems, capturing data on substance abusers who come or are brought to hospital emergency departments, particularly for negative and/or unexpected reactions. These cases are usually thought of as overdoses, attributable to (a) tolerance effects (the need to use increasingly larger doses to achieve the same PSYCHOACTIVE effects). (b) inexperienced users with panic reactions, or (c) the use of a substance of greater strength than intended or expected. There also is increasing evidence that users of some drugs, such as COCAINE, can experience medical emergencies and deaths from seizure disorders and allergic reactions. This is true not only for first-time users but also experienced users at their regular (and sometimes low) dosage levels. Other frequently noted hospital emergency department (and medical examiner) cases involve SUICIDE ATTEMPTS (and successes) where medications and/or drugs are the means chosen.

Chronic Drug-Caused Problems. Intravenous drug users can suffer from chronic cardiovascular problems that may be primarily attributable to infections and damage from "fillers"—talcum powder, cornstarch, or baking soda added to drugs to increase volume, unit sales, and profits—that have been injected. Some drugs—especially the DESIGNER DRUGS, where an easily added molecule can produce a deadly variant of the intended substance—are neurotropic/neuropathic (have an affinity for and do damage to nerve endings and tissue). Researchers and clinicians are studying and treating individuals who are afflicted with Parkinson's disease caused by such "party" drugs.

One chronic drug-caused problem is particularly tragic because the individuals most damaged are totally innocent and defenseless against the substances that can cause them permanent disabilities or even death. I refer here to teratogenic drug use (use by pregnant women that causes abnormal fetal development).

When the use of CRACK-cocaine exploded in the mid-1980s, fetal developmental damage resembling FETAL ALCOHOL SYNDROME began to be noted among infants born to women who had used this drug during the pregnancy. Although accurate counts are difficult to obtain, estimates cited in a 1990 government report—which are not based on representative national samples—range from 100,000 infants annually exposed to cocaine alone to as many as 375,000 annually exposed to drugs in general; more recent research, however, produced much lower estimates.

Acute Drug-Related Problems. Acute drug-related problems are typified by physical trauma; because of inebriation- or intoxication-impaired judgment or motor control/coordination, substance-abusing individuals can and do accidentally injure themselves.

Common examples in this category are accidental weapon discharges and motor vehicle and boating accidents; drownings, falls, and electrocutions are less common but not rare.

Even more unfortunate are instances where others—clean and sober—are injured or killed by the impaired substance abuser. (These cases often go unnoted; for example, DAWN records only cases where the injured or deceased had drugs "on

board.'') In addition to the types of accidental injuries noted immediately above, there are anecdotal (verbally reported but not documented) accounts which suggest that spouses, children, other relatives, and friends often are the victims of drug-impaired individuals. With diminished emotional control, inhibitions, and judgment, substance abusers often inflict physical trauma (e.g., gunshot, blunt force, or penetrating injuries) in chance encounters with strangers (other drivers, businesspeople), in disputes with coworkers or friends, in domestic disputes, or in physical abuse of their children. Moreover, analysis by Brookoff and his colleagues (1993) conclude that DAWN reporting procedures seriously underreport drug-involved emergency department cases, especially those with serious trauma.

Chronic Drug-Related Problems. Some of the most common chronic drug-related problems are similar to those resulting from in utero exposure of fetuses to cocaine and other development-impairing substances, in that severe illness and death are frequently involved and individuals other than the users themselves are victims. This class of drug-related problems is most closely associated with injecting drug use because the category is defined by infectious diseases that can be transmitted via sharing of unsanitized hypodermic needles. The most deadly infectious agent spread in this manner is the HUMAN IMMUNODEFICIENCY VIRUS (HIV), which causes ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS). Another potentially deadly infection spread in this manner is the liver disease hepatitis B. Various sexually transmitted diseases (including AIDS, syphilis, and gonorrhea), as well as tuberculosis, are among the other debilitating and often fatal diseases that are chronic problems related to substance abuse.

DRUG-SPECIFIC NEGATIVE CONSEQUENCES

Discussion in this section is limited to the specific negative consequences of a few of the most prevalent illicit drugs: marijuana, cocaine, and heroin. Other illicit substances that could have been discussed here include LYSERGIC ACID DIETHYLAMIDE (LSD), PHENCYCLIDINE (PCP), and other ''alphabet'' or ''designer'' drugs (''ecstacy,'' etc.), AMPHETAMINE, and METHAMPHETAMINE (and its smokable form, ''ice''). This discussion also could well include legal substances that are abused by inhalation, such as gasoline, airplane glue, and various solvents, which are mundane but widely used—especially in economically depressed areas of the United States and in developing nations. These are very harmful to lungs and brain cells, and are often deadly.

Marijuana. The smoking of marijuana, probably the most widely used illicit drug, may well have more serious acute and chronic consequences than once thought. Recent and continuing research is casting new light on the chronic health risks posed by marijuana smoking, contradicting the conventional wisdom that it is less harmful than either drinking alcoholic beverages or smoking tobacco. For example, it is reported that three times the tar is delivered (and four times more is deposited) to the mouth and lungs per puff from a marijuana joint than from a filter-tipped cigarette. Smoking marijuana also produces up to five times more carbon monoxide in the user's blood than does tobacco. Knowledge is also being accumulated regarding the specific health-damaging mechanisms from the 426 known chemicals contained in *Cannabis sativa* (which are transformed into over 2,000 when ignited).

Among the pertinent facts already established is that 70 of these chemicals are fat-soluble and accumulate in fatty body tissue, notably the brain, lungs, liver, and reproductive organs. This represents a persistence-of-residue effect in which portions of THC (delta-9-TETRAHYDROCANNABINOL), the most potent psychoactive chemical in marijuana, not only remain in the body (and are thus detectable) for several weeks following use, but also accumulate with repeated use. This THC buildup is particularly noteworthy when one considers that the potency (THC content) of marijuana has increased dramatically since the 1960s, when the average potency was about 0.2 percent.

Regular marijuana smoking can contribute to emotional and other behaviorally defined mental-health problems through degraded interpersonal relationships and arrested development. The mechanism for this seems to be a drug-induced perception of well-being and problem abatement that may not reflect reality and contributes to avoidance rather than coping with life situations.

Research findings from the 1980s and 1990s highlight a marijuana health risk that is largely unmeasured but may be much greater than gener-

ally considered. Researchers examined blood samples from over 1,000 individuals brought to a hospital trauma unit with severe injuries. Two-thirds of these individuals had accidental injuries associated with the operation of motor vehicles (drivers, passengers, and pedestrians injured by cars, trucks, or motorcycles). Using a blood test that normally ceases to detect THC around 4 hours after use, the researchers found about 34 percent of these accident victims had psychoactive levels of THC in their blood when they arrived at the hospital. A more recent study found that 45 percent stopped for reckless driving tested positive for marijuana. Given that there currently are no simple, legally recognized tests to detect marijuana use, the extent of marijuana-related vehicular injuries and deaths is an unknown but potentially sizable statistic.

Cocaine. Many readers undoubtedly have heard that Sigmund FREUD, the famous Viennese psychoanalyst, was an avid user and proponent of cocaine. The initial account of his observations of the drug's effects for himself and some of his patients indeed was glowing. It was translated from German and reprinted in an American medical journal in the mid-1880s, thus popularizing the drug in the United States and prompting its incorporation into products from patent medicines to soft drinks. Less well reported is the fact that Freud and his colleagues had discovered the significant negative effects of cocaine by the end of that same decade and had withdrawn their support for its applications in medical therapy.

Cocaine has had several periods of popularity in the United States as a drug of abuse, with the most recent beginning in the early 1980s. Touted as a safe, nonaddicting, recreational drug, cocaine hydrochloride (in powder form) was "snorted" (inhaled) by millions who liked the absence of hypodermic needles, the lack of lung-cancer risk, and the rapid high, with its feelings of alertness, wittiness, and sexual prowess.

Unfortunately, cocaine users often progress from casual to compulsive patterns of use. The grandiose perceptions of heightened mental and physical abilities inevitably wane (typically within 20 minutes following use), and the resulting dysphoria (opposite of euphoria) is so marked in contrast that it resembles depression. Trying to relieve the depression and regain the euphoria, cocaine abusers repeat this cycle over long periods (called binges) until their supplies, resources, and/or stamina are exhausted. In the study of reckless drivers noted earlier, 25 percent tested positive for cocaine.

The risk of infection with HIV and other sexually transmitted diseases is high among compulsive cocaine users, particularly female crack users. Often forsaking socially acceptable means of earning income, they live an existence that revolves around crack use. Many maintain their crack supply by repeatedly selling or trading their sexual services, with each unprotected sexual contact increasing the chance that they will have an HIV-infected partner.

Other manifestations of negative effects of cocaine abuse include hyperstimulation; digestive disorders, nausea, loss of appetite and weight; tooth erosion; nasal mucous membrane erosion, including perforations of the nasal septum (holes in the membrane separating the nostrils); cardiac irregularities; stroke (from vascular constriction); convulsions (especially among individuals prone to seizure disorders); and paranoid psychoses and delusions of persecution. Cocaine is a notoriously fickle drug—some experts say it behaves as though it belongs in other pharmacological categories besides stimulant. A highly publicized case was the 1986 cocaine-induced death of the athlete Len Bias, who reportedly was a first-time user of a small amount. In addition, research indicates that the concurrent use of cocaine and alcohol (a common practice) produces a new, liver-and brain-accumulating and -damaging drug (COCA-ETHYLENE) within the user's body. It is implicated in puzzling low-dosage "excited delirium" fatalities and increased mortality risks for individuals with existing heart problems.

Some of the fetal damage from maternal cocaine use occurs because cocaine is a vasoconstrictor, a useful characteristic for topical application in delicate medical procedures, such as eye surgery, but a decided negative as it concerns the placenta of a pregnant woman. The restriction of blood flow through the placenta limits nutrients and oxygen to the fetus, leading to retarded growth and development of vital organs. Heavy cocaine use during pregnancy also can cause spontaneous abortion, and anecdotal reports of cocaine being used intentionally for this purpose are not uncommon. Premature separation of the placenta from the uterus, another common medical complication among cocaine-using pregnant women, results in either a premature birth or a stillbirth. Surviving infants usually have low (sometimes very low) birth-

weights, and low birthweight itself increases risk for a variety of problems. Cocaine-exposed underweight newborns have been documented to be at greater risk for stroke and respiratory ailments, and at much greater risk for sudden infant death syndrome (SIDS or crib death). Research studies are being conducted to confirm anecdotal and preliminary studies that indicate higher rates of retarded emotional, motor, and cognitive development, including ATTENTION DEFICIT DISORDERS, among such children entering school.

Heroin. Paradoxically, the negative direct physiological consequences to the user that are attributable to heroin itself are less than from the use of tobacco, alcohol, cocaine, or many prescription drugs. This does not mean that heroin is a drug whose use is without negative consequences, however. Heroin is highly addictive, and once its central nervous system depressive effects wear off, (typically in 4 to 6 hours), users tend compulsively to seek sources and means for another "hit." This often leads to socially unproductive, self-neglectful lifestyles, not uncommonly involved with income-producing crime committed to maintain the addiction. Fetuses exposed to heroin from their mothers' use during pregnancy suffer many of the same negative effects as those exposed to cocaine.

In addition, heroin users frequently experience negative reactions and overdoses because of TOLERANCE effects, since the drug purity and type of filler may vary widely among dealers. Often, they are unknown with certainty by the user. However, the greatest threat to current heroin users' health and lives undoubtedly stems from the risks of hepatitis and HIV infection from sharing contaminated hypodermic syringes and needles. Their risks of infection with HIV and other diseases through sexual activity are elevated in ways similar to those described for cocaine users. The risks of fetal HIV infection among pregnant heroin-using women is also increased because of their own needle use and the likelihood that they have had at least one intravenous-drug-using sexual partner.

ECONOMIC COSTS OF SUBSTANCE ABUSE

Numerous studies designed to estimate the cost or burden of drug abuse were conducted in recent years. This presentation will focus on two of the most recent and authoritative reports.

Gerstein and Harwood (1990) produced a set of estimated societal ECONOMIC COSTS of the illicit drug problem in the United States totaling 71.9 billion dollars. This total is broken down into categories; almost half ($33.3 billion) of the total estimated costs was attributed to productivity losses resulting from substance-abuse-impaired workers. More than half of the total estimated cost was attributed to the criminal aspects of drug abuse ($5.5 billion to tangible losses to crime victims; $12.8 billion to law enforcement; $17.6 billion to lost economic productivity because of time spent in crime or in prison). A minor portion of the estimated costs was assigned to drug prevention and treatment ($1.7 billion) and drug-related AIDS ($1.0).

The Gerstein and Harwood estimate is an update incorporating "a number of statistical updates and revisions" of a similar estimate for 1980 (Harwood et al., 1984), which totaled $46.9 billion dollars.

Rice et al. (1990) produced what is probably the most authoritative work currently available on this topic: *The Economic Costs of Alcohol and Drug Abuse and Mental Illness: 1985*. Among the multiple objectives of the study were the following: to estimate as precisely as possible the economic costs to society of alcohol abuse, drug abuse, and mental illness (ADM) for 1985, the most recent year for which reliable data were available; to update previous cost estimates, using new data sources and a revised methodology; and to develop an improved approach to deal with the issues of COMORBIDITY (the tendency for cases to overlap, that is, for individuals to have problems in more than one ADM category). Briefly, Rice et al. produced ADM cost estimates for 1980, 1985, and 1988. Estimated costs for illicit drug problems (in billions of dollars) were 1980, 46.9; 1985, 44.1; 1988, 58.3.

Their cost estimates are considered to be the best available, but even the analysts who produced them will readily agree that they are probably not very precise. It is probable, in my opinion, that the health and social care costs for AIDS-infected drug abusers is underestimated even for the years addressed, and are undoubtedly significantly higher in the new millenium. Similarly, health and social service costs for infants and children who have suffered prenatal exposure to drugs undoubtedly have mushroomed since 1985, the approximate beginning of the crack-cocaine epidemic (these costs

could become astronomical if the worst fears regarding permanent learning disabilities are confirmed).

(SEE ALSO: *Accidents and Injuries from Alcohol; Alcohol: History of Drinking; Dover's Powder; Driving, Alcohol, and Drugs; Fetus: Effects of Drugs on; Social Costs of Alcohol and Drug Abuse*)

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JAMES E. RIVERS

ACETALDEHYDE *See* Complications: Liver (Alcohol), Disulfuram

ACETAMINOPHEN *See* Analgesic; Drug Metabolism; Pain, Drugs Used for

ACETYLCHOLINE Acetylcholine (ACh) is a major NEUROTRANSMITTER in the central and peripheral nervous systems. It is the ester of acetate and choline, formed by the enzyme choline acetyltransferase, from choline and acetyl-CoA. This was the first substance (ca. 1906) to meet the criteria of identification for a neurotransmitter. Later, acetylcholine was shown to be the general neurotransmitter for the neuromuscular junctions. In all vertebrate species, it is the major neurotransmitter for all autonomic ganglia and the neurotransmitter between parasympathetic ganglia and their target cells. Acetylcholine neurotransmission occurs widely within the central nervous system. Collections of NEURONS arising within the brain—the medulla, the pons, or the anterior diencephalon—innervate a wide set of cortical and subcortical targets; some of these circuits are destroyed in Alzheimer's disease.

(SEE ALSO: *Scopolomine and Atropine*)

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FLOYD BLOOM

ACETYLMETHADOL *See* L-Alpha-Acetylmethadol (LAAM)

ACID *See* Lysergic Acid Diethylamide (LSD) and Psychedelics; Slang and Jargon

ACUPUNCTURE *See* Treatment Types: Acupuncture

ADAMHA *See* Treatment, History of

ADDICT *See* Addiction; Concepts and Definitions

ADDICTED BABIES Technically, the term *addicted babies* should refer to infants who are born passively physically dependent on drugs. In practice, it is used to refer to all babies extensively exposed to drugs before birth. According to a recent federal government report estimated that in the United States each year some 320,000 babies are

born exposed to alcohol and illicit drugs while in the uterus; a far larger number have been exposed, in utero to sedatives and nicotine. The increased recognition of such drug-exposed babies parallels the dramatic increase in drug use, both licit and illicit, by women since the beginning of the 1970s.

Drug-addicted women often use multiple substances—including ALCOHOL, NICOTINE, MARIJUANA, TRANQUILIZERS, COCAINE, and OPIOIDS (e.g., HEROIN and METHADONE). The drugs are carried across the placenta from mother to fetus. The clinical presentation of the newborn (neonate) depends on the substance, the amount and frequency used during pregnancy, and the time since last use. Withdrawal will occur in 55 to 94 percent of infants exposed to heroin and other opioids. Infants of regular heavy users usually have a low birth weight, because of intrauterine growth retardation and frequent premature births.

If the mother has used a large dose of depressant drug (alcohol, any number of sedative-hypnotics, or heroin) immediately before delivery, the neonate may have respiratory depression and may require resuscitation. If the mother has used one of these drugs regularly during pregnancy, there may be a neonatal abstinence or withdrawal syndrome, which has as key features irritability, tremor, and increased muscle tone. Other symptoms include poor nervous system irritability, gastrointestinal disorders that lead to poor feeding, vomiting, and diarrhea; high-pitched cry; difficulty in sleeping; and sneezing, sweating, yawning, nasal stuffiness, rapid breathing, and seizures. Withdrawal from heroin generally occurs within forty-eight hours of birth, but it can be somewhat delayed with the longer-acting methadone. Alcohol-exposed infants may develop a very similar withdrawal syndrome, except with the more frequent occurrence of seizures.

Cocaine, a stimulant, constricts blood vessels, thereby decreasing oxygen delivery to the fetus. Consequently, neonatal stroke has occurred. Although cocaine withdrawal symptoms have been reported in neonates, they probably reflect acute cocaine intoxication when the mother's last use was close to the time of birth. Such infants often appear less alert and less responsive to external stimuli than noncocaine-affected newborns, which may represent true withdrawal—comparable to the so-called crash seen in adults.

MANAGEMENT

A thorough alcohol and drug history should be obtained from the expectant mother—and this should be corroborated by testing the urine of both mother and newborn for alcohol and other drugs. Newborns should be closely monitored for signs of withdrawal for a minimum of forty-eight to seventy-two hours, and longer when the mother has been on METHADONE-maintenance treatment. Since symptoms of withdrawal are nonspecific and may be confused with a variety of infections or metabolic disturbances, a search for concurrent illness to explain any symptoms is mandatory.

Most hospital nurseries use a standardized neonatal abstinence-syndrome scoring system. After the infant is born the hospital will monitor their sleep habits, temperature, and weight. The earliest withdrawal symptoms are treated by intravenous fluids, swaddling, holding, rocking, a low-stimulation environment, and small feedings of hypercaloric formula—for weight gain. If symptoms continue or increase, medication may be initiated. Common medications include PAREGORIC (camphorated tincture of opium), or Phenobarbital for opioid withdrawal; PHENOBARBITAL or DIAZEPAM for alcohol withdrawal. Diazepam is also used to help with cocaine hyper excitability.

Interviewing the mother is essential in reviewing the anticipated home environment. Unfortunately, addicted babies are often at high risk for either abuse or neglect or both. Normal maternal-infant bonding is difficult in the case of an irritable poorly responding neonate and a mother dealing with the guilt, low self-esteem, poverty, inadequate housing, and an abusive or absent partner or parent, which often accompany her own drug addiction. A referral to child protection services may therefore be indicated.

OUTCOME

Some studies have indicated that addicted babies have an increased risk for breathing abnormalities and sudden infant death syndrome (SIDS). Many studies of opioid-exposed children up to school age show few differences from nonexposed children of a similarly disadvantaged environment. According to a study conducted by the Society for Maternal-Feral Medicine, children who were exposed to cocaine in utero are 2.4 times more likely

to have language problems than children not exposed to cocaine. The outcome for babies with prenatal alcohol exposure depends on the extent of the damage to the fetus born with FETAL ALCOHOL SYNDROME (FAS).

The drug-addicted mother's lifestyle is often characterized by inadequate or no prenatal care, poor nutrition, and prostitution, any or all of which may result in a high risk for medical and obstetrical complications. Needle use may result in infection with hepatitis B and HIV. Methadone-maintenance programs for heroin-addicted mothers generally offer medical and social services to help mitigate these negative influences and contribute to the improved outcome seen in their babies, despite a continuation of opioid drug addiction on their part.

Drug WITHDRAWAL, in the absence of other problems, is now readily managed in hospitals. The outcome for addicted babies depends on any permanent medical sequelae as well as the quality of the postnatal environment. These babies often require ongoing medical, school, and social services to ensure that they reach their maximal potential.

(SEE ALSO: *Fetus, Effects of Drugs on; Pregnancy and Drug Dependence*)

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REVISED BY SHEILA DOW

ADDICTION (formerly the *British Journal of Addictions*) is the oldest specialist journal in its field, originating in 1884 as the *Proceedings for the Society for the Study and Cure of Inebriety*. The bound volumes provide a unique perspective on the historical development of clinical practice, policy debates, and the emergence of a scientific tradition. *Addiction* is today among the most international of journals focusing on addiction. In addition to publishing refereed research reports, editorial policy has been directed at establishing it as a leading forum for informed debate—specially commissioned "commentary" series contribute to this purpose. The prestigious *Addiction* Book Prize is awarded annually. In furtherance of its role as an international medium of scientific exchange, the journal, which has its head office in Britain, in 1993 established regional offices in the United States and Australia.

GRIFFITH EDWARDS

ADDICTION RESEARCH UNIT (ARU) (U.K.) The Addiction Research Unit of the Institute of Psychiatry, University of London, was set up in 1967 on the Joint Maudsley Hospital/Institute of Psychiatry campus in Camberwell, South East London, England. Its funding has come from many different sources (principally the Medical Research Council), but its fundamental identity has always been that of a university center, which has close ties and valued links with a postgraduate psychiatric medical school (the Institute) and a teaching hospital (the Maudsley). Its present scientific staff number thirty, with the mix of psychiatrists, psychologists, statisticians, and social scientists reflecting the ARU's interdisciplinary commitment. The ARU's field of study embraces TOBACCO as well as ALCOHOL and other drugs.

The ready access to hospital facilities has, over the years, greatly aided the ARU's ability to conduct clinical research. One line of investigation continuously developed from this base has, for example, concentrated on definition, description, measurement, and validation of the dependence-syndrome concept—in relation to both alcohol and opiates. The Smoking Section has done much to demonstrate that the cigarette habit is indeed nicotine dependence. A sustained effort has also been directed at the development of methods for assessing treatment efficacy through controlled trials. As regards both alcohol dependence and smoking, results have generally tended to support research in

fairly simple, minimalist interventions, often delivered in the primary-care setting. Another line of research has focused on long-term follow-up studies and the determinants of "natural history and career."

If the above paragraph identifies some of the ARU's core activities, much else has also gone on. For example, the ARU, for a number of years, employed a professional historian, who did much to open up an understanding of the history of opiate use in Britain. Epidemiological research has at times been undertaken. As of the 1990s, the ARU has been developing a computerized method for handling interview texts. In the psychophysiological laboratories, studies of cue responsiveness are being conducted with patients and normal subjects. A new line of research is focusing on the relationship between the two axes of problems and dependence. The Smoking Section has contributed to studies on the impact of passive smoking.

Besides the research, a great deal of clinical and research training is being undertaken, and the ARU runs a full-time one-year course leading to a Master of Science (MSc) in clinical and public health aspects of addiction. The ARU enjoys the benefits of an extensive national and international network of friendships and professional contacts through its many former staff and students who today hold influential positions. There are particularly strong links with the developing world, and support has often been given by the ARU to the World Health Organization.

In April 1991, the ARU moved to a purpose-built office and laboratory accommodation on the same campus, and it became associated with the newly established National Addiction Centre. The ARU's Smoking Section has also been strengthened by involvement with the recently established Health Behaviour Research Unit, funded by the Imperial Cancer Research Fund.

GRIFFITH EDWARDS

ADDICTION SEVERITY INDEX (ASI)

This is a semistructured interview designed to provide important information about aspects of the life of patients that may contribute to their substance-abuse problems. The Addiction Severity Index (ASI) provides a general overview of substance-abuse problems rather than a focus on one particular area (200 questions on 7 subscales). Developed by McLellan and coworkers in 1981, the ASI has been translated into seventeen languages—Japanese, French, Spanish, German, Dutch, and Russian among them—and was designed to be administered by a technician or counselor. Consistent guidelines for each question on the ASI have been compiled in training materials including two videos and three instructional manuals. Self-training can be accomplished by using the video along with the administration manual, although a one-day formal training seminar is recommended. (Since the instrument is in the public domain, there is no charge for it; only a minor fee is charged for copies of the administration materials and the computer scoring disk.)

The interview is based on the idea that addiction to drugs or alcohol is best considered in terms of the life events that preceded, occurred at the same time as, or resulted from the substance-abuse problem. The ASI focuses on seven functional areas, or subscales, that have been widely shown to be affected by the substance abuse: medical status, employment and support, drug use, alcohol use, legal status, family and social status, and psychiatric status. Each of these areas is examined individually by collecting information regarding the frequency, duration, and severity of symptoms of problems both historically over the course of the patient's lifetime and more recently during the thirty days prior to the interview. Within each of the problem areas, the ASI provides both a 10-point, interviewer-determined severity rating of lifetime problems as well as a multi-item composite score (computer-calculated) that indicates the severity of the problems in the past thirty days.

The ASI is widely used clinically for assessing substance-abuse patients at the time of their admission for treatment. It takes about an hour to gather the basic information that forms the first step in the development of a patient profile for subsequent use by the staff in planning treatment. Researchers have found the ASI useful because the composite scores and the individual variables can be compared within groups over time as a measure of improvement, or between groups of patients at a posttreatment follow-up point as a measure of outcome of treatment. The ASI has shown excellent reliability and validity across a range of types of patients and treatment settings in this country and

abroad, although reliability in patients with severe mental illness varies considerably.

The ASI is particularly useful in the diagnosis and treatment of alcohol problems. It provides information on the frequency, duration, and intensity of alcohol and drug use. The ASI also examines psychosocial functioning (medical, legal, employment, psychological, and social/family), which is crucial to understanding alcohol dependency. The ASI is also a cost-effective alternative for the as[-fj]sessment of alcohol problems—when compared to the Structured Clinical Interview for DSM-III-R (SCID), a more formal and more expensive approach.

The ASI has been used in many studies, including one in the late 1990s, involving cocaine-dependent pathological gamblers. This particular study used the ASI to determine socioeconomic characteristics of the subjects. Another late-1990s study (involving methadone patients) used results of the ASI in comparison with criminal history and personality traits to support the idea that antisocial behavior is more than just a personality disorder. In 2000, the outcome of a study on cocaine dependents illustrated, by comparing Spielberger State-Trait Anxiety Inventory and the Alcohol Composite Index of the ASI, that high anxiety scores decrease with time, regardless of clinical management.

To further increase the usefulness of the ASI, clinicians and researchers have added questions to supplement the "old" version, including questions about leisure time activities, childhood religion, childhood illnesses, age of first drug/alcohol use, sexual orientation, and military service. The addition of these questions is thought to supplement the already-sought information, since much of the impetus for these questions is from those who administrate the ASI and deal directly with the tested individuals. Also, a T-ASI (Teen-Addiction Severity Index) has been developed to help adolescents. It is an age-appropriate modification of the original ASI with 133 questions in 7 domains: Psychoactive Substance Use, Family Function, Peer-Social Relationships, School-Employment Status, Legal Status, and Psychiatric Status.

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ADDICTION: CONCEPTS AND DEFINITIONS This article deals with a number of concepts related to the basic nature of addiction, that are widely used but often misused, and that have undergone significant changes since the term *addiction* first came into the common vocabulary. In the following discussion, the terms are grouped according to themes, rather than being arranged in alphabetic order.

ABUSE AND MISUSE

In everyday English, *abuse* carries the connotations of improper, perverse, or corrupt use or practice, as in child abuse, or abuse of power. As applied to drugs, however, the term is difficult to define and carries different meanings in different contexts. In relation to therapeutic agents such as Benzondiazepines or Morphine, the term *drug abuse* is applied to their use for other than medical purposes, or in unnecessarily large quantities. With reference to licit but non-therapeutic substances such as Alcohol, it is understood to mean a level of use that is hazardous or damaging, either to the user or to others. When applied to illicit substances that have no recognized medical applications, such as Phencyclidine (PCP) or Mescaline, any use is generally regarded as abuse. The term *misuse* refers more narrowly to the use of a therapeutic drug in any way other than what is regarded as good medical practice.

Substance abuse means essentially the same as drug abuse, except that the term "substance" (shortened form of psychoactive substance) avoids any misunderstanding about the meaning of "drug". Many people regard as drugs only those compounds that are, or could be, used for the treatment of disease, whereas "substances" would also include materials such as organic solvents, Morning Glory Seeds or toad venoms, that have no medical applications at present but are "abused" in one or more of the senses defined above.

The best general definition of drug abuse is the use of any drug in a manner that deviates from the approved medical or social patterns within a given culture at a given time. This is probably the concept underlying the official acceptance of the term *abuse* in such instances as the names of the National Institute on Drug Abuse (USA) and the Canadian Centre on Substance Abuse. Such official acceptance, however, does not prevent the occurrence of ambiguities such as those mentioned in the next section.

RECREATIONAL OR CASUAL DRUG USE

These two terms are generally understood to refer to drug use that is small in amount, infrequent, and without adverse consequences, but these characteristics are not in fact necessary parts of the definitions. In the terminology recommended by the World Health Organization (WHO), the two terms are synonymous. However, *recreational use* really refers only to the motive for use, which is to obtain effects that the user regards as pleasurable or rewarding in some way, even if that use also carries some potential risks. *Casual use* refers to occasional as opposed to regular use, and therefore implies that the user is not dependent or addicted (see below), but it carries no necessary implications

with respect to motive for use or the amount used on any occasion. Thus, a casual user might become intoxicated (see below) or suffer an acute adverse effect on occasions, even if these are infrequent.

Occasional use may also be *circumstantial* or *utilitarian*, if employed to achieve some specific short-term benefit under special circumstances. The use of AMPHETAMINES to increase endurance and postpone fatigue by students studying for examinations, truck drivers on long hauls, athletes competing in endurance events, or military personnel on long missions, are all instances of such utilitarian use. Most observers also consider the first three of these to be abuse or misuse, but many would not regard the fourth example as abuse because it is or was prescribed by military authorities under unusual circumstances, for necessary combat goals. Nevertheless, in all four instances the same drug effect is sought for the same purpose (i.e., to increase endurance). This illustrates the complexities and ambiguities of definitions in the field of drug use.

INTOXICATION

This is the state of functional impairment resulting from the actions of a drug. It may be acute, i.e., caused by consumption of a high dose of drug on one occasion; it may be chronic, i.e., caused by repeated use of large enough doses to maintain an excessive drug concentration in the body over a long period of time. The characteristic pattern of intoxication varies from one drug to another, depending upon the mechanisms of action of the different substances. For example, intoxication by alcohol or barbiturates typically includes disturbances of neuromuscular coordination, speech, sensory functions, memory, reaction time, reflexes, judgment of speeds and distances, and appropriate control of emotional expression and behavior. In contrast, intoxication by amphetamine or cocaine usually includes raised blood pressure and heart rate, elevation of body temperature, intense hyperactivity, mental disturbances such as hallucinations and paranoid delusions, and sometimes convulsions. The term may be considered equivalent to *overdosage*, in that the signs of intoxication usually arise at higher doses than the pleasurable subjective effects for which the drug is usually taken.

HABIT AND HABITUATION

In everyday English, a *habit* is a customary behavior, especially one that has become largely automatic or unconscious as a result of frequent repetition of the same act. In itself, the word is simply descriptive, carrying no fixed connotation of good or bad. As applied to drug use, however, it is somewhat more judgmental. It refers to regular persistent use of a drug, in amounts that may create some risk for the user, and over which the user does not have complete voluntary control. Indeed, an *alcohol habit* has been defined in terms very similar to those used to define dependence (see below). In older writings, *habit strength* was used to characterize the degree of an individual's habitual drug use, in terms of the average amount of the drug taken daily. Reference to a *drug habit* implies that the drug use is the object of some concern on the part of the user or of the observer, but that it may not yet be sufficiently strongly established to make treatment clearly necessary.

Habituation refers either to the process of acquiring a drug habit, or to the state of the habitual user. Since habitual users frequently show increased tolerance (decreased sensitivity to the effects of the drug; see below), habituation is also used in the earlier literature to mean an acquired increase in tolerance. In its early reports, the WORLD HEALTH ORGANIZATION EXPERT COMMITTEE ON DRUG DEPENDENCE (as it is now known, after several changes of name) used the term *habituation* to refer to a state arising from repeated drug use, that was less serious than addiction in the sense that it included only psychological and not physical dependence, and that harm, if it occurred, was only to the user and not to others. Drugs were classified according to whether they caused habituation or addiction. These distinctions were later recognized to be based on misconception, because (1) psychological (or psychic) dependence is even more important than physical dependence with respect to the genesis of addiction; (2) any drug that can damage the user is also capable of causing harm to others and to society at large; and (3) the same drug could cause effects that might be classed as "habituation" in one user and "addiction" in another. The WHO Expert Committee later recommended that both terms be dropped from use, and that *dependence* be used instead.

PROBLEM DRINKING

In an effort to avoid semantic arguments and value judgments about abuse or addiction, clinical and epidemiological researchers have increasingly made use of objective operational definitions and measures. *Problem drinking* is alcohol consumption at an average daily level that causes problems, regardless of whether these are of medical, legal, interpersonal, economic, or other nature, to the drinker or to others. The actual level, in milliliters of absolute alcohol per day, will obviously vary with the individual, the type of problem, and the circumstances. The advantage of this term is that a drinker who may not meet the criteria of dependence or who is reluctant to accept a diagnostic label of alcoholism or addiction can often be led to acknowledge that a problem exists and requires intervention.

ADDICTION AND DEPENDENCE

The term *addiction* was used in everyday and legal English long before its application to drug problems. In the sixteenth century it was used to designate the state of being legally bound or given over (e.g., bondage of a servant to a master) or, figuratively, of being habitually given over to some practice or habit; in both senses, it implied a loss of liberty of action. At the beginning of the twentieth century it came to be used more specifically for the state of being given over to the habitual excessive use of a drug, and the person who was "given over" to such drug use was described as an *addict*. By extension from the original meanings of addiction, drug addiction meant a practice of drug use that the user could not voluntarily cease, and loss of control over drinking was considered an essential feature of alcohol addiction. The emphasis was placed upon the degree to which the drug use dominated the person's life, in such forms as constant preoccupation with obtaining and using the drug, and inability to discontinue its use even when harmful effects made it necessary or strongly advisable to do so.

During the first half of the twentieth century, however, the pharmacological and social consequences of such use came increasingly to be the defining criteria. In 1957, the WHO Expert Committee defined addiction as "a state of periodic or chronic intoxication produced by the repeated consumption of a drug (natural or synthetic). Its characteristics include (1) an overpowering need (compulsion) to continue taking the drug and to obtain it by any means; (2) a tendency to increase the dose [later said to reflect tolerance]; (3) a psychic (psychological) and generally a physical dependence on the effects of the drug; and (4) detrimental effect on the individual and on society". *Physical dependence* is an altered physiological state arising from the regular heavy use of a drug, such that the body cannot function normally unless the drug is present. This state is recognizable only by the physical and mental disturbances that occur when drug use is abruptly discontinued or "withdrawn", and the constellation of these disturbances is known as a *withdrawal syndrome*. The specific pattern of the withdrawal syndrome varies according to the type of drug that has been used, and usually consists of changes opposite in direction to those originally produced by the action of the drug. For example, if opiate drugs cause constipation, their withdrawal typically produces diarrhea; if cocaine causes prolonged wakefulness and euphoria, the withdrawal syndrome will include profound sleepiness and depression; if alcohol decreases the reactivity of nerve cells, the withdrawal syndrome will include signs of over-reactivity, such as exaggerated reflexes or convulsions. In all cases, however, the withdrawal syndrome is quickly abolished by resumption of administration of the drug or of a substitute drug with a very similar pattern of actions.

It is now well recognized that a person can become physically dependent on a drug given in high doses for medical reasons (e.g., morphine given repeatedly for relief of chronic pain) and yet not show any subsequent tendency to seek and use the drug for non-medical purposes. The WHO Expert Committee therefore revised its definitions and concepts in 1973, substituting the single term dependence for the two terms addiction and habituation. Unfortunately, this change has not led to uniform terminology or concepts.

In essence, dependence is a state in which the individual can not function normally—physically, mentally or socially—in the absence of the drug. A simple definition given in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* published by the American Psychiatric Association (DSM-IV) includes only one fundamental element: compulsive use of the drug despite the occurrence of adverse consequences. However, a

more detailed description of the *dependence syndrome* includes both physical components (increased tolerance to the drug; repeated experience of withdrawal symptoms; use of the drug to prevent or relieve withdrawal symptoms) and behavioral signs of loss of control over drug use (e.g., increasing prominence of drug-seeking behavior, even at the cost of disruption of other important parts of the user's daily life; use of larger amounts than intended; inability to cut down the amount used, despite persistent desire to do so; and awareness by the user of frequent craving.

Psychic dependence or *psychological dependence* refers to those components of the dependence syndrome other than tolerance and withdrawal symptoms, in particular the urgency of drug-seeking behavior, craving, inability to function in daily life without repeated use of the drug, and the inability to maintain prolonged abstinence. It has been attributed to a distress or tension, especially during periods of abstinence from the drug, that the user seeks to relieve by taking the drug again. This is, however, really a description, rather than an explanation.

Because of these differences in definition of dependence by different authorities, the term has proven to be less clear than intended, and has not displaced the term addiction from common use. The latter carries a clearer emphasis on the behavior of the individual, rather than the consequences of that behavior, as in the concept of nicotine addiction. A committee report of the Academy of Sciences of the Royal Society of Canada concluded that the only elements common to all definitions of addiction are a strongly established pattern of repeated self-administration of a drug in doses that reliably produce reinforcing psychoactive effects, and great difficulty in achieving voluntary long-term cessation of such use, even when the user is strongly motivated to stop.

REINFORCEMENT AND ITS RELATION TO DEPENDENCE

No drug can give rise to dependence unless (1) it produces some effect that causes the user to make efforts to obtain and use the drug again or (2) it is taken frequently enough to establish a strong pattern of drug-related behavior that is resistant to eradication. The effect that leads to repetition of drug-taking is a *psychoactive effect*, that is to say, an effect that alters the user's perceptions, thoughts and emotions in a manner that is usually (but not always) experienced as pleasurable or rewarding. The various drugs that are potentially abused or addictive are all thought to act in different ways to stimulate a common nerve-cell pathway originating in the midbrain and running to the base of the forebrain, where it releases the transmitter chemical *dopamine*. This pathway is often referred to in scientific shorthand as the *reward system*, though this is probably a misnomer. Activation of this pathway leads to an increased probability that the behavior that caused the activation (in this case, the drug-taking) will be repeated or reinforced, and the drug is called a *reinforcer*. A drug must have a reinforcing effect if it is to become addictive, but it is important to recognize that reinforcement is not the same as addiction. Reinforcement is an essential mechanism for survival, learning and adaptation. The satisfaction of thirst by drinking water, and of hunger by eating food, as well as the avoidance of harm by escape, are all examples of types of reinforcement by natural and necessary behaviors. Addictive drugs are regarded as "usurpers" of the reward system that produce reinforcement by direct drug action on it without serving any necessary biological function.

Nevertheless, drug-induced reinforcement, like reinforcement by food, water, sexual activity, or escape from harm, simply means that the behavior that caused it has an increased likelihood of being repeated. Some other process or processes must enter into play if that behavior is to become so strongly entrenched that it comes to dominate the individual's thinking and activities. Various hypotheses have been put forward concerning the nature of such additional processes. One suggestion is that activation of the reward system is controlled by something analogous to a thermostat, regulating the "set-point" of the system, and that frequent repetition of drug-taking leads to a change in set-point so that reinforcement grows progressively stronger over time. Another, perhaps related, hypothesis is that the degree of reinforcement by a given drug is regulated by genetic factors, and therefore vulnerability to addiction is greater in those who inherit either an abnormally high sensitivity to the reward system or a low sensitivity to the aversive (punishing, disagreeable) effects of the drug. Another view holds that the essential feature leading to addiction is not reward (i.e., pleasure or

liking for the drug) but drug-induced sensitization of the process of *incentive saliency* (i.e., the subject's awareness of, and "wanting" for, drug-related stimuli becomes progressively greater, so that they have a steadily increased probability of controlling behavior). Yet another, and closely related, hypothesis is that drug-taking generally occurs within certain specific environmental or social contexts, and cues arising from these contexts can become linked to the drug effects as *conditional stimuli*, which then become able to elicit drug-taking behavior and further reinforcement. This is analogous to the role of the bell in Pavlov's experiments in which salivation, at first elicited by the feeding of meat to a dog, could eventually be elicited by the bell alone if the bell was always sounded just before the presentation of the meat. In this view, when the drug-taking comes under the control of such extraneous stimuli and is no longer a purely voluntary act, the transition to addiction has occurred. These various hypotheses, and possibly others, require much further research before the relation of reinforcement to addiction can be fully explained. Moreover, all such hypotheses must recognize that the degree of risk that any given individual will become addicted to a particular drug, even a strongly reinforcing one such as cocaine, is strongly influenced by environmental, social, economic and other factors.

CRAVING AND RELATED CONCEPTS

Craving refers to an intense desire for the drug, expressed as constant, obsessive thinking about the drug and its desired effects, a sense of acute deprivation that can be relieved only by taking the drug, and an urgent need to obtain it. This state is probably induced by exposure to bodily sensations and external stimuli that have in the past been linked to circumstances and situations in which drug use has been necessary, such as self-treatment of early withdrawal symptoms by taking more drug. *Drug hunger* is essentially synonymous with craving, and *urge* represents the same phenomenon but of lesser intensity.

The behavioral consequence of an urge or craving is usually a redirecting of the person's thoughts and activities towards obtaining and using a new supply of drug. All the behaviors directed toward this end, such as searching drawers and cupboards for possible remnants of drug, getting money (whether by legal or illegal means), contacting the sources of supply, purchasing the drug, and preparing it for use, are included under the term *drug-seeking behavior*. The more intense the craving, the more urgent, desperate, or irrational this behavior tends to become.

TOLERANCE AND SENSITIZATION

The term *tolerance*, which has long held a prominent place in the literature on drug dependence, has a number of different meanings. All of them relate to the degree of sensitivity or susceptibility of an individual to the effects of a drug. *Initial tolerance* refers to the degree of sensitivity or resistance displayed on the first exposure to the drug; it is expressed in terms of the degree of effect (as measured on some specified test) produced by a given dose of the drug, or by the concentration of drug in the body tissues or fluids resulting from that dose: the smaller the effect produced by that dose or concentration, the greater is the tolerance. Initial tolerance can vary markedly from one individual to another, or from one species to another, as a result of genetic differences, constitutional factors, or environmental circumstances.

The more frequent meaning of tolerance, however, is *acquired tolerance* (or *acquired increase in tolerance*)—increased resistance or decreased sensitivity to the drug as a result of adaptive changes produced in the body by previous exposure to that drug. This is expressed in terms of the degree of reduction in the magnitude of effect produced by the same dose or concentration, or (preferably) the increase in dose or concentration required to produce the same magnitude of effect. Acquired tolerance can be due to two quite different processes. *Metabolic tolerance* (also known as *pharmacokinetic tolerance* or *dispositional tolerance*) is produced by an adaptive increase in the rate at which the drug is inactivated by metabolism in the liver and other tissues. This results in lower concentrations of drug in the body after the same dose, so that the effect is less intense and of shorter duration. *Functional tolerance* (also known as *pharmacodynamic tolerance* or *tissue tolerance*) is produced by a decrease in the sensitivity of the tissues on which the drug acts, primarily the central nervous system, so that the same concentration of drug produces less effect than it did originally.

Acquired functional tolerance can occur in three different time frames. *Acute tolerance* is that which is displayed during the course of a single drug exposure, even the first time it is taken. As soon as the brain is exposed to the drug, compensatory changes begin to develop and become more marked as time passes. As a result, the degree of effect produced by the same concentration of drug is greater at the beginning of the exposure than it is in the later part; this phenomenon is sometimes called the *Mellanby effect*. A second time pattern of tolerance development is known in the experimental literature as *rapid tolerance*. This refers to an increased tolerance seen on the second exposure to the drug, if this occurs not more than one or two days after the first exposure. *Chronic tolerance* is that form of acquired tolerance that develops progressively over an extended period of time in which repeated exposure to the drug takes place. There is suggestive evidence that these three forms may involve the same or very similar mechanisms. All experimental interventions so far tested have produced virtually identical effects on rapid and chronic tolerance, and chronic tolerance is accompanied by an increase in the rate of development of acute tolerance.

Although acquired tolerance involves important physiological changes in the nervous system, it is also markedly influenced by learning. Tolerance develops much more rapidly if the individual is required to perform tasks under the influence of the drug, than if the same dose of the same drug is experienced without any performance requirement. Similarly, environmental stimuli that regularly accompany drug administration can come to serve as Pavlovian conditional stimuli that elicit tolerance as a conditional response, so that tolerance is demonstrated much more rapidly in the presence of these stimuli than in their absence.

Sensitization refers to a change opposite to tolerance, that occurs with respect to certain effects of a few drugs (most notably, central stimulant drugs such as cocaine and amphetamine, or low doses of alcohol that produce behavioral stimulation rather than sedation) when these are given repeatedly. The degree of effect produced by the same dose or concentration grows larger rather than smaller. For example, after repeated administration of amphetamine a dose that initially produced only a slight increase in physical activity can come to elicit very marked hyperactivity, and a convulsion can be produced by a dose that did not initially do so. This does not apply to all effects of the drug, however; tolerance can occur towards some effects (such as the inhibition of appetite) at the same time that sensitization develops to others. The reason for this difference is not yet known.

CROSS-TOLERANCE AND CROSS-DEPENDENCE

The term *acquired tolerance* is applied to tolerance developing to the actions of the same drug that has been administered repeatedly. However, if a second drug has actions similar to those of the first, an individual who becomes tolerant to the first drug is usually also tolerant to the second drug, even on the first occasion when the latter is used. This phenomenon is called *cross-tolerance*, and it may be partial or complete—it may extend to all the effects of the second drug, or only to some of them. The adaptive changes in the nervous system that give rise to acquired tolerance are believed by most researchers (though not all) to be responsible also for the development of physical dependence. Thus, an adaptive change in cell function, opposite in direction to the effect of the drug, will offset the latter when the drug is present (tolerance), but will give rise to a withdrawal sign or symptom when the drug is removed. The term *neuroadaptive state* has been proposed to designate all the physiological changes underlying the development of tolerance and physical dependence. If the second drug, to which cross-tolerance is present, is given during withdrawal from the first, it can prevent or suppress the withdrawal effect; this is known as *cross-dependence*. A related concept is that of *transfer of dependence*, from a first drug on which a person has become dependent to a second drug with similar effects, that has been given therapeutically to relieve the withdrawal signs produced by the first.

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ADDICTIVE PERSONALITY The term *addictive personality* has been used in various ways, most commonly to refer to a recurrent pattern observed in many alcoholics and other substance abusers: impulsivity, immaturity (dependency and neediness), poor frustration tolerance, anxiety, and depression. Many of these features disappear during extended periods of abstinence, however, suggesting that they may be either related directly to the drug use, to the life it imposes, or to social response, rather than to personality. Addictive personality—more accurately preaddictive personality—has also been used to refer to personality characteristics presumed to predate drug use and as such are predictive of such use. These aspects of personality are likely to include early difficulties in impulse control and submission to authority—and sensitivity to anxiety and depresssion.

(SEE ALSO: *Addictive Personality and Psychological Tests; Causes of Substance Abuse; Childhood Behavior and Later Drug Use; Coping and Drug Use*)

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ADDICTIVE PERSONALITY AND PSYCHOLOGICAL TESTS Psychological tests and measurements (psychometrics) are structured ways of evaluating an individual's inner mental life and external behaviors. They present subjects with more or less standard stimuli to which the subjects respond. Depending on the test, these responses tell us something about their intelligence, abilities and skills, educational and vocational interests and achievements, and personality. Often, the tests are especially helpful in diagnosing organic brain disease—its presence, presumed location, and the particular resulting functional deficits. The tests themselves range from structured questionnaires or interviews, to pen-and-pencil tasks, to obtaining responses to purposely ill-defined stimuli such as ink blots (Rorschach test). They have been used (1) to evaluate the probability of the presence of a substance-abuse problem, (2) to examine the impact of substance use on behavior and brain function both acutely and chronically, and (3) to assess personality features—profiling which ones are predisposed to use and abuse or which are the result of such use.

Historically, we have moved from the search for a single trait as *cause* to looking for a cluster of traits (the *addictive personality*), to the recognition that a number of different pathways lead to addictive behavior(s); we now understand that different types of people may use drugs for different reasons. Some underlying trait or combination of traits, however, may predispose individuals to problems with drugs. In addition, the "addictive life" may so structure behavior that it imposes similarities in attitudes, responses, and the like; it may present us with a state-related personality—that is, a pattern of typical response and behavior that is present while living in the drugged state but that disappears, in part or in whole, after a period of abstinence. For example, Vaillant's long-term population studies (1983) have shown that people diagnosed with personality disorders or other psychopathologies while drinking often appear to lose these "illnesses" after they have been alcohol-free for some time.

A review of studies of personality in alcoholics concluded that there appeared to be six constellations: (1) those who drink to escape the pain of frustration; (2) those for whom drink gratifies childish dependency; (3) those who drink to reduce guilt and anxiety; (4) those who escape disappointment into fantasy; (5) social isolates for whom alcohol supplies a pseudo-life; (6) social context-driven alcoholics. Other studies have defined additional groups, such as unsocial aggressive, psychopathic, and inhibited-conflicted. Similar findings and classifications have been described in users of other drugs. The overlapping, but not identical, or at times contradictory descriptions can be accounted for at least partly. Each study has differed from the others in obvious ways: different measures of personality (e.g., the MINNESOTA MULTIPHASIC PERSONALITY INVENTORY or the Rorschach Inkblot test); different subject populations (e.g., ALCOHOLICS ANONYMOUS members or hospitalized patients); different comparison groups; or different statistical analyses of the data. Future research in the understanding of addictive personality(ies) needs to define in advance each of these parameters and build in true replications.

While psychometric studies of personality have so far led to contradictory or confusing findings, they have proven useful in other ways: A variety of structured questionnaires are good screening devices and help the clinician toward further inquiry concerning alcohol or other drug use; tests of organic brain function have identified both acute and lasting effects of a wide variety of drugs; and the tests have helped us identify some of the common accompanying psychopathologies, such as antisocial personality disorder and depressive illness.

(SEE ALSO: *Causes of Substance Abuse; Conduct Disorder and Drug Use*)

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ADJUNCTIVE DRUG TAKING Drug abuse is usually viewed as a behavior that occurs because of what the drugs do in the body. Most people assume that drug abuse is mainly driven by the immediate, attractive effects and sensations produced by the drugs—their physiological and psychological effects. But there is another, less direct way drugs can become of overwhelming importance to a person and dominate life. Excessive drug use can develop as a side effect of other strongly desired behavior that, for various reasons, cannot be engaged in or completed.

For example, for any one of a number of reasons a person may not be able to do something they are motivated to do: They may not be permitted to do it, the goals of the behavior may not be available to them, or the person may not have the skills or knowledge required to perform the behavior successfully. When blocked in any of these ways from performing a desired behavior, a person may turn aside to some sort of easy, satisfying alternative—an adjunctive behavior. The following is a description of the first kind of *adjunctive behavior* that was produced in an experimental laboratory. It does not involve drug taking, but it has many of the exaggerated and compulsive characteristics usually thought to be a result of drug use.

Normal, adult laboratory rats were first reduced in body weight; then it was arranged for them to receive much of their daily food ration in individual chambers. The food was given as 45 milligram pellets, and each pellet was available only about once per minute over a 3-hour period each day. Although water was always freely available back in each animal's home cage, this schedule of intermittent food availability, in which the eating of small food portions was spread out over a 3-hour period, produced a strange result. When an animal received a food pellet, it quickly ate it and then took a large drink of water. Since an animal received a total of about 180 pellets during each daily, 3-hour session, and drinking occurred after almost every pellet, by the end of the 3 hours an animal had drunk an amount of water equal to about one-half its body weight. That is an excessive amount, and the overdrinking occurred day after day during each daily session. It seemed compulsive, for the animals did not lose interest in drinking water heavily after a few days. The exaggerated drinking that occurred under this feeding condition did not disappear as long as the intermittent schedule of feeding remained in force.

It was easy to prove that the excessive drinking was not a physiological effect of reducing the daily intake of food: If, instead of being given pellets spread across the session, rats were given the same 180 pellets all at once as a single ration, then they drank about 10 milliliters of water over the next 3 hours, rather than almost 100 milliliters. So, the rats did not really need the excessive amount of water they drink under the schedule of intermittent pellet delivery. Something about doling out bits of food to them over time drove the exaggerated drinking behavior. They got what they needed of something of crucial value (in this case, food), but the individual portions were rather small, and there was a time delay between these portions.

The details of how the size of the portions, the amount of time between them, as well as the state of food deprivation, affects the degree of excessive drinking have been worked out in many experiments. The details of these relations will not be considered here, but a few additional facts indicate the widespread nature of this excessive-behavior phenomenon. As described, this overdrinking of plain water is referred to as *schedule-induced polydipsia*. The term *polydipsia* means the food schedule can induce excessive drinking of many sorts of fluids. But in general, behaviors that become exaggerated under these sorts of conditions are known as *adjunctive behaviors*.

The phenomenon of adjunctive behavior is not limited to the rat species and it can be induced in ways other than food-schedule intermittency. And adjunctive behaviors other than drinking can occur. Adjunctive behavior can be induced in many animal species, for example, mice, monkeys, pigeons and chimpanzees, as well as in humans. The behavior can be induced by a *generator schedule* that is not based on doling out food; other kinds of incentives also are effective in inducing excessive behavior. For example, humans reinforced intermittently with money, by the opportunity to gamble, or even by maze solving, showed adjunctive increases in fluid intake, general activity, eating or smoking. In animal experiments, excessive aggres-

sion, overactivity, and eating are a few of the behaviors that occurred adjunctively owing to the intermittent availability of some important commodity or activity.

The adjunctive behavior of greatest interest with respect to the problem of drug abuse is, of course, the excessive seeking and taking of drugs. A few examples will be given. But first, as indicated above, it is important to understand that adjunctive drug taking is only one kind of excessive behavior that can be driven by a generator schedule. Thus, drug abuse is just one sort of adjunctive behavior; it is not a special problem driven exclusively by the pharmacological actions of drugs of abuse.

Once research had established the conditions inducing adjunctive drinking, it was of interest to determine if fluids other than water would be drunk to excess, especially drug solutions. Briefly, if the drug concentration of a drinking solution is not too high (a high concentration is usually too bitter), a much greater unforced (voluntary) oral drug intake can be induced by a generator schedule than would occur otherwise. Several classes of drugs have been investigated, and excessive intakes of alcohol, opioids (e.g., morphine), sedatives (e.g., barbiturates), anxiolytics (e.g., benzodiazepines), stimulants (e.g., cocaine), and other agents (e.g., nicotine) have been sustained for many months under these inducing conditions. For some of these agents, physical dependence results from the excessive amounts ingested by animals. However, if the generator schedule is discontinued, drug intakes decrease immediately to much lower levels. Thus, without the generator schedule, the high drug intakes do not continue. Similar generator schedules (intermittent food delivery) also can induce excessive intravenous drug self-administration.

These and related studies clarify the nature of what sustains excessive intake of drugs (drug abuse). It is important to note that, by this analysis, drug abuse arises from a background of excessive behavior that is induced by a generator schedule. It is produced by the limited and intermittent availability of crucially important environmental events. Such schedules are similar to conditions in natural and social environments that provide commodities we need, and activities we desire, but only in little bits at a time, and with delay intervals in between the bits. The adjunctive behavior generated may be largely noninjurious, like water drinking. Or the adjunctive behavior may be creative, like an intense hobby. But it also can result in aggression or drug taking, depending upon personal history, the skills one possesses, and currently available opportunities.

The pharmacological consequences of drug taking are only one factor sustaining drug abuse. The environmental generating conditions and the context of alternative opportunities are of crucial importance. Drug abuse occurs out of conditions already generating behavioral excesses. Although drug abuse often is described as if it is a direct consequence of exposure to a drug with abuse potential, the great majority of people experimenting with such drugs do not become abusers—they simply lose interest and turn to other pursuits. (This is not to approve of such hazardous experimentation.) Those self-administering such drugs for medical reasons (opioids are prescribed on a long-term basis for controlling chronic pain) almost never become drug dependent or motivated to abuse these agents.

Adjunctive behavior studies teach us that drug abuse stems more from environmental generating conditions, together with a lack of, or poor utilization of, other opportunities, than from any overwhelming intrinsic attractiveness of agents with abuse liability. A few drug-abuse situations can be described briefly to clarify how drugs come to be so attractive to some individuals, and what causes this behavior to persist in the face of the trouble it causes for them.

An example frequently given is that of a poorly educated youth, living in an urban ghetto, with minimal job prospects, who deals in drugs for economic reasons, and comes to use them owing to the sparse schedule of conventional opportunities and satisfactions available in that environment. Although that may be true, drug abuse is a problem that occurs not just for disadvantaged persons. The conditions of life for the affluent rich do not strike us as being extreme conditions that can lead to drug abuse. But consider the overprivileged young person, sent to a superior boarding school by parents with great expectations (i.e., demands), but with little time to provide their children with direct social reinforcement. They may be too engaged with their own lives, management responsibilities, and range of personal advantages, to afford their children much of their valuable time. The lean schedule of social reinforcement for the children, the often competitive nature of social interactions in

private schools, the young person's migrant status in a new or isolating school, together with a high disposable income, can lead to engaging in drug abuse, particularly if this activity gains one local power and social status, while the threat of enduring legal consequences is negligible. And the person, although perhaps expected to take over the family business, together with its social obligations, is not yet empowered to do so, or to have their opinions taken seriously, so that their current social status is weak even if they appear privileged.

Consider a sales representative who travels for a corporation, making intermittent sales agreements (which may be subject to cancellation), with little effective influence on company policies or politics, with little to do in brief stop-overs in strange towns. Given the demand characteristics of the job, the uncertainty and intermittency of reinforcement, and the sparse opportunities for creative efforts when "on the road," a person so exposed may be vulnerable to drinking too much in the easy ambiance of hotel bars.

Studies on the schedule-induced production and chronic maintenance of excessive intake for a variety of drugs indicate that drug abuse is a problem that has its roots in the behavioral effects induced by environmental conditions, as well as in the pharmacological consequences of these elevated intakes. The exaggerated and problematic behavior designated as drug abuse is not behavior that is specific to, or the outcome of, a person's interaction with drugs. It is one possible adjunctive outcome of a set of conditions that comprise economically and/or socially restricted schedules of reinforcement that generate and sustain a host of possible exaggerated and persistent behaviors.

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ADMINISTRATIVE AND PUBLIC HEALTH LAW Civil remedies are defined as procedures and sanctions, specified by civil statutes and regulations, used to prevent or reduce criminal problems and incivilities (Mazerolle & Roehl, 1998). Drug control is a primary application of many civil remedy programs. Police departments, city prosecutors, and community members use civil remedies in an effort to disrupt illegal activities at drug-selling locations. This approach to drug control typically targets nonoffending third parties (e.g., landlords, property owners) and utilizes nuisance and drug abatement statutes. These types of abatement statutes include repair requirements, fines, padlocks/closing, and property forfeiture and seek to make owners and landlords maintain drug- and nuisance-free properties. Police often work with teams of city agency representatives to inspect drug nuisance properties, coerce landowners to clean up blighted properties, post "no trespassing" signs, enforce civil law codes and municipal regulatory rules, and initiate court proceedings against property owners who fail to comply with civil law citations.

Growth in the use of civil remedies as a crime control or crime prevention tactic is attributable to several factors. First, the accessibility of civil remedy tools provides frustrated and disadvantaged communities with alternative avenues to reverse the spiral of decline. Second, the increasing use of civil remedies comes at a time when communities

and law enforcement officials recognize that many criminal remedies are neither effective nor desirable for a wide range of problems. Third, growth in civil remedy approaches to crime control coincides with increasing societal emphasis on prevention.

Many civil remedy actions seek to reduce signs of physical (e.g., broken windows, graffiti, trash) and social (e.g., public drinking, loitering, public urination) incivilities in the hope that cleaned-up places will break the cycle of neighborhood decline and subsequently decrease victimization, fear of crime, and alienation. Code enforcement, drug nuisance abatement, neighborhood cleanup and beautification, and Crime Prevention Through Environmental Design (CPTED) interventions comprise civil remedy actions that are typically used to control drug problems. Youth curfews, gang injunctions, ordinances controlling public behavior, and restraining orders are other examples of civil remedies that seek to alter criminal opportunities and prevent drug-selling problems from escalating.

Pressures on property owners and managers often result in corrections of health and safety violations, enforced cleanup and upkeep efforts, evictions of problem tenants, and improved property management. Bans on drug paraphernalia, alcohol-related billboard advertising, spray paint, and cigarette machines in high crime areas are used in an attempt to disrupt the illegal activities at drug-selling locations. Injunctions against gangs, youth curfews, and domestic violence restraining orders are used to prevent and deter potential perpetrators from engaging in criminal behavior. When useful civil statutes are absent, community groups, legislators, and policy makers often work together to enact new legislation.

Unlike traditional criminal sanctions, civil remedies attempt to resolve underlying problems: the motel's poor management, the absentee owner's neglect. The use of civil remedies tends to be proactive and oriented toward prevention, whereas, at the same time, civil remedies aim to enhance the quality of life and eliminate opportunities for problems to occur or reappear.

Police use existing public health and controlled substances acts to send warning letters to property owners informing them that complaints of problem activities (e.g., drug dealing) have been reported on their property, advise them of steps to take in preventing or minimizing the problems, and offer assistance in resolving the problem. The letters serve as an official notice of drug activity. Fines and other civil penalties may occur if violations are not corrected, and there are fees for reinspections to cover city costs. If owners do not correct the problem, there are penalties that include fines, closure of the property for up to one year, and sale of the property to satisfy city costs. The city attorney's office can file suit against owners who do not take responsibility for their property.

Civil remedies offer an attractive alternative to criminal remedies since they are relatively inexpensive and easy to implement. Citizens can make a difference by documenting problems, pressuring police and prosecutors to take appropriate civil action, or spearheading drives to establish useful local ordinances. A group of neighbors can pursue a nuisance abatement action in small claims court without the assistance of police or public prosecutors (Roehl, Wong & Andrews, 1997). Moreover, civil laws require a lower burden of proof than criminal actions and do not involve the requirements of due process, making them easier to apply yet open to concerns about fairness and equity (Cheh, 1991).

The use of civil remedies to solve crime and disorder problems continues to grow in popularity. Police regularly use civil laws, local city regulations, and ordinances to control drug, disorder, and other crime problems. Community groups often work with policy makers to instigate civil remedy action to solve intractable neighborhood problems. The civil remedy approach appears to be an effective and relatively cost-effective approach to control drug problems (Mazerolle, Price & Roehl, 2000).

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ADOLESCENTS AND DRUG USE As individuals pass through adolescence, they undergo many physical, cognitive, social, and emotional changes. Most learn to adapt to these changes in healthy ways. For others, turmoil, conflict, and deviant behavior lead to upheaval and disorganization as they attempt to cope. Drug use as a behavior may serve many functions in this attempt to cope, and it can have many consequences. A single episode of drug use does not necessarily lead to further use—but several episodes may lead to ever increasing use, with abuse and dependence the result.

Use of a drug, age of first use, and reasons for use are all factors related to continued drug use. Early adolescents who try one type of drug may venture on to sample a diverse number of substances. This can lead to regular use of certain drugs (e.g., daily cigarette or MARIJUANA smoking); it may become part of a pattern of multiple drug use (e.g., weekend drinking and smoking or daily uppers and downers) that by late adolescence becomes dependence or abuse. Factors related to initiation and progression into other drug phases—regular drug use, abuse, and dependency—or into the use of multiple drugs are important to understand in order to develop appropriate PREVENTION programs aimed at reducing all drug use—whether legal or illicit.

REASONS FOR DRUG USE

For both pharmacological and psychological reasons, an adolescent who tries a particular type of drug is more likely to use that substance again if he or she enjoys the drug's effects. However, if unpleasant experiences are associated with the use, trying it again is less likely.

Because the body becomes accustomed to the effect of a drug, often the drug amount will need to be increased in order to obtain an effect. This phenomenon is known as *tolerance*, and once tolerance to a drug develops, the level of drug use may escalate into larger and larger doses. Continued use

Teenagers share a joint during an annual marijuana legalization rally in New York City's Washington Square Park, May 4, 1996.
(AP Photo/Bebeto Matthews)

of drugs may also occur because of unpleasant symptoms—withdrawal—that may appear as the drug (e.g., heroin, nicotine, caffeine) begins to wear off. To avoid these withdrawal symptoms, a user may feel compelled to establish a regular pattern of use, possibly resulting in physical dependence.

The way a drug is used is also a factor in developing tolerance and physical dependence. For example, an adolescent who sniffs COCAINE may find that the amount he or she has to inhale to get the desired effects becomes enormous. Because of this, the user may switch to injecting the cocaine instead of inhaling it. This new route of administration exposes the user to a more potent form of the drug as well as to increased medical complications.

Other reasons that adolescents continue using a particular drug may be socially and environmentally driven. Teenagers looking for peer acceptance or wanting to appear "cool" or mature might decide to use drugs. For example, although the use of TOBACCO and ALCOHOL is illegal for adolescents, it is both legal and socially acceptable for adults. ADVERTISING, the media, and role models portray drinking and smoking as desirable. Associating and socializing with peers who are using drugs provides an opportunity for access to drugs that can encourage experimentation and ongoing use.

Researchers have investigated the influence of parents and the family environment on children's alcohol and drug use, dysfunctional patterns of coping, and delinquent activity. In one study, a large group of New Jersey adolescents was interviewed by phone at two different times, three years

apart. Between 1979 and 1981, 1,380 subjects aged 12, 15, and 18 were interviewed. Three years later, 95 percent of them (1,308 subjects) were interviewed again. The interviews included topics of family harmony and cohesion, parenting styles, and the attitudes and behaviors of parents. The results showed that the alcohol consumption of the younger children was influenced by the alcohol use and attitudes of the parent of the same gender as the child. Older adolescents, though, were most strongly affected by the father's alcohol use. Parental hostility and lack of warmth toward the children was associated with use of drugs and alcohol among adolescents (Johnson & Pandina, 1996).

A national household sample of 4,023 adolescents aged 12 to 17 years was interviewed by telephone about substance use, victimization experiences, familial substance use, and posttraumatic reactions to identify risk factors for substance abuse or dependence. A major finding was that adolescents who had been physically assaulted or sexually assaulted, who had witnessed violence, or who had family members with alcohol or drug use problems had increased risk for current substance abuse or dependence (Kilpatrick et al., 2000).

Data from the Centers for Disease Control and Prevention Youth Risk Behavior Survey (YRBS) was used from 4,800 subjects to examine the relationship between adolescents' employment and substance abuse behaviors. The study concluded that among public high school students with extracurricular jobs, those who worked above 15 hours per week appeared to have an increased risk for substance abuse (Valois, 2000).

A study that examined the effects of family structure and family environments on the initiation of illicit drug use among a sample of Hispanic, African American, and white adolescent boys found large differences in family structure among the three groups. African American adolescents reported the lowest incidence of illicit drug use initiation, and the weakest effects of family structure and environment on substance use. Deteriorating changes in family environments were stronger predictors of the initiation of drug use among Hispanic immigrants than nonimmigrants, and family socioeconomic status was a predictor for immigrant Hispanics only. For all groups, the accumulation of family risk factors was a stronger predictor of illicit drug initiation than family structure (Gil, 1998).

DRUG-USE SEQUENCE

The use of one drug is often related to the subsequent use of another. Typically, drug use begins with alcohol and cigarettes, which are followed by marijuana and other illicit drugs. This typical sequence of drug use was established in the 1970s (Kandel & Faust, 1975) and was found to continue into the 1990s, in different populations and in different ethnic and cultural groups. Problem drinking typically fits into the pattern between ongoing marijuana use and the use of other illicit drugs (Donovan & Jessor 1983).

Cocaine use tends to follow marijuana use, with crack-cocaine use occurring after cocaine use (Kandel & Yamaguchi, 1993). For example, it is likely that someone who smokes CRACK has already tried tobacco, alcohol, marijuana, and cocaine. Many adolescents who use drugs in one category, however, do not necessarily progress to drug use in a "higher" category; many stop before becoming involved in further use or habitual use.

An important factor in the progression through the sequence of drug use is age of onset or initiation. The use of alcohol and cigarettes typically—but not always—begins at an earlier age than does the use of illicit drugs. Adolescents who progress to using illicit drugs such as crack generally begin smoking and drinking earlier than those who do not. Early drug use (before age fifteen) is highly correlated with the development of drug and alcohol abuse in adulthood (Robins & Przybeck, 1985).

Studies of adult populations provide additional support for a connection between regular adolescent drug use and later, further drug use. For example, illicit drug use during adolescence and early adulthood has been found to occur more often in adults who have used psychotherapeutic medicines (e.g., tranquilizers, sedatives) (Trinkoff, Anthony, & Muñoz, 1990). Studies of people going to drug-treatment centers often demonstrate that these people are not only entering treatment for use of substances such as cocaine or heroin but that they are also addicted to caffeine, tobacco, and/or alcohol, the very substances they first started using.

MULTIPLE-DRUG USE

The use of one type of drug may lead to experimenting with other drugs as users add to or find substitutes for their original drug of choice. Some

of this progression may be an effect of maturation—that is, of drug-using adolescents moving on to other drugs as they grow older—or it may be attributable to the cost and availability of different types of drugs, introduction to new substances by drug-using peers, or drug-seeking behavior in which individuals continue trying different drugs until they achieve the desired effect. Polydrug (multiple-drug) use can also occur when people try to counteract the effect of one drug by the use of another—for example, by taking tranquilizers (which relieve symptoms of anxiety) in order to counteract the anxiety-producing effects of cocaine.

RESEARCH METHODS

Much of the most useful data for studies of adolescent drug use are collected either by self-administered questionnaires or during interviews. Often the questionnaires are given out in classrooms for students to complete anonymously. This type of study, which obtains data from all respondents at the same time, is known as a cross-sectional survey, and it provides important clues about how the use of different substances relates to such factors as age, gender, and ethnicity. In the drug-sequencing studies, researchers collect information as to whether a substance was used and the age of the person at the time of first use. Then, using a statistical technique called Guttman scaling, they combine each drug category and the ages of first use for the entire sample to establish a predominant sequence of use, by age, for the different substances.

Longitudinal cohort study is the term used for the study design whereby researchers test the progression of drug users to stronger substances. In these studies, people are interviewed or given a questionnaire to fill out repeatedly over time. For example, the same youths may be contacted annually to provide information on their drug use during the year. Although this method may help establish the correct timing and order of drug-use initiation for any given individual, it is a very difficult approach because of the cost and time involved in tracking people for many years.

Since illegal drug use has an antisocial connotation, people may underreport their use, although some teenagers may exaggerate reports of their drug use to create an impression. The biggest hurdle in studying drug use is obtaining accurate information. Reports are assumed to be honest and correct, based on the respondents' memory. Researchers try to promote honesty and accuracy by providing memory aids (e.g., pictures of drugs) as well as by assurances of anonymity and confidentiality.

Another concern of researchers is that reports of drug use will be affected by the way the population is sampled or by those participating. For example, a survey conducted in an inner-city public school may not reflect all adolescents. High school dropouts who are not in class when the data are collected and students enrolled in private schools may have levels of drug use that are different from those of students attending public schools.

PREVENTION IMPLICATIONS

Adolescent drug use must be considered in relation to the normal developmental challenges of adolescence. Because individuals use drugs in different ways for many reasons, no single prevention program will be effective with all groups at all ages. Understanding the factors that determine the link between the usage of one drug to the usage of another has important policy implications for developing prevention and educational programs. The sequential nature of drug use, as it is now understood, would indicate that prevention efforts targeted toward reducing or delaying adolescents' initiation into use of alcohol and cigarettes would reduce these adolescents' use of marijuana and other drugs. Similarly, efforts targeted toward reducing adolescents' marijuana use might reduce the rates of these adolescents' progression to "higher" stages of drug involvement. Prior drug use is a risk factor for progression; that is, the use of one drug may increase the likelihood of use of another drug, but it is not in itself a cause of further progression.

Data from the National Longitudinal Study on Adolescent Health reveal that there are many factors that determine whether teenagers will be predisposed to engage in harmful behaviors. The survey of 12,118 teenagers found that teenagers who felt close to their parents and siblings, teachers, and classmates were less likely to engage in risky behaviors. (Resnick, 1997)

Educating young people on the dangers of drugs has had some measurable success. A school-based

series of classes on the dangers of anabolic steroid use appeared to help reduce steroid use among teenage athletes. Researchers evaluated seven weekly, 50-minute classes that gave 702 teenage football players comprehensive education in the dangers of steroids and alternatives to their use. This intervention improved the athletes' ability to drop steroid use when compared to a group of 804 athletes who just received a pamphlet about steroids. Athletes often use steroids to boost their performance, but the drugs can have dangerous side effects. (Goldberg, et al., 1996)

TRENDS

The annual survey of nearly 50,000 students around the country, "Monitoring the Future," conducted at the Institute for Social Research at the University of Michigan, reported in 1999 that, after declining in recent years, drug use among American teens generally held steady. However, there were slight increases in adolescents' use of anabolic steroids and the drug ecstasy. The report also noted that teen tobacco smoking dropped slightly but was still well above rates of the early 1990s. Drugs that showed little change in use included marijuana, amphetamines, hallucinogens, tranquilizers, and heroin. The only significant decline was in the use of crack cocaine among eighth and tenth graders, after several years of gradually increasing use. The Michigan study had been tracking high school seniors for 25 years and following eighth and tenth graders for the previous nine years. The survey included 45,000 students from 433 schools across the country. Researchers pointed out that drug use rates were down from the peak levels in overall illicit drug use by American teenagers that were reached in 1996 and 1997.

(SEE ALSO: *Conduct Disorder and Drug Use; Coping and Drug Use; High School Senior Survey*)

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ADULT CHILDREN OF ALCOHOLICS (ACDA) Taken literally, this term indicates people over the age of eighteen, who have at least one biological parent with severe and repetitive life problems with alcohol. Because of their genetic and familial relationship to an alcoholic, these people

carry an increased risk of severe alcohol problems themselves (a probability of two to four times that of children of nonalcoholics). Probabilities also indicate that they are not more vulnerable to severe psychiatric disorders (such as schizophrenia or manic depressive disease) and that they do not carry a heightened risk for severe problems with some drugs of abuse (such as heroin). Nevertheless, it is possible that when children of alcoholics reach adolescence or adulthood, they might be slightly more likely to have problems with marijuana-type drugs or with stimulants (such as cocaine or amphetamines). It has also been observed that if their childhood home has been disrupted by alcohol-related problems in either or both parents, the children may have greater difficulties with a variety of areas of life adjustment as they mature or go off on their own.

The label Adult Children of Alcoholics has been given to a self-help group, often abbreviated as ACOA. Within the group, people with at least one alcoholic parent can meet with others for discussion, the sharing of old and current experiences, and the chance to find interpersonal support—which helps place their own individual experiences into perspective. People who join this voluntary organization are likely to be those who both feel impaired and seek help toward coping with their past and/or present problems.

The term ACOA has also been thought to describe a group of characteristics of individuals who grew up with alcoholic parents in the home. There are research projects that do indicate that such men and women have a greater than chance likelihood of having problems expressing their feelings, feeling comfortable with intimacy, or in maintaining long-term relationships including marriages. There are less data to support conclusions regarding a possible association between an ACOA status and problems with developing trust, impairment in feelings of well-being and achievement, enhanced feelings of a need to rescue other people when they are in emotional or psychological distress, excessive feelings of a need to control a situation, and a more global dissatisfaction with life and current situations than would be expected from chance alone.

While common sense would dictate that being raised in the home of an alcoholic individual might contribute to problems with intimacy and cause levels of psychological discomfort, most of the existing studies do not control for important factors in attempting to draw conclusions. For example, it is well established that children of alcoholics are much more likely to develop alcoholism themselves, with the possibility that many of the characteristics being described relate to the consequences of their own alcohol problems as they developed. A second problem is the variety of backgrounds that can contribute to an alcoholism risk, with the possibility that severe impulse-control disorders or the presence of an ANTISOCIAL PERSONALITY disorder in the parents could have been associated with passing on several biological characteristics to *some*, but certainly not all, children of alcoholics. In this instance, the characteristics described would relate to the associated disorder, such as the antisocial personality disorder, rather than the childhood experiences. In considering these factors, it is also important to remember that a substantial proportion, perhaps a majority, of children of alcoholics never develop alcoholism, are not likely to join ACOA groups, and appear to demonstrate many personality and behavioral characteristics that resemble those of individuals who do not have alcoholic parents.

In summary, the term adult children of alcoholics has a variety of meanings. First, biological children of alcoholic parents carry a two-to-four-fold increased risk for alcoholism themselves. Thus, this designation is an important risk factor for the future development of alcoholism. Second, the abbreviation ACOA relates to a self-help group where a minority of children of alcoholics, especially those who expressed levels of discomfort, have joined together to share experiences and offer support. The third and least meaningful definition of adult children of alcoholics relates to a variety of inadequately studied personality characteristics that might relate to the childhood environment in which an individual was raised, might be a result of additional psychiatric conditions among the parents, might reflect general factors associated with a disordered childhood home but have nothing specifically to do with alcoholism, or might relate to specific alcohol-related experiences in the childhood home.

(SEE ALSO: *Al-Anon; Codependence; Conduct Disorder and Drug Use*)

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ADVERTISING AND THE ALCOHOL INDUSTRY The beverage alcohol industry includes companies that market beers and brews (malt liquors), wines and sparkling wines (fermented), and distilled spirits—whiskey, vodka, scotch, gin, rum, and flavored liquors. Sales of these products, usually through distributors, are limited to those businesses that have obtained special licenses to sell one or more of the above categories of products. For example, if a restaurant has only a license to serve beer and wine, it cannot serve other types of alcoholic beverages.

In the United States, alcoholic beverages and tobacco products are the only consumer goods that are legally restricted for sale only to those who are not minors—at least 21 years of age in the case of alcohol or 18 (19 in three states) in the case of tobacco. Sales to anyone under those ages, respectively, are illegal, yet every day thousands of minors buy beer, wine coolers, cigarettes, and snuff with no questions asked by store clerks or owners. Even if a store refuses to sell to minors, they can usually find a vending machine or ask an older friend to buy for them.

ALCOHOL USE BY AMERICANS

In a survey conducted in 1996, 109 million Americans age *12 and older* had used alcohol in the previous month (51% of the population). About 32 million people (15.5%) engaged in binge drinking, defined as five or more drinks on the same occasion. Of these, about 11 million Americans (5.4%) were heavy drinkers, defined as five or more drinks on the same occasion on at least five different days in the past month.

The percentage of college freshmen who say they drink beer frequently or occasionally was 51.8 percent in 1998. Among all college students, the overall binge-drinking rate has stayed constant between 1993 and 1999 at 44 percent, but frequent binge drinkers rose from 20 percent in 1993 to 23 percent in 1999.

The percentage of high school seniors who reported having five or more drinks in a row in the last 2 weeks was 30.8 percent in 1999, up from 27.5 percent in 1993.

The Bureau of Alcohol, Tobacco and Firearms (ATF), in 2000, estimated that 14 million U.S. residents suffer from alcohol abuse and dependence, and 76 million are affected by the alcoholism of a family member.

The illegal use of alcoholic beverages by teenagers has generated a high level of concern on the part of health-care professionals, police, parents, and activist groups such as Mothers Against Drunk Driving (MAAD) and the Center for Science in the Public Interest (CSPI).

Another cause for concern is that the level of alcohol use in 1996 was strongly associated with illicit drug use. Thirty-one percent of the heavy drinkers were current illicit drug users; 16 percent of the binge (but not heavy drinkers) were illicit drug users; 5.3 percent of the other drinkers, but only 1.9 percent of the nondrinkers, were illicit drug users.

THE ADVERTISING PROBLEM

The high level of alcohol use by those under the age of 21 creates an advertising problem for the

companies that market alcoholic beverages. How do you advertise to the 21-and-over group and also appear not to be appealing to the under-21 group? Since teenagers have a very strong desire to grow up fast, or at least participate in activities they view as *adult*, they are very vulnerable to anything they believe would help them achieve adulthood.

Critics accuse the alcoholic-beverage companies of making their advertising and promotional programs inviting to teenagers, who are already very receptive to the ideas of engaging in adult activities, being successful, being more confident, and being more attractive to the opposite sex. The alcoholic beverage companies respond that they follow the industry voluntary advertising guidelines and do *not* target teenagers. They also point to programs like the public service initiatives sponsored by America's beer industry, which encourages drinkers to "know when to say when," "drink smart or don't start," "think when you drink," or "drink safely."

WHO MINDS THE STORE?

The U.S. Bureau of Alcohol, Tobacco, and Firearms (ATF) in the Department of the Treasury is the federal agency with responsibility for overseeing the alcohol industry. Its rules discourage advertising claims that are obscene or misleading, as well as those that associate athletic ability with drinking. Also, the ATF takes the position that "unqualified health claims on products that pose increased health risks are deceptive."

Alcoholic beverages sold in the United States have to carry a warning on the container that states: "GOVERNMENT WARNING: (1) According to the Surgeon General, women should not drink alcoholic beverages during pregnancy because of the risk of birth defects. (2) Consumption of alcoholic beverages impairs your ability to drive a car or operate machinery, and may cause health problems."

Until the 1990s, the alcohol content of beer could not be included on the labeling of the container or in any associated advertising. As a result of a suit by Adolph Coors Co., a federal court decision overturned this restriction on labeling, and so companies are permitted to label their beers and malt liquors with the alcohol content. Beer averages 5 percent alcohol, ales average 6 percent, malt liquors average 4.1 percent, wine 12 percent to 20 percent and distilled spirits from 40 percent (80 proof) to 50 percent (100 proof). Beer is usually sold in 12-ounce containers, whereas malt liquors are usually sold in 40-ounce bottles.

The Federal Trade Commission (FTC) also reviews advertising, with emphasis on instances of false or misleading ads. Neither the ATF nor FTC has been very aggressive in challenging ads that seem to be targeted to young drinkers or encourage heavy drinking.

The Food and Drug Administration (FDA) in the Department of Health and Human Services has no jurisdiction over alcohol advertising, with the exception of wines with less than 7 percent alcohol. Unlike pharmaceuticals, there is no mandate that labels or advertising materials for alcoholic products provide a listing of the risks/consequences, as well as the benefits. Americans regularly see ads, company logos, and billboards that encourage people to drink, but such advertising fails to provide information about the down side of drinking, especially excessive drinking.

WHAT IS ADVERTISING

Merriam-Webster's Collegiate Dictionary defines the verb *advertise*: "to call public attention to especially by emphasizing desirable qualities so as to arouse a desire to buy or patronize," The noun *advertising* includes "by paid announcements." The broad umbrella of advertising—in addition to television, radio, and print media—uses billboards, point-of-purchase signs and displays, and increasingly, sponsorship of special events such as music festivals; auto, bicycle, and boat racing; and other sports.

ROLE OF ADVERTISING

Advertising is used as a major tool in marketing. When a company first introduces a new product, the goals generally are:

1. To inform potential purchasers that a particular product is available and why they might like to try this new product.
2. To persuade people that they should go out and buy the product.

3. To let people know where the product can be purchased.
4. To reassure people who buy the product that they have made a wise choice in doing so.

Where more than one company sells products in a given category, the goals generally become the following:

1. To increase market share by taking business away from a competitive product. This can be done by offering a better product or a better value and/or by increasing the level of advertising and promotion to out-shout the competition.
2. To increase the size of the market by inducing more people to start using the product. In the case of alcoholic beverages, this can be done by aggressively promoting features that will appeal to the potential purchaser, that is, makes you more confident, more outgoing, more appealing to the opposite sex, and, in the case of minors, leads to participation in adult-type activities.
3. To increase the size of the market by inducing people to increase their usage of your product(s). This can be done by tying the product to occasions such as spring break and by promoting the product heavily to the target audiences.
4. To keep reassuring heavy drinkers that they are in good company by drinking the particular brand of beer or liquor being advertised. Since the ten percent of those who drink most heavily account for about 50 percent of all alcohol consumed in the United States, this is a very important reason to advertise.

ADVERTISING EXPENDITURES

The alcoholic-beverage companies spend between $1 and $2 billion each year in the print and broadcast media to advertise their products. In 1998, Anheuser-Busch Co., spent $630 million, Adolph Coors Co. spent $351 million, and Seagram Co. spent $461 million. An estimated $1 billion more was spent on other alcohol-related advertising and promotional programs.

Brewers and beer distributors spent many millions of dollars sponsoring sporting events, rock concerts, spring break promotions, and other activities heavily oriented to students on college campuses. They were also heavy advertisers and supporters of baseball, football, racing events, and concerts or other cultural events.

CORRELATION OF ADVERTISING TO CONSUMPTION

In August 1993, the NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM (NIAAA) had this to say about alcohol, media, and advertising: The effects of mass communications in either promoting or preventing alcohol consumption and the problems associated with it are equivocal. Alcohol advertisements and broadcast media programming have been found to encourage a favorable view of alcohol use. Yet studies provide only modest support for the hypothesis that favorable presentations lead to positive attitudes and distorted perceptions, and consequently to increased consumption, particularly among youthful viewers. The effects of advertising bans and linkages between advertising expenditures and per capita consumption also appear to be weak and inconsistent.

There is, however, a general feeling that advertising, including all of the other promotions associated with it, plays a significant role in creating an image of desirability as far as the use of alcohol is concerned. Recent reviews in the literature (Atkin 1995; Lastovicka, 1995; Grube, 1995) show a large body of research indicating that exposure to or awareness of advertising contributes to an increase in drinking. For example, Atkin et al. (1983) found that greater exposure to advertising stimulates drinking, excessive drinking, and drinking and driving (or riding with a driver who has been drinking).

Although the majority of research supports an association between advertising and consumption, researchers do not agree on the magnitude of advertising's contribution to heavy drinking. G. Frank and G. Wilcox (1988) reported: Analysis of the results reveals no significant relationship between total advertising expenditures and consumption of beer. Significant relationships were found, however, between consumption of wine and distilled spirits and their advertising. It is emphasized that the relationships are correlational, not necessarily causal.

BEVERAGE ALCOHOL PER CAPITA CONSUMPTION

In the United States, per capita consumption of all alcoholic beverages combined reached its peak in 1980 to 1981; that of wine did not reach its peak

until 1986. From 1980 to 1989, there was a 12 percent decrease in per capita ethanol (drinking alcohol) consumption—the only sustained decrease since Prohibition—down to 2.43 gallons per person. The greatest decrease was seen in the consumption of distilled spirits. The U.S. Department of Health and Human Services has an objective for the year 2000: To reduce the per capita alcohol consumption to no more than two gallons of ethanol (drinking alcohol) per person annually.

BEVERAGE ALCOHOL SALES

Beer ranks fourth (behind soft drinks, milk, and coffee) in per capita consumption of any kind of beverage, a position it has held for many years. Beer sales, at retail in 1999, were reported by *Beverage World* to be $54.2 billion, compared to $54.3 billion for soft drinks. This represents 5.99 billion gallons of beer or approximately 500 million bottles/cans of beer.

For 1999, *Beverage World* reported that Anheuser-Busch Co. had increased its share of the market from 46.6 to 49.9 percent. Miller Brewing Co. (part of Phillip Morris) had 21.3 percent of the market, and Adolph Coors 11.0 percent. According to Anheuser-Busch, in 1998, Budweiser commercials featuring Louis the Lizard and his catchphrase "We could have been huge" were rated America's most popular campaign ever. In the January 2000 Super Bowl ads, Budweiser's "Rex the Dog" ad rated very high and the catchphrase "Whassup" received an advertising award.

The alcoholic spirits market in 1999 tallied $34.05 billion in retail sales and totaled 330 million gallons. Wine came in third in 1999 with retail sales of $17.38 billion, but second in gallonage at 530 million gallons. The combined retail sales of all three totaled $105.63 billion.

BEER INSTITUTE'S ADVERTISING AND MARKETING CODE

The Beer Institute's Advertising and Marketing Code is cited when the industry tries to deflect some of its critics' charges. A copy of the entire Advertising and Marketing Code may be obtained from the Beer Institute or the Internet. Some of the guidelines are:

- These guidelines apply to all brewer advertising and marketing materials, including internet and other cyberspace media.
- Beer advertising and marketing materials are intended for adults of legal purchase age who choose to drink and beer consumption is intended as a complement to leisure or social activity.
- Beer advertising and marketing materials should not contain any lewd or indecent language or images, nor should it portray sexual passion, promiscuity or any other amorous activities as a result of consuming beer.
- Beer advertising and marketing activities on college and university campuses, or in college media, should not portray consumption of beer as being important to education.

Anheuser-Busch has a College Marketing Guide. In its Event Sponsorship and Promotion statement it lists the following:

- **Events on Campus:** Anheuser-Busch will limit its event sponsorship and promotion on campus to licensed retail establishments and those activities open to the general public, where most of the audience is reasonably expected to be above the legal purchase age.
- **Spring Break:** At spring break destination locations, Anheuser-Busch will not conduct beer advertising, event sponsorships or promotions on beaches or at other outdoor locations or non-licensed premises where most of the audience is reasonably expected to be below the legal purchase age.

LIMITATIONS OF VOLUNTARY BEER INDUSTRY ADVERTISING CODES

The beer industry's voluntary advertising codes are written so as to restrict advertising practices as little as possible. Qualifications like "advertising should not be used where most of the audience is reasonably expected to be below the legal purchase age" or "will not conduct any event sponsorships or promotions on beaches . . . where *most* of the audience is reasonably expected to be below the legal purchase age" allows beer manufacturers a lot of leeway.

THE COSTS OF ALCOHOLISM

In the United States alcoholism is the most widespread form of drug abuse, affecting at least five million persons.

In 1995, according to the Substance Abuse and Mental Health Services Administration, alcohol abuse and alcoholism cost an estimated $166.5 billion, while drug abuse dependence cost $109.8 billion. More than 100,000 deaths each year are related to alcohol abuse.

(SEE ALSO: *Advertising and the Pharmaceutical Industry; Advertising and Tobacco Use; Social Costs of Alcohol and Drug Abuse*)

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ADVERTISING AND THE PHARMACEUTICAL INDUSTRY The pharmaceutical industry, which researches, develops, produces, and markets prescription drugs in the United States, is the most heavily regulated of all industries when it comes to the advertising and promotion of its products. Through its Drug Marketing, Advertising, and Communications Divisions, the Food and Drug Administration (FDA) regulates all advertising and promotional activities for prescription drugs, including statements made to physicians and pharmacists by pharmaceutical sales representatives. Advertising of over-the-counter (OTC) drugs, which is not regulated by the FDA, is under the jurisdiction of the Federal Trade Commission (FTC).

Before a new prescription drug is approved for marketing, the FDA and the pharmaceutical company must agree on the "full prescribing information" that will accompany the product and that must be included in all ads, brochures, promotional pieces, and samples. This full prescribing information must include, in the correct order, the following information about the drug: its trade name, its assigned name, the strength of its dosage form, a caution statement (stating that a prescription is required), a description of its active ingredient, the clinical pharmacology of the drug, indications for its usage, contraindications for usage, precautions, adverse reactions, instructions on what to do in case of overdosage, dosage and administration, and how the drug is supplied.

Typically, this information is very detailed, and even when it is given in six-point type, it can run to two printed pages. The majority of pharmaceutical companies pay to have this information published in the *Physician's Desk Reference*, which is sent to physicians free of charge. The book is also sold in bookstores or is available on library reference shelves for use by consumers who want to know more about specific drugs.

All promotional pieces and ads to be used when a new drug is marketed must first be approved by

the FDA before marketing begins—to ensure that the statements being made are consistent with those in the official labeling. After the introduction of a new drug has been completed, copies of all subsequent ads and promotional pieces must be sent to the FDA at the time of their first use, too, but they do not have to be preapproved. The FDA reviews ads, brochures, direct-mail pieces, and sales aids to ensure that a "fair balance" has been maintained in presenting both the benefits and risks of a medication. In the 1990s, the FDA directed its attention to "scientific symposia" and other medical meetings at which information about new drugs, or new indications for drugs, are presented. This ensures that they are not just promotional programs for a single drug. In no other industry are advertising and promotion required to meet such strict standards.

THE CHANGING ROLE OF PHARMACEUTICAL ADVERTISING

Traditionally, the advertising and promotion of pharmaceutical products were directed primarily to physicians, with some limited advertising and promotion being directed to pharmacists. With the expiration of patents on some major drugs in the 1980s and 1990s, generic versions of the drugs became available from competing manufacturers. The generic drugs were priced lower than the brand-name products, so pharmacists got laws passed allowing them to substitute generic products for the brand-name products. This gave pharmacists more control over which generic company's products to purchase and dispense. Advertising and promotion to pharmacists increased. When committees, usually composed of pharmacists, became very important in deciding which drugs could, or could not, be prescribed or reimbursed under third-party payment programs (medicaid, HMOs, and other insurance programs), advertising and promotion were also directed to the decision makers in those organizations. More recently, advertising is also being directed to the consumer.

DIRECT-TO-CONSUMER ADVERTISING

In the mid-1980s, two pharmaceutical companies began direct-to-consumer advertising (DTC). Pfizer led the way with its health-care series of ads to the general public. Merrell Dow was next, using DTC ads to inform the public that physicians had a new treatment to help smokers who wanted to stop smoking. When the company's new, nonsedating antihistamine became available, it used DTC ads to tell allergy sufferers that physicians now had a new treatment for allergies. The ads did not mention the name of the products; rather, they asked patients with specific problems or symptoms to see their physician.

The next phase of DTC advertising led to ads in magazines and newspapers that mentioned the name of the product *and* its indication for use. The advertising of prescription drugs on television or radio remained greatly restricted at this time since it was not possible to include the necessary brief summary of prescribing information on the air. Because of this limitation, the ads on television or radio had to focus on either the name of the product or the indication for the product.

To promote Nicorette, a NICOTINE-containing gum designed to help smokers stop smoking, Merrell Dow advertised it on television with the message that Nicorette was now available at phamacies but only by prescription and under a doctor's supervision.

According to FDA rules at that time, Merrell Dow could not say that Nicorette was useful in helping smokers who wanted to stop smoking since it had included the name of the product in the commercial. When a company has the only—or the major—product in the market, this approach can be very effective because it increases awareness among patients that a new treatment is available and influences them to see their doctors.

In 1997 the FDA changed the regulations regarding DTC advertising of prescription products on television and radio. It now allows both the name of the product *and* indications for it to be advertised, as long as the main precautions or warnings are given in the commercial. This has led to many prescription products being advertised on television, such as Rogaine, Claritin, Allegra, Viagra, Pravachol, Prilosec, and others. Nicorette by this time had been cleared by the FDA to be sold over-the-counter and, since it no longer required a prescription, the product was no longer governed by FDA rules but rather by FTC regulations.

PROMOTIONAL EXPENDITURES

The use of DTC ads on television and radio for prescription drugs has doubled since 1997 and reached $1.9 billion in 1999. By 2000, there were ninety-seven different prescription products that had been advertised on television or in print ads. In addition to this advertising, a separate budget was formulated for advertising and promotion to physicians, seminars, and symposia, and the large force of medical service representatives who call on doctors, pharmacists, and other health-care professionals. These representatives inform them about their companies' products; that is, how to use them, the side effects to anticipate, and the different dosage forms available for each product. They also provide starter doses (samples) to physicians, which may be used to initiate treatment for a patient or, in some cases, to provide medication for a patient who cannot afford to buy it. The total for all advertising and promotion, including the medical service representatives, can run as high as 25 percent of sales.

Some members of Congress feel that pharmaceutical companies are spending too much on advertising and promotion, and some would even like to limit these expenditures. Such restrictions are already in effect in Great Britain. Proponents of spending limits feel that they would result in lower prices for prescription drugs; these same individuals do not believe that the dissemination of information about new drugs and new treatment procedures would suffer as a result. However, many in the health industry think that if physicians had to depend on their medical journals for information about new drugs, they might not know about them for several years. Meanwhile, their patients in need of the new drugs would be deprived of the latest advances in medical care. In the United States, only 8.8 percent of health-care dollars was spent on drugs. By comparison, in Canada 12.5 percent of the total health-care dollars were spent on drugs. The advertising and promotion of prescription drugs, including the cost for medical service representatives who call on the nation's physicians and other health-care professionals, make up about 2 percent of the total health-care expenditures in the United States.

CODE OF PHARMACEUTICAL MARKETING PRACTICES

The member companies of the Pharmaceutical Research & Manufacturers of America (PhRMA) have worked together to create guidelines for the ethical promotion of prescription pharmaceutical products.

The pharmaceutical industry undertakes:

- that all products it makes available for prescription purposes to the public are backed by the fullest technological service and have full regard for the needs of public health: to produce pharmaceutical products under adequate procedures and strict quality assurance;
- To have the claims for substances and formulations on valid scientific evidence, thus determining the therapeutic indications and conditions of use;
- To provide scientific information with objectivity and good taste, with scrupulous regard for truth, and with clear statements with respect to indications, contraindications, tolerance, and toxicity;
- To use complete candor in dealing with public health officials, health-care professionals, and the public, and to comply with the regulations and policies issued by the Food and Drug Administration.
- Information on pharmaceutical products should be accurate, fair, and objective, and presented in such a way as to conform not only to legal requirements but also to ethical standards and to standards of good taste.
- Information should be based on an up-to-date evaluation of all the available scientific evidence and should reflect this evidence clearly.
- No public communication should be made with the intent of promoting a pharmaceutical product as safe and effective for any use before the required approval of the pharmaceutical product for marketing for such product is obtained.
- Particular care should be taken that essential information as to pharmaceutical products' safety, contraindications, and side effects or toxic hazards is appropriately and consistently communicated subject to the legal, regulatory, and medical practices of the United States.
- Medical representatives should be adequately trained and possess sufficient medical and

technical knowledge to present information on their company's products in an accurate and responsible manner.
- Symposia, congresses, and the like are indispensable for the dissemination of knowledge and experience. Scientific objectives should be the principal focus in arranging such meetings, and entertainment and other hospitality should not be inconsistent with such objectives.
- Scientific and technical information should fully disclose properties of the pharmaceutical product as approved in the United States based on current scientific knowledge and FDA regulations.
- Samples may be supplied to the medical and allied professions to familiarize them with the products or to enable them to gain experience with the product in their practice. The requirements of the Prescription Drug Marketing Act of 1987 should be observed.

The PhRMA Board also includes these four position statements as an adjunct to the PhRMA *Code of Pharmaceutical Marketing Practices*.

> Gifts, hospitality or subsidies offered to physicians by the pharmaceutical industry ought not to be accepted if acceptance might influence or appear to others to influence the objectivity of clinical judgment. A useful criterion in determining acceptable activities and relationships is: Would you be willing to have these arrangements generally known?
>
> Independent institutional and organizational continuing medical education providers that accept industry-supported programs should develop and enforce explicit policies to maintain complete control of program content.
>
> Professional societies should develop and promulgate guidelines that discourage excessive industry-sponsored gifts, amenities, and hospitality to physicians at meetings.
>
> Physicians who participate in practice-based trials of pharmaceuticals should conduct their activities in accord with basic precepts of accepted scientific methodology.

The PhRMA Board of Directors also adopted, as part of the PhRMA *Code of Pharmaceutical Marketing Practices*, the following guidelines on gifts given to physicians from industry as set forth in the Opinion of the Council on Ethics and Judicial Affairs.

> Any gifts accepted by physicians individually should primarily entail a benefit to patients and should not be of substantial value. Accordingly, textbooks, modest meals, and other gifts are appropriate if they serve a genuine educational function. Cash payments should not be accepted.
>
> Individual gifts of minimal value are permissible as long as the gifts are related to the physician's work. (e.g., pens and notepads).
>
> Subsidies to underwrite the costs of continuing medical education conferences or professional meetings can contribute to the improvement of patient care and therefore are permissible. Since the giving of a subsidy directly to a physician by a company's sales representative may create a relationship which could influence the use of the company's products, any subsidy should be accepted by the conference sponsor, who in turn can use the money to reduce the conference's registration fee. Payments to defray the costs of a conference should not be accepted directly from the company by the physicians attending the conference.
>
> Subsidies from industry should not be accepted to pay for the costs of travel, lodging, or other personal expenses of physicians attending conferences or meetings, nor should subsidies be accepted to compensate for the physicians' time. Subsidies for hospitality should not be accepted outside of modest meals or social events held as a part of a conference or meeting. It is appropriate for faculty at conferences or meetings to accept reasonable honoraria and to accept reimbursement for reasonable travel, lodging, and meal expenses. It is also appropriate for consultants who provide genuine services to receive reasonable compensation and to accept reimbursement for reasonable travel, lodging, and meal expenses. Token

consulting or advisory arrangements cannot be used to justify compensating physicians for their time or their travel.

Scholarship or other special funds to permit medical students, residents, and fellows to attend carefully selected educational conferences may be permissible as long as the selection of students, residents, or fellows who will receive the funds is made by the academic or training institution.

No gifts should be accepted if there are strings attached. For example, physicians should not accept gifts if they are given in relation to the physician's prescribing practices. In addition, when companies underwrite medical conferences or lectures other then their own, responsibility for and control over the selection of content, faculty, educational methods, and materials should belong to the organizers of the conferences or lectures.

(SEE ALSO: *Advertising and the Alcohol Industry; Advertising and Tobacco Use*)

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ADVERTISING AND TOBACCO USE

Tobacco companies spend more than $5 billion annually to advertise and promote cigarettes and other tobacco products. Tobacco companies claim that the purpose and desired effect of marketing are merely to provide information and to influence brand selection among current smokers, although only about 10 percent of smokers switch brands in any one year. Since more than one million adult smokers stop smoking every year and almost half a million other adult smokers die from smoking-related diseases, the tobacco companies must recruit an average of 3,300 new young smokers every day to replace those who die or otherwise stop smoking. Tobacco companies contend that smoking is an "adult habit" and that adult smokers "choose" to smoke. However, many medical researchers assert that cigarette smoking is primarily a childhood addiction or disease and that most of the adults who smoke started as children and could not quit.

Unlike the pharmaceutical companies, which are tightly regulated as to their advertising and promotion, the tobacco industry has had few regulations. The basic restrictions have been that companies cannot use paid advertising on television or radio, they cannot claim what they cannot prove (e.g., that low-tar cigarettes are less hazardous to health), and they must include one of four warnings on cigarette packages and ads. The fact that warning labels are printed on a pack of cigarettes has been successfully used by the tobacco companies as a defense against tobacco victims' lawsuits.

The whole picture changed when Florida, Minnesota, Mississippi, and Texas were able to reach an agreement in 1997 and early 1998 with the major tobacco companies and won compensation for the effects of smoking on their health-care expenses. Minnesota was able to obtain copies of long-secret memos, reports, letters, and other documents that were made public as part of the $6.6 billion settlement reached in their lawsuit against cigarette makers.

On November 23, 1998, the major tobacco companies entered into an agreement with the other forty-six states. This agreement, which is known as the Master Settlement Agreement (MSA), settled litigation brought by the states and other entities that were seeking the reimbursement of expenditures related to smoking and health. Under this agreement, the states and tobacco companies jointly agreed to concrete provisions to reduce youth smoking, new public health initiatives, and important new rules for governing a tobacco company's way of doing business.

The cigarette companies agreed to pay $368.5 billion over 25 years. Of this, $246 billion goes to the states, and they have started to receive payments under this agreement. The state of Florida receives $450 million each year under this agreement, Iowa $54.9 million, and the other states differing amounts. Iowa, Kansas, and Washington have agreed to set aside this money entirely for health care. Iowa has passed a law that their money shall go to three areas: access to health care, public health and smoking prevention, and substance-abuse treatment and prevention. In other states, this new-found money has created hot political battles over how much of their tobacco settlement to spend on tobacco prevention programs.

A two-year education effort and ad campaign has lowered the number of teen smokers in Florida. The campaign reduced middle-school smoking by more than half and lowered smoking among high-school students by 24 percent. This multimillion-

The Marlboro man, a controversial tobacco icon, on a billboard in downtown Denver, June 20, 1997. (Archive Photos, Inc.)

dollar campaign was financed by Florida's $11.3 billion tobacco settlement, of which Florida has already received $2 billion.

The MSA has changed the way cigarette companies can market, advertise, and promote their cigarettes. The agreement specifically includes the following:

- No participating manufacturer may take any action, directly or indirectly, to target youth in the advertising, promotion, or marketing of tobacco products. It also prohibits any action the *primary purpose* of which is to initiate, maintain, or increase the incidence of youth smoking.
- Effective April 23, 1999, billboards, stadium signs, and transit signs advertising tobacco are banned. However, this does *not* apply to retail establishments selling tobacco. They may have signs up to 14 square feet inside or outside their stores.
- Effective May 22, 1999, the use of cartoon characters in advertising, promoting, packaging, or labeling of tobacco products is banned. (This applies only to ''exaggerated depictions, or depictions of entities with superhuman powers''. It does *not* cover the standard camel logo or simple drawings of a camel. It does not prohibit the continued use of the Marlboro man or other human characters.)
- Beginning July 1, 1999 participating manufacturers and others licensed by them may no longer market, distribute, offer or sell, or license any apparel or merchandise bearing a tobacco brand name.
- Free product sampling is banned anywhere, except for a facility or enclosed space where an operator may ensure that no minors are present.
- Manufacturers may not sell or distribute cigarette packs containing less than twenty cigarettes until the year 2001.
- There shall be no payment for the use of tobacco products in movies, TV programs, live performances, videos, or video games. (Does not apply to media viewed in an adult-only facility or to media not intended for distribution to or display to the public.)
- There shall be no licensing of third parties to use or advertise any brand name in a way that would constitute a violation of the MSA if done by the participants.
- No nonbranded item may be given in exchange for the purchase of tobacco products or redemption of coupons or proof of purchase without proof of age.
- No use of a tobacco brand name as part of the name of a stadium shall be allowed.
- Tobacco sponsorships are limited to one per year, after a three-year grace period (from November 1998). Such brand-name sponsorship may not include concerts, events in which any paid participant or contestants are youth, or any athletic event between opposing teams in any football, basketball, baseball, soccer, or hockey league.

The previous voluntary cigarette advertising and promotion code rules are also still in effect:

- Cigarette smoking is an adult custom. Children should not smoke. Laws prohibiting the sale of cigarettes to minors should be strictly enforced. The cigarette manufacturers advertise and promote their products only to adults smokers. They support the enactment and enforcement of state laws prohibiting the sale of cigarettes to persons under 18 years of age.

Advertising.

1. Cigarette advertising shall not appear in publications directed primarily to those under 21 years of age, including school, college, or university media (such as athletic, theatrical, or

other programs). Comic books or comic supplements are included.
2. No one depicted in cigarette advertising shall be or appear to be under 25 years of age.
3. Cigarette advertising shall not suggest that smoking is essential to social prominence, distinction, success, or sexual attraction, nor shall it picture a person smoking in an exaggerated manner.
4. Cigarette advertising may picture attractive, healthy-looking persons provided there is no suggestion that their attractiveness and good health are due to cigarette smoking.
5. Cigarette advertising shall not depict as a smoker anyone who is or has been well known as an athlete, nor shall it show any smoker participating in, or obviously just having participated in, a physical activity requiring stamina or athletic conditioning beyond that of normal recreation.
6. No sports or celebrity testimonials shall be used or those of others who would have special appeal to persons under 21 years of age.

All the agreed-on advertising and promotional restrictions spelled out in the MSA should be very helpful in curbing underage smoking, but the tobacco companies have always found ways to bypass the bans and advertise in other venues. Billboard advertising is now banned but tobacco companies have increased their level of advertising in magazines, many of which are read by teenagers. Seventy-three percent of teens (aged 12 to 17) reported seeing tobacco advertising in the previous 2 weeks, compared to only 33 percent of adults surveyed. Since billboards were banned, 61 percent of teens who recalled tobacco advertising saw it in magazines, compared to 50 percent the year before.

A survey also revealed that 77 percent of teens say it is easy for people under the age of 18 to buy cigarettes and other tobacco products. Many displays of cigarettes in convenience stores are at waist level, making them plainly available to children. The campaign to restrict access by youths under the age of 18 to cigarettes has not been too successful.

A California suit against R.J. Reynolds Tobacco, filed on May 11, 2000, charges that the company has violated the legal settlement with state governments by improperly distributing large quantities of free cigarettes by mail. This case marks the first time an attorney general has taken a cigarette company to court to enforce the terms of the MSA. Reynolds said it was part of a program of "consumer testing" and was therefore allowable under the agreement. The attorney general alleges Reynolds mailed the free cigarettes "under the guise of consumer testing or evaluation in order to market and advertise its products." According to the suit, Reynolds sent more than 900,000 multipack cigarette mailings to more than 115,000 California residents during 1999, some receiving as many as ten packs at a time.

In his memoirs, former Surgeon General C. Everett Koop said this about the tobacco industry, "After studying in depth the health hazards of smoking, I was dumbfounded—and furious. How could the tobacco industry trivialize extraordinarily important public-health information: the connection between smoking and heart disease, lung and other cancers, and a dozen or more debilitating and expensive diseases? The answer was—it just did. The tobacco industry is accountable to no one."

UNDERSTANDING THE SMOKING HABIT

Almost all smokers started before the age of 21—most before the age of 18—many before the age of 14. Young people who learn to inhale cigarette smoke and experience the mood-altering effects from the inhaled nicotine quickly become dependent on cigarettes to help them cope with the complexities of everyday life. Having developed a nicotine dependence, they find they must continue smoking to avoid the downside of nicotine withdrawal. The earlier they start to smoke, the more dependent they seem to become—and the sooner they start to experience smoking-related health problems. Six years of research at the National Center on Addiction and Substance Abuse at Columbia University reveals that a child who reaches age 21 without smoking, using illegal drugs, or abusing alcohol is virtually certain never to do so.

A survey conducted by the U.S. Department of Health and Human Services among high school students who smoked half a pack of cigarettes a day found that 53 percent had tried to quit but could not. When asked whether they would be smoking 5 years later, only 5 percent said they would be—but 8 years later, 75 percent were still smoking.

PURPOSE OF CIGARETTE ADVERTISING

The tobacco companies are very adept at using advertising and different kinds of promotional programs to help them accomplish several major objectives:

1. To reassure current smokers. To offset the effect of thousands of studies showing the adverse health effects of smoking and of the requested warning labels on cigarette packages, the tobacco industry has continued to claim that no one has yet "proven" that smoking "causes" health problems—that these are just "statistical associations." But, recently in Florida, a six-person jury decided, on April 7, 2000, that tobacco companies' cigarettes were a deadly, addictive, and defective product and caused cancer for three smokers who sued the industry in a class-action lawsuit. The companies must pay $12.7 million to the plaintiffs. The jury has yet to decide the punitive damages, which could be massive. The state of Florida, in order to protect its tobacco payments in the future, passed a law capping the amount of bond the companies would have to post in order to appeal such punitive damages at $100 million or 10 percent of the company's net worth, whichever is less.
2. To associate smoking with pleasurable activities. In their ads, tobacco companies show healthy young people enjoying parties, dancing, attending sporting events, having a picnic at the beach, sailing, and so on. The implication is that if you smoke, you too will experience the kind of good times enjoyed by the smokers in the ads.
3. To associate smoking with other risk-taking activities. Since as indicated by the warning labels on every package of cigarettes, smoking involves risk to one's health, the tobacco companies attempt to counter this by showing in their ads such risk-taking activities as ballooning, mountain climbing, sky diving, and motorcycle riding. This is the industry's not so subtle way of saying: Go ahead and take a risk by smoking. You are capable of deciding the level of risk you want to assume. The tobacco companies are betting on the fact that most young people consider themselves to be immortal and do not believe any of smokings bad effects will ever happen to them.
4. To associate cigarette smoking with becoming an adult. Realizing that teenagers desire to be considered adults, to be free to make their own decisions, and to be free from restrictions on what they can and cannot do, the tobacco companies go to great lengths to stress that smoking is an "adult habit"—that only adults have the right to choose whether or not to smoke. Since teenagers are in a hurry to grow up and be free, the simple act of smoking cigarettes can become their way of showing to the world that they are indeed adults.
5. To associate cigarette smoking with attractiveness to the opposite sex. Many ads for cigarettes imply that if you smoke, you will also be attractive to members of the opposite sex. In fact, surveys of young people and adults show that most people prefer to date nonsmokers.
6. To associate smoking with women's liberation. "You've come a long way baby" was the theme of the early ads for Virginia Slims cigarettes. What these ads did not say is that women who smoke like men will die like men who smoke. The slogan "Torches of Freedom" coupled with an image of women smoking cigarettes while marching down Fifth Avenue in the Easter Parade was a cigarette company's public relations ploy years ago to influence women to start smoking. In the 1990s, lung cancer became the number one cancer found in women, exceeding the incidence of breast cancer.
7. To show that smoking is an integral part of our society. The sheer number of cigarette ads—those on billboards, on articles of clothing, on signs at sporting events—leave the impression that smoking is socially accepted by the majority of people. This image is supported by movies that include scenes of cigarette smoking. Many events sponsored by tobacco companies include the name of a major brand of cigarettes or smokeless tobacco, such as the Kool Jazz Festival, the Benson & Hedges Blues Festival, the Magna Custom Auto Show, the Winston Cup (stock car racing), the Marlboro Cup (soccer), the Marlboro Stakes (horse racing), and the Virginia Slims Tennis Tournament, just to name a few. Although tobacco advertising is legally prohibited on television, the ban has been ignored by the strategic placement of tobacco-product ads in baseball and football stadiums, basketball arenas, and hockey rinks, around

auto racetracks, and at tractor pulls and other sporting events.

8. To discourage articles in magazines about the health risks of smoking. So important to magazines are the ads they carry for cigarettes, beer, food, and other products, which are marketed by the major cigarette companies or their parent companies, that many publishers are very reluctant to antagonize cigarette producers by running articles on the health risks of smoking. This is especially true with women's magazines.
9. To gain legitimacy. Tobacco companies seek public acceptance and recognition by supporting worthwhile groups and programs. Many groups receive significant amounts of funding from the tobacco companies to support their programs. One especially large grant, from RJR Nabisco, was a contribution of $30 million for "innovative education programs" to schools across the country. In1989, Philip Morris made arrangements to sponsor the Philip Morris Bill of Rights Exhibit, which toured the United States in celebration of the 200th anniversary of the Bill of Rights. In this way, Philip Morris tried to associate its company—including its tobacco subsidiary—with the Bill of Rights, and to reap positive press coverage as the exhibit went on display in each city.

HISTORY OF TOBACCO ADVERTISING AND PROMOTION

Tobacco companies' advertising, before restrictions were implemented, was focused on television, radio, newspapers, and magazines. The advertising was represented by ads such as "I'd walk a mile for a camel" or the "Call for Phillip Morris" or "More doctors smoke Camels" or "Not a cough in a carload." This evolved into the "Joe Camel" ads, the "Kool Penguin" ads, and the "Newport Menthol" cigarette ads.

Tobacco advertising and promotional expenses have steadily increased. In 1997, the tobacco companies spent $5.66 billion to promote their products, up from $5.11 billion in 1996. The largest category of spending was for promotional allowances to wholesalers and retailers, $2.4 billion, more than double their spending in 1990. Next were expenditures for retail value added. At $970 million, this category includes non-cigarette items given away with cigarettes. Coupons and multiple pack offers were an additional $552 million, followed by specialty item distribution, $512 million; point of sale advertising, $305 million; outdoor advertising, $295 million; magazines, $236 million; public entertainment, $195 million; and $130 million for all other forms of advertising.

There were no restrictions on cigarette advertising in the United States until the first *Report of the Surgeon General* was released on January 11, 1961. Because of the health hazards described therein, the report led to the Federal Cigarette Labeling and Advertising Act of 1965 and, beginning in 1966, Congress mandated that a health warning appear on all cigarette packages, although not in advertisements. On June 2, 1967, the Federal Communications Commission (FCC) ruled that the Fairness Doctrine in advertising applied to cigarette ads on television and radio and required broadcasters who aired cigarette commercials to provide "a significant amount of time" to citizens who wished to point out that smoking "may be hazardous to the smoker's health." This rule went into effect on July 1, 1967. The FCC required that there be one free public-service announcement (PSA) for every three paid cigarette commercials. During the three-year period of 1968 to 1970, in which the PSAs were mandated by the Fairness Doctrine, per capita cigarette sales decreased by 6.9 percent.

In January 1970, the cigarette industry offered voluntarily to end all cigarette advertising on television and radio by September 1970—a move that would also eliminate any PSAs, which were hurting sales. Ultimately, Congress approved the Public Health Cigarette Act of 1969, which prohibited cigarette advertising in the broadcast media as of January 1, 1971.

In September 1973, the Little Cigar Act of 1973 banned broadcast advertising of little cigars (cigarette-sized cigars). During the three-year period of 1971 to 1973, following the end of the PSAs required by the Fairness Doctrine and the beginning of the broadcast advertising ban, cigarette sales increased by 4.1 percent.

More than a decade later, smokeless-tobacco advertising in the broadcast media was banned by the Comprehensive Smokeless Tobacco Health Education Act of 1986. This ban took effect on August 27, 1986. The Federal Trade Commission (FTC) Bureau of Consumer Protection ruled in 1991 that the Pinkerton Tobacco Company violated the 1986

statute banning the advertising of smokeless tobacco and prohibited it from "displaying its brand name, logo, color or design during televised (sports) events" of its Red Man Chewing Tobacco and snuff. This was the first action of its kind by the FTC.

STAT (Stop Teenage Addiction to Tobacco), at their 1991 STAT-91 Conference, addressed the problem of tobacco companies' efforts to encourage tobacco addiction in young people. It was learned that the RJR Nabisco cartoon camel was at the center of the most extensive advertising campaign ever created to influence the values and behavior of young people. Camel's share of the teenage market rose from almost nothing to almost 35 percent in just three years by "this sleazy dromedary."

(SEE ALSO: *Advertising and the Alcohol Industry; Tobacco: Dependence; Tobacco: Industry*)

CHARLES M. RONGEY

AFRICAN AMERICANS *See* Ethnic Issues and Cultural Referance in Treatment; Ethnicity and Drugs; Vulnerability as Cause of Substance Abuse: Race

AGGRESSION AND DRUGS: RESEARCH ISSUES ALCOHOL, narcotics, HALLUCINOGENS, and PSYCHOMOTOR STIMULANTS differ markedly one from the other in terms of pharmacology and neurobiological mechanisms, dependence liability, legal and social restraints, expectations and cultural traditions. No general and unifying principle applies to all these substances, and it would be misleading to extrapolate from the conditions that promote violence in individuals under the influence of alcohol to those with other drugs. Different types of drugs interact with aggressive and violent behavior in several direct and indirect ways from (1) direct activation of brain mechanisms that control aggression, mainly in individuals who have already been aggressive in the past; (2) drug states, such as alcohol or hallucinogen intoxication, serving as license for violent and aggressive behavior; (3) drugs such as heroin or cocaine serving as commodities in an illegal distribution system, drug trafficking, that relies on violent enforcement tactics; to (4) violent behavior representing one of the means by which an expensive cocaine or heroin habit is financed. Systematic experimental studies in animals represent the primary means to investigate the proximal and distal *causes* of aggressive behavior, whereas studies in humans most often attempt to infer causative relationships mainly from *correlating* the incidence of violent and aggressive behavior with past alcohol intake or abuse of other drugs. The ethical dilemma of research on aggression in animals and humans is the demand for reducing harm and risk to the research subject, on the one hand, and on the other, to validly capture the essential features of human violence that is by definition injurious and harmful.

Methodologically, aggression research stems from several scientific roots, the experimental-psychological, ethological, and neurological traditions being the most important. The use of aversive environmental manipulations in order to produce defensive and aggressive behavior has been the focus of the experimental-psychological approach. During the 1960s, "models" of aggression were developed that rely upon prolonged isolated housing or crowding; exposure to noxious, painful electrical shock pulses; omission of scheduled rewards; or restricted access to limited food supplies as the major aversive environmental manipulations. The behavioral endpoints in these models are defensive postures and bites in otherwise placid, domesticated laboratory animals. The validity of such experimental preparations in terms of the ethology of the animal, i.e. how animals normally react outside of the laboratory, and in their relation to human aggressive and violent behavior remains to be determined. Aggression research using human subjects studied under controlled laboratory conditions has employed aversive environmental manipulations, such as the administration of electric shocks, noxious noise, or loss of prize money to a fictitious opponent. This type of experimental aggression research highlights the dilemma of attempting to model essential features of valid violence under controlled laboratory conditions without risking the harm and injury that are characteristic of such phenomena. While it is unethical to demand experimental studies that involve "realistic" violent behavior, the relation between competitive behavior in laboratory situations to violence outside the laboratory remains to be validated.

In addition to environmental manipulations, histopathological findings of brain tumors in violent patients prompted the development of experimental procedures that ablate and destroy tissue in areas of the brain such as the septal forebrain, medial hypothalamus, or certain midbrain regions of laboratory rats and other animals. Such experimental manipulations most often result in ragelike defensive postures and biting, often called rage, hyperreactivity, hyperdefensiveness. Alternatively, electrical stimulation of specific brain regions can evoke predatory attack, aggressive and defensive responses in certain animal species. When animals are given very high, near-toxic amphetamine doses and similar drugs, bizarre, rage-like responses may emerge. Similarly, aggressive and defensive behavioral elements are induced by exposure to very high doses of hallucinogens and during withdrawal from opiates. The inappropriate context, the unusually fragmented behavioral response patterns, and the limitation to domesticated laboratory rodents make aggressive and defensive reactions that are induced by lesions, electrical brain stimulation, drugs and toxins difficult to interpret or generalize to the human situation.

In contrast to the emphasis on aversive environmental determinants or on neuropathologies, the ethological approach to the study of animal aggression has focused on adaptive forms of aggressive behavior. Defense of a territory, rival fighting among mature males during the formation and maintenance of a group, defense of the young by a female, and anti-predator defense are examples of these types of aggressive, defensive, and submissive behavior patterns, often referred to as agonistic behavior. Sociobiological analysis portrays these behavior patterns as having evolved as part of reproductive strategies ultimately serving the transmission of genetic information to the next generation. The focus on aggressive behavior as it serves an adaptive function in the reproductive strategies, however, complicates the extrapolation to violent behavior as it is defined at the human level. How the range of human violent acts relate to the various types of animal aggression and how they may share common biological roots remains to be specified.

How have these ethological, neurological, and experimental-psychological research traditions contributed to our understanding of the link between drugs of abuse and alcohol to human aggression and violence? Epidemiological and criminal statistics link alcohol to aggressive and violent behavior in a human pattern large in magnitude, consistent over the years, widespread in types of aggressive and violent acts, massive in cost to individual, family, and society, and serious in suffering and harm. Systematic experimental studies have identified the early phase after a low acute (short-term) alcohol dose as a condition that increases the probability of many types of social interactions, including aggressive and competitive behaviors, and high-dose alcohol intoxication as the condition most likely to be linked to many different kinds of violent activities. Yet, most alcohol drinking is associated with acceptable social behavior. This is because individuals differ markedly in their propensity to become intoxicated with alcoholic beverages and to subsequently engage in violent and aggressive behavior, rendering population averages poor representations of how alcohol causes individuals to behave violently. The sources for the individual differences may be genetic, developmental, social, and environmental. Genetic association between antisocial personality, possibly diagnosed with the aid of certain electrophysiological measures, and alcoholism remains to be firmly established. In the early 1990s, the neurobiological mechanisms of alcohol action for a range of physiological and behavioral functions began to be identified; it appears that the actions of alcohol on brain serotonin and the benzodiazepine/$GABA_A$ receptor complex are particularly relevant to alcohol's effects on aggressive and violent behavior. For example, studies in rodents and primates indicate that benzodiazepine-receptor antagonists prevent the aggression-heightening effects of alcohol. Similarly, the actions of alcohol on neuroendocrine events that control testosterone and adrenal hormones appear important in the mechanisms of alcohol's aggression-heightening effects. Among the environmental determinants of alcohol's effects on violence that are of paramount significance are social expectations and cultural habits as well as the early history of the individual in situations of social conflict. Impaired appraisal of the consequences, inappropriate sending and receiving of socially significant signals, disrupted patterns of social interactions are characteristic of alcohol intoxication that contribute to the violence-promoting effects. A particularly consistent observation is the high prevalence of alcohol in victims and targets of aggres-

sion and violence. In contrast to heroin and cocaine, since alcohol is not an illicit drug, its link to violence is not a characteristic of the economic distribution network for this substance.

Violence in the context of drug addiction is due largely to securing the resources to maintain the drug habit as well as to establishing and conducting the business of drug dealing. Neither animal nor human data suggest a direct, pharmacological association between violence and acute or chronic administration of opiates. Although measures of hostility and anger are increased in addicts seeking methadone treatment, these feelings usually do not lead to aggressive or violent acts. Rather, the tendency to commit violent crimes correlates with preaddiction rates of criminal activity. However, experimental studies in animals point to the phase of withdrawal from chronic opiates as the most vulnerable period to be provoked to heightened levels of aggressive behavior. Nevertheless, although humans undergoing opiate withdrawal may experience increased feelings of anger, there is no evidence suggesting that they are more likely to become violent as a result.

The most serious link of amphetamine to violence is in individuals who, after taking intravenous amphetamine—most often chronically—develop a paranoid psychotic state during which they commit violent acts. Most psychiatric reports and police records do not support a psychiatric opinion of the early 1970s that "amphetamines, more than any other group of drugs, may be related specifically to aggressive behavior." The prevalence of violence by individuals who experience amphetamine paranoid psychosis may be less than 10 percent in general population samples and as high as 67 percent among individuals who showed evidence of psychopathology prior to amphetamine use. Low acute amphetamine doses can increase various positive and negative social behaviors; higher doses often lead to disorganizing effects on social interactions and to severe social withdrawal. At present, the neurobiological mechanisms for the range of amphetamine effects on aggressive and social behavior remain unknown.

There are surprisingly few pharmacological and psychiatric studies on cocaine's effects on aggression and violence; the available evidence points to psychopathological individuals who may develop the propensity to engage in violent acts. However, the far more significant problem is the violence associated with the supplying, dealing, and securing of crack-cocaine, as documented in epidemiological studies.

Most experimental studies with animals and humans, as well as most data from chronic users, emphasize that *Cannabis* preparations (e.g., marijuana, hashish) or the active agent tetrahydrocannabinol (THC) decrease aggressive and violent behavior. Owing to the relatively widespread access, lower cost, and characteristic pattern of use, socioeconomic causes of violence in *Cannabis* dealing and procuring are less significant than they are with cocaine or heroin.

LSD was not of significance in the early 1990s, but older data suggest that certain psychopathological individuals who begin using LSD may engage in violent acts; however, this phenomenon is rare.

Phencyclidine (PCP) cannot be causally linked to violent or assaultive behavior in the population as a whole. Generally, personality traits and a history of violent behavior appear to determine whether or not PCP intoxication leads to violence. PCP violence is a relatively rare phenomenon, although when it occurs, it stands out by its highly unusual form and intensity. It depends on the individual's social and personal background.

The impact of genetic predispositions to be susceptible to becoming involved with dependence-producing drugs—such as alcohol, heroin, or cocaine—and to act violently has, as of yet, not been delineated in terms of specific neural mechanisms. Similarly, the modulating influences of learning, social modeling, or parental physical abuse on the neural substrate for drug action and for aggressive behavior have not been specified. Since these critical connections remain poorly understood, it is not possible at present to support specific modes of intervention on the basis of neurobiological data.

(SEE ALSO: *Alcohol: Psychological Consequences of Chronic Abuse; Crime and Alcohol; Crime and Drugs*)

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KLAUS A. MICZEK

AGING, DRUGS, AND ALCOHOL One of the most important developments of the twentieth century has been the enormous rise in worldwide population in general, and especially the survival of an estimated six hundred million people aged sixty or older (Ikels, 1991). The increase in the percentage of elderly in the total population results from medical, economic, and social factors plus a decline in the birthrate. According to 1989 U.S. Bureau of the Census figures, persons over sixty-five represented 12 percent of the U.S. population, and it is projected that this proportion will almost double by the year 2030—since the baby-boom generation, born after 1945, will start reaching 65 in 2010.

This fastest-growing segment—the elderly—uses pharmacological and health services more often than any other part of the population (Brock, Guralnik, & Brody, 1990). Aging people are more susceptible to infectious disease. Many suffer from multiple chronic diseases and often from conditions that have grown slowly worse throughout their lifetimes. Some conditions are the result of accidents and some are from degenerative diseases. The latter include many kinds of cancer; diseases of the immune system such as lupus; diseases of the heart and blood vessels such as stroke and hardening of the arteries; diseases of the glands such as diabetes; bone and joint diseases such as arthritis and osteoporosis; and diseases of the lungs such as emphysema. Like the rest of the population, the elderly also suffer from psychiatric disorders, some of which may respond to medication. Hence physicians (sometimes multiple physicians) often prescribe multiple medicines for treatment. If each physician does not know all the medications prescribed by all the other physicians treating the patient, two or more of these medications may interact, sometimes even causing death (Monane, M; Monane, S; & Semla, 1997; Stein, 1994). Although they comprised only 12 percent of the U.S. population in 1988, the aging accounted for 35 percent of prescription-drug expenditures (Health Care Financing Administration, 1990). Furthermore, the elderly—like the rest of the population—may also take over-the-counter drugs such as aspirin or allergy tablets, smoke tobacco, drink caffeine-laden and alcoholic beverages, and even use illicit drugs. Because of certain changes in their bodies, their responses to all medicines and to the interactions of one drug to another drug, and of medicines to alcohol, may differ from those in younger people (Montamat, Cusack, & Vestal, 1989).

The use and abuse of ALCOHOL is a public-health problem. Among people sixty-five years of age and older, 33 percent report using some alcohol (National Household Survey on Drug Abuse, 1999). About 6 percent of the elderly are considered heavy drinkers (more than two drinks per day), but about 5 to 12 percent of men and 1 to 2 percent of women in their sixties are problem drinkers (Atkinson, 1984). Alcoholism and prescription-drug abuse may result in physical, psychological, and social illnesses and premature death among the elderly from either severe withdrawal symptoms, medical complications, or suicide. Medicines intended to affect the mind (including ones intended to combat psychosis, depression, anxiety, and sleep problems) are commonly prescribed for the elderly (Rummans, Evans, Krahn, & Fleming, 1995). Studies suggest that the BENZODIAZEPINES or other SEDATIVE-HYPNOTICS are the most commonly prescribed classes of these medicines. The effects of these medicines add to and interact with those of alcohol (Scott, 1989). All these factors taken together—alcohol, old age, multiple diseases, and multiple medications—can lead to poisonous, even fatal, interactions of two or more medicines. The complexities of alcohol, age, and drug interactions are discussed in the sections that follow.

NORMAL AGING AND BODILY CHANGES

Today, there is great interest in gerontology, the study of aging, because there are now more older persons in society than ever before, and their number is expected to rise dramatically. The present goal of gerontology is not necessarily to increase the life span but rather to increase the health span—

that is, the number of years that a person will enjoy good health. Aging comprises multiple ongoing processes; disease and disability are disruptions.

Several factors once thought part of normal human aging have now been shown to be diseases. With aging, the immune system no longer performs as it once did. For example, the thymus gland, one of the central pacemakers of the immune system, gradually decreases in size, and eventually most of it is replaced by fat and connective tissue. The risk of cancer and autoimmune diseases increases among the elderly. An older person exhibiting a weaker response to bacteria and may produce autoantibodies (antibodies which work against his or her own tissues), instead of defending against foreign parasites and aggressors (Weksler, 1990). This problem, if it occurs, shows that the immune system is no longer functioning normally. These immune changes may be responsible for the increased risk among the elderly of sickness and death from infectious diseases. A decline in the hormonal system may affect many different organs of the body. For example, diabetes is a common development in older persons. The pancreas makes insulin, but cells in the body cannot utilize it as effectively as they used to do. Both thyroid-stimulating hormone produced in the pituitary gland and thyroid hormone secreted by the thyroid gland itself show a decline with advancing age. This process is functionally reflected in a decrease in the basal metabolism of as much as 20 percent from age thirty to age seventy. Thus, aging may result in part from the loss of hormonal activities and a decline in the functions they control.

With advancing age, persons tend to have slower reactions to stimuli, wider variations in function, and slower return to resting states. This decline in stability within the body (or homeostasis) is found in a number of body systems. For example, the sensitivity of the baroreceptors, which help maintain a normal blood pressure by changing the heart rate and the tension in blood vessels, declines with age. Likewise, the elderly are prone to being too hot (hyperthermia) or cold (hypothermia) because of a weakened ability to regulate body temperature. A large proportion of age-related problems in the stomach and intestines, such as constipation, are caused and made worse by long-term abuse of laxatives, poor eating habits, not drinking enough fluids, and lack of exercise. Some elderly persons are not aware of the importance to their general health of diet and exercise. For example, diseases involving hardening of the arteries are less prevalent in populations that eat no meat and little fat.

For large populations, increased age is associated with increased variability in most dimensions of health. Thus it is difficult to discriminate between normal and abnormal states. Moreover, even those aging changes considered usual or normal within a defined population do not necessarily happen in a particular aging person.

AGING AND ALTERED DRUG RESPONSE

Some drugs act differently in old persons than they do in the young or middle-aged. The difference stems from age-related changes in PHARMACOKINETICS, the bodily processes that absorb, distribute within the body, make use of, and excrete medicines (Vestal & Cusack, 1990). All these factors can affect the levels of medicines in blood and tissues. For example, with aging, the percent of water and lean tissue (mainly muscle) in the body decreases, while the percent of fat tissue increases. These changes can affect the distribution of a drug to different parts of the body, the length of time that it stays in the body, as well as the amount that is absorbed by body tissues. One reason that drinking the same amount of alcohol has a greater effect on the elderly is that there is a smaller volume of total body fluids, resulting in higher blood-alcohol levels than in the young (Vestal et al., 1977). Most medicines are eliminated from the body by metabolism in the liver followed by excretion by the kidney. (To a limited extent, metabolism occurs in other organs as well, including the stomach and intestines, the kidneys, and the lungs.) Although enzymes continue in general to metabolize at the same rate in the old as in the young, both the total weight of the liver as a percentage of total body weight and the total blood-flow through the liver decrease with aging (Loi & Vestal, 1988). As a result, the overall capacity of the liver to convert some medicines to their inactive break-down products declines with age. For example, some studies show that medicines such as diazepam (Valium), alprazolam (Xanex), chlordiazepoxide (Librium), propranolol, valproic acid, lidocaine, and theophylline are metabolized at a slower rate in old persons than in young ones. This decline is highly

variable, however, and not all drugs metabolized by the liver show an age-related slowing in the rate of metabolism. In fact, the metabolism of alcohol by the liver does not decline with age (Vestal et al., 1977).

The most consistent physiological change with aging is a decline in kidney function. Both the rate at which tiny blood vessels in the kidney filter the blood and the total flow of blood through the kidneys decline with age. As a result, medicines that are in general excreted by the kidneys regularly are excreted more slowly in the urine of the elderly and hence build up more quickly in their bloodstream. This fact is particularly important for medicines with a narrow therapeutic window (a small difference between the amount of the medicine which is enough to do any good and the amount of the medicine which is poisonous) such as digoxin, aminoglycoside antibiotics, lithium, and chlorpropamide (Greenblatt, Sellars, & Shader, 1982).

Another mechanism of age-related changes in the response to some medicines is an apparent change in how sensitive the nerve cells are to the presence of the drug and how well they take the drug inside the nerve cell through tiny pipe-like structures called receptors which are found in the cell wall. In general, drugs acting on the central nervous system produce a stronger effect in older patients. Any drug that affects alertness, coordination, and balance will likely cause more falls and other accidents in elderly persons than in younger ones. Thus, hangover effects of sedative-hypnotic drugs and other mind-altering medicines such as ANTIPSYCHOTICS, ANTIDEPRESSANTS, and anxiolytics) are common and often more serious in the elderly. The dangerous consequences of the hangover effects, such as falls which cause broken hips, suggest, in part, that the receptors in the nerve cells in the elderly are more sensitive, even supersensitive, to the presence of these medicines. In contrast to mind-altering medications, the response of the heart to stimulation by adrenalin and other such substances is diminished in the elderly. For example, a larger dose of isoproterenol is needed to achieve the same increase in heart rate in the elderly as in the young (Vestal, Wood, & Shand, 1979).

AGING AND ADVERSE DRUG REACTIONS

In general, because of multiple and chronic diseases, older patients often take multiple prescription and over-the-counter drugs. Persons over sixty-five may take seven or more prescription drugs in addition to some over-the-counter drugs (Stewart & Cooper, 1994). However, such multiple-drug therapy predisposes the elderly to an increased risk of unintended, adverse drug reactions (ADRs). The overall incidence of ADRs in this age group is two to three times that found in young adults. Although the results of studies vary, about 20 percent of all adverse drug reactions occur in the elderly (Korrapati, Loi, & Vestal, 1992); they may result from drug overuse, from drug misuse, from slowed drug metabolism, or from slow elimination of the drug in the urine. These bad side-effects may be caused or increased by age-related chronic diseases, by intake of alcohol, and/or by incompatibilities between the foods and the medicines which the elderly person takes. Furthermore, ADRs are more severe than among young adults. At increased risk are women, persons living alone, persons suffering from multiple diseases, persons taking multiple drugs (especially prescribed by multiple physicians who do not each know what the other physicians have prescribed), persons with poor nutritional habits, and persons with less sharp sense perceptions or mental clarity. Some of the age-related physiological causes for increased levels of medicines remaining in the bloodstream and examples of increased sensitivity of nerve cells to drugs have already been discussed. The elderly who drink regularly, even if they are not alcoholic, place themselves at increased risk for bad interactions between alcohol and their medicines. This risk would be greater still if an elderly person combined alcohol, prescription medicines, and illegal drugs. Thus, since both the kidneys and the liver are often slower at eliminating substances from the body in old age, medicines should generally be taken at lower initial doses by older patients.

AGING AND ALCOHOLISM

Alcohol is an addictive drug for many individuals. Although its victims often do not recognize their alcoholism as a disease, it does meet the medical criteria for a disease: it has definite symptoms; it

is chronic; and it often progresses until it causes death—but it is treatable. It destroys its victims not only physically but also mentally, emotionally, and spiritually. Many people with this disease die from physical complications, from accidents, even from suicide. In Western society, smoking cigarettes and excessive drinking of alcohol are two of the most insidious forms of drug abuse. Yet they are often considered socially acceptable. In the United States, two-thirds of all adults use alcohol occasionally. It is estimated that between 2 and 10 percent of persons over the age of sixty suffer from heavy drinking that interferes with their health and well-being. These persons by definition suffer from alcoholism (Jinks & Raschko, 1990). If cigarette smoking is excluded, alcoholism is by far the most serious drug problem in the United States and in most other countries. Alcohol and drug abuse causes thousands of premature deaths, and the cost of complications contributes billions of dollars to any large nation's health expenditure.

Men in their sixties continue to drink at a rate that is almost equal to that of their twenties, but fortunately problem drinking decreases in the mid-seventies. The prevalence of alcoholism and problem drinking is lower in women than in men. A large majority of male alcoholics have strong family histories of alcoholism, begin problem drinking early in life, and become alcoholics not slowly and gradually but suddenly and severely. These are the early-onset or problem drinkers, called type II alcoholics (Rigler, 2000; Atkinson, 1984). It is suspected that alcoholism in this group is largely genetic. The other main group consists of later-onset alcoholics. They may drink from grief, loneliness, or a need to numb pain and to try to escape from other consequences of poor health. The many losses and stresses of later life make the elderly especially vulnerable to alcoholism and suicide (Schonfeld & Dupree, 1991). Depression puts the elderly at particular risk for suicide, especially when it is heightened by alcohol and drug abuse.

ALCOHOL AND ITS COMPLICATIONS IN THE ELDERLY

Health-care costs for a family with an alcoholic member are typically twice those for other families, and up to half of all emergency-room admissions are alcohol-related. Alcohol abuse contributes to the high health-care costs of elderly beneficiaries of government-supported health programs. In general, the medical complications of alcohol abuse observed in older individuals are the same as those found in younger alcoholics. They include alcoholic liver disease, acute and chronic inflammation of the pancreas, gastrointestinal (affecting the stomach and intestines) bleeding and other GI-tract diseases, an increased risk of infections, and disturbances in metabolism. The elderly tolerate GI bleeding and infection less well than do younger persons. They are particularly prone to vitamin deficiencies, malnutrition (that is to getting too few calories overall and consuming too little protein on a daily basis), anemia, loss of bone mass (lighter, weaker bones are more apt to break), diseases of the central and peripheral nervous systems, heart conditions, and cancer. Finally, alcohol-induced degeneration of the brain and the rest of the nervous system will add to the effects of the normal loss of nerve cells that occurs with age.

A number of studies have shown that alcohol in moderate amounts is actually a good medicine for the elderly (even the Prohibition Amendment permitted the sale of alcohol for medicinal purposes) and that it improves social interaction, mental alertness, and several signs of physical health. Alcohol is primarily a drug which depresses or deadens the central nervous system (CNS). Paradoxically, in moderate amounts it may seem to act as a stimulant with mood-elevating effects that account for its popularity. What it is actually doing in these cases is to depress or deaden inhibitions. The lack of inhibitions contributes to feelings of relaxation, confidence, and euphoria. However, alcohol abuse can result in serious damage to the brain and to the rest of the nervous system. It can cause brain tissue to shrink or waste away, unsteadiness and lack of coordination in movement, and damage to nerves throughout the body. Large doses of alcohol cause inflammation of the stomach, pancreas, and intestine that can hurt the digestion of food and the absorption of nutrients into the bloodstream. The adult population appears less knowledgeable about the many adverse effects of alcohol on health than about the effects of smoking. For example, although many people recognize that heavy alcohol drinking often leads to cirrhosis of the liver, only about one-third are aware of the association between alcohol use and cancers of the mouth and throat. Alcohol use can lessen the effectiveness of routine drug therapy or can create new

medical problems requiring additional therapy. Excessive alcohol use together with medications in the elderly can severely compromise and complicate a well-planned therapeutic program. Thus, even casual use of alcohol may be a problem for the elderly, particularly if they are taking medications that interact badly with alcohol. Difficulties can also arise from the interaction of alcohol and over-the-counter (OTC) medications. The combination of alcohol and prescribed or OTC sleeping pills, for example, could decrease intellectual function by producing an organic brain syndrome; frequent results include confusion, falls, wild swings in emotions, and other adverse drug reactions (Adams,1995).

PRECAUTIONS WHILE USING ALCOHOL

Patients with liver disease and GI ulcers should not use alcohol. Alcohol should be avoided by patients with damage, caused by previous drinking, to the heart muscle or other muscles. Clearly, it should be taken only in strict moderation or not at all. For older individuals who have no medical reasons to the contrary and who take no drugs (prescription or over-the-counter or illegal) that interact with alcohol, one drink a day is a prudent level of alcohol consumption. In general, the use of alcohol in the presence of any particular disease or medication is a matter that the physician and patient must decide.

ALCOHOL AND DRUG ABUSE IN THE ELDERLY

Alcohol, itself a drug, mixes unfavorably with many other drugs, including those purchased over the counter. In addition, use of certain prescription drugs may intensify the older person's reaction to alcohol, leading to more rapid intoxication. Alcohol, when combined substantially and quickly with certain groups of drugs, can dangerously slow down performance skills such as driving, running machinery, and even walking. It lessens judgment, and reduces alertness when taken with drugs such as those prescribed against psychosis, those meant to lessen anxiety, sedative-hypnotics, pain-killers derived from opium, antihistamines, and certain blood-pressure medicines (Table 1). Large amounts drunk quickly reduce the clearance of some drugs by the liver. In contrast, alcohol consumed on a regular basis brings on the manufacture of enzymes in the body, leading in turn to accelerated metabolism and increased clearance of some drugs, including blood-thinners, oral diabetic medicine, and medicine prescribed against convulsions. Thus, these therapeutic drugs can become less effective, so the patient needs closer monitoring. Alcohol-drug interactions do not generally result directly in death. However, there is evidence for a contributory role of alcohol in drug-related fatalities, for example, in car accidents. Anyone who drinks even moderately should ask a physician or pharmacist about possible alcohol-drug interactions.

It is very difficult to determine the actual incidence of combined drug and alcohol use by the elderly, but it is likely to be reasonably high for the following reasons: the average adult over sixty-five takes two to seven prescription medicines daily in addition to over-the-counter medications; most elderly persons do not view alcohol as a drug and therefore falsely assume that modest amounts of alcoholic beverages can do little harm to an already aged body; and few elderly persons hold to the traditional notion that mixing alcohol and medications will have bad consequences. Certainly not every medication reacts dangerously with alcohol; however, a variety of drugs interact consistently. The most dangerous of these reactions occurs when alcohol is combined with another CNS depressant. Since alcohol itself is a potent CNS depressant, its use with antihistamines, barbiturates, sedative-hypnotics, or other mind-altering drugs adds to and reinforces synergistic CNS-depressant effects, effects that in turn may inhibit one's mental alertness and even consciousness as well as one's control of movement (Gerbino, 1982). In one study, diazepam, codeine, meprobamate (Equanil), and fluorazepam (Dalmane) were the top four agents responsible for drug-alcohol interactions (Jinks & Raschko, 1990). Antihistamines, including diphenhydramine (Benadryl), dimenhydrinate (Dramamine), and most cold medications and anticholinergics such as scopolamine, which are found in over-the-counter medications, can also cause confusion in the elderly. An important consideration in the elderly is the confused and altered behavior that so regularly follows excessive consumption of alcohol. Many times, elderly alcoholics show symptoms of falls, confusion, and self-ne-

TABLE 1
Drug–Alcohol Interactions and Adverse Effects

Drug	*Adverse effects with alcohol*
Acetaminophen	Severe hepatotoxicity with therapeutic doses of acetaminophen in chronic alcoholics
Anticoagulants, oral	Decreased anticoagulant effect with chronic alcohol abuse
Antidepressants, tricyclic	Combined CNS depression decreases the psychomotor performance, especially in the first week of treatment
Aspirin and other nonsteroidal antiinflammatory drugs	Increased the possibility of gastritis and GI hemorrhage
Barbiturates	Increased CNS depression (additive effects)
Benzodiazepines	Increased CNS depression (additive effects)
Beta-adrenergic blockers	Masked signs of delirium tremens
Bromocriptine	Combined use increases the GI side effects
Caffeine	Possible further decreased reaction time
Cephalosporins	Antabuse-like reaction with some cephalosporins
Chloral hydrate	Prolonged hypnotic effect and adverse cardiovascular effects
Cimetidine	Increased CNS depressant effect of alcohol
Cycloserine	Increased alcohol effect or convulsions
Digoxin	Decreased digitalis effect
Disulfiram (Antabuse)	Abdominal cramps, flushing, vomiting, hypotension, confusion, blurred vision, and psychosis
Glutethimide	Additive CNS depressant effect
Guanadrel	Increased sedative effect and orthostatic hypotension
Heparin	Increased bleeding
Hypoglycemics, sulfonylurea	Acute ingestion-increased hypoglycemic effect of sulfonylurea drugs Chronic ingestion-decreased hypoglycemic effect of these drugs Antabuse-like reaction
Isoniazid	Increased liver toxicity
Ketoconazole	Antabuse-like reaction
Lithium	Increased lithium toxicity
Meprobamate	Synergistic CNS depression
Methotrexate	Increased hepatic damage in chronic alcoholics
Metronidazole	Antabuse-like reaction
Nitroglycerin	Possible hypotension
Phenformin	Lactic acidosis (synergism)
Phenothiazines	Additive CNS depressant activity
Phenytoin	Acutely ingested, alcohol can increase the toxicity of phenytoin Chronically ingested, alcohol can decrease the anticonvulsant effect of phenytoin
Quinacrine	Antabuse-like reaction
Tetracyclines	Decreased effect of doxycycline

Adapted from M.A. Rizack & C.D.M. Hillman (1987), Adverse interactions of drugs. In *The Medical Letter handbook of adverse drug interactions.* New York: Medical Letter.

glect. Such changes may lessen the elderly patient's ability to adhere to a prescribed treatment, and increase the risk of mistakes or mishaps in dosage (Gerbino, 1982). Some of the well-described interactions are discussed in the following sections.

ALCOHOL AND ANALGESICS

Aspirin is the active ingredient in many over-the-counter arthritis pain formulas and in numerous nonprescription combination headache-and-minor-pain products. The ability of aspirin to cause inflammation of the stomach, erosion of the GI tract, and GI bleeding is well recognized. Alcohol not only produces inflammation of the stomach but also increases the risk of GI bleeding caused by aspirin and other nonsteroidal anti-inflammatory drugs (Bush, Sholtzhauer, and Imai, 1991). Elderly people at high risk for bleeding should avoid regular use either of alcohol or of aspirin. Chronic alcohol abuse can cause poisoning of the liver in a patient taking acetaminophen (Tylenol), probably because it leads to the production of enzymes which in turn lead to the formation of poisonous intermediary breakdown products of the Tylenol.

ALCOHOL AND CENTRAL NERVOUS SYSTEM (CNS) DEPRESSANT MEDICINES

Alcohol and medicines that by themselves depress the CNS, when combined with each other, may depress the system more than either does by itself. Much controversy exists as to whether the combined effect is merely additive (what one would expect by adding the two effects together) or whether it is synergistic (greater than the sum of its two parts), whether each somehow reinforces the action of the other as well as adding its own action. When combined with CNS depressants, alcohol—even in small quantities—produces undesirable and sometimes dangerous effects. The interaction of alcohol with benzodiazepine drugs, however, may be much greater in the elderly than in the other age groups. This is especially true for diazepam (Valium) and chlordiazepoxide (Librium). Commonly observed side-effects include high blood pressure, sleepiness, confusion, and depression of the CNS that may lead to slowing down of breathing, or even to stopping it. Two drinks can bring about a drug-alcohol interaction with a medicine that depresses the CNS (Hartford & Samorajski, 1982). Therefore, as a general rule, elderly patients should be instructed to stay away from alcohol while taking such medicines, including benzodiazepines, barbiturates, muscle relaxants, and antihistamines (both by prescription and as over-the-counter cold remedies or sleeping aids). Alcohol increases the clinical effects of these drugs, which already are hazardous in a segment of the population with decreased agility and greater danger of serious complications from falls and other accidents.

ALCOHOL AND MIND-ALTERING DRUGS

When alcohol is combined with mind-altering (psychotropic) drugs such as those prescribed to fight psychosis and depression, the combined effects of alcohol and the medicine are less predictable than with other drugs. Antipsychotic drugs inhibit the metabolism of alcohol and may thus markedly increase its effects on the CNS in the elderly. Antidepressants increase the response to alcohol and harm one's control over one's mo-

TABLE 2
Drugs Producing Antabuse-like Reactions with Alcohol

Disulfiram (Antabuse)
Hypoglycemic agents
- chlorpropamide (Diabinese)
- tolbutamide (Orinase)

Other drugs
- cefamandole (Mandole)
- cefmetazole (Zefazone)
- cefoperazone (Cefobid)
- cefotetan (Cefotan)
- chloramphenicol (Chloromycetin)
- furazolidone (Furoxone)
- griseofulvin (Fulvicin)
- ketoconazole (Nizoral)
- metronidazole (Flagyl)
- monoamine oxidase inhibitors (e.g., phenelzine and tranylcypromine)
- moxalactam (Moxam)
- procarbazine (Matulane)
- quinacrine (Atabrine)

Alcohol sensitizing mushrooms
- *Coprinus atramentarius* (inky cap mushroom)
- *Clitocybe clavipes*

tions—a significant hazard in the elderly for whom falls often lead to broken bones. Depression of the CNS may range from drowsiness to coma and therefore death, because acute alcohol consumption may increase the CNS effects of antidepressants. Alcohol may also increase the risk of dangerously lowering body temperature in the elderly taking tricyclic antidepressants. Hence the avoidance of alcohol in elderly patients taking any of these drugs is a prudent recommendation (Scott & Mitchell, 1988).

ALCOHOL AND OTHER DRUGS

Many elderly patients with adult-onset (Type II) diabetes take antidiabetic pills instead of insulin. When alcohol is taken along with pills such as sulfonylureas, it may cause dangerously low levels of blood sugar, especially in patients whose diet calls for decreasing the eating of carbohydrates. Another problem associated with this combination is an Antabuse-like reaction (fortunately quite rare and usually mild), causing nausea, vomiting, headache, blurred vision, and flushing. However, symptoms of severe Antabuse-like reactions include speeding up of the heart to more than one hundred beats a minute, abdominal distress, sweating, episodes of low blood pressure, death of heart muscle, and tearing of the esophagus brought about by vomiting; psychosis may also occur, and fatal reactions have been reported. Use of alcohol at the same time with a variety of other drugs (Table 2) can also lead to an Antabuse-like reaction. Cough medicines may contain a narcotic pain-killer such as codeine in combination with antihistamines. When taken together with alcohol, these drugs are hazardous and can cause altered alertness, even loss of consciousness, and may slow down one's breathing or even stop it. Despite the fact that heart disorders are very common in older individuals, few of those who suffer from these problems modify their drinking patterns. This tendency may be dangerous, since as little as one cocktail can severely reduce the efficiency of the heart in the presence of heart disease. For example, alcohol consumption in a person suffering from angina (pain felt in the heart during physical activity) can mask the pain that might otherwise serve as a warning signal of a heart attack (Horowitz, 1975).

ALCOHOL AND ILLICIT DRUGS

Abuse of hallucinogens, illicit psychomotor stimulants and sedatives, and marijuana is uncommon in old age; use of these drugs by the elderly is almost exclusively by longstanding users of opium-like substances and by aging criminals. The low incidence of this type of substance abuse in old age may result from the fact that users of illegal drugs die young, and even from the fact that the use of such drugs by the elderly is often underreported. However, problem drinkers may abuse drugs such as sedatives, opioids, marijuana, and amphetamines. Sometimes these drugs are used in combination with alcohol; at other times, such drugs are taken in preference to alcohol, and alcohol is used only when the drug of choice is not available.

TABLE 3
Guidelines for Use of Alcohol by the Elderly

- Elderly patients are advised to avoid alcohol consumption just before going to bed in order to avoid sleep disturbances.
- Because of the potential for alcohol–drug interaction, alcohol ingestion should be avoided before driving.
- Abstinence from alcohol by elderly patients receiving CNS depressants, analgesics, anticoagulants, antidiabetic drugs, and some cardiovascular drugs is recommended.
- A doctor or pharmacist should be consulted about alcohol–drug interactions.
- Any side effect or loss of energy should be immediately reported to the physician.
- Older individuals who want to drink, have no medical contraindications, and take no medications that interact with alcohol may consider one drink a day to be a prudent level of alcohol consumption. Alcohol when taken in moderation may be useful.

Adapted from M. C. Dufour, L. Archer, & E. Gordis (1992), Alcohol and the elderly. *Clinical Geriatric Medicine, 8*(1), 127–141.

SUMMARY

Elderly people are the fastest-growing segment of world population and consume about 25 percent of all the medicines prescribed. Their capacity to handle medication differs from that of the young because of age-related changes in various systems of the body. Alcohol abuse among older people (as in any other) can lead to falls, fractures, and other similar medical complications. The addition of medications (prescription and over-the-counter) to alcohol drinking can lead to disastrous complications and even premature death. However, in the absence of any indications to the contrary such as the taking of the medications discussed above, drinking a small quantity of alcohol may be beneficial in some elderly persons. In case of doubt, the elderly and their families or caregivers are encouraged to seek the advice of the pharmacist or family physician and to follow the guidelines given in Table 3.

(SEE ALSO: *Social Costs of Alcohol and Drug Abuse*)

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AGONIST An agonist is a drug or an endogenous substance that binds to a RECEPTOR (it has affinity for the receptor binding site) and produces a biological response (it possesses intrinsic activity). The binding of a drug agonist to the receptor produces an effect that mimics the physiological response observed when an endogenous substance (e.g., hormone, NEUROTRANSMITTER) binds to the same receptor. In many cases, the biological response is directly related to the concentration of the agonist available to bind to the receptor. As more agonist is added, the number of receptors occupied increases, as does the magnitude of the response. The potency (strength) of the agonist for producing the physiological response (how much drug is needed to produce the effect) is related to the strength of binding (the affinity) for the receptor and to its intrinsic activity. Most drugs bind to more than one receptor; they have multiple receptor interactions.

(SEE ALSO: *Agonist/Antagonist (Mixed); Antagonist*)

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AGONIST-ANTAGONIST (MIXED) A mixed agonist-antagonist is a drug or receptor ligand that possesses pharmacological properties similar to both AGONISTS and ANTAGONISTS for certain RECEPTOR sites. Well-known mixed agonist-antagonists are drugs that interact with OPIOID (morphine-like) receptors. Pentazocine, nalbuphine, butorphanol, and BUPRENORPHINE are all mixed agonist-antagonists for opioid receptors. These drugs bind to the μ (mu) opioid receptor to compete with other substances (e.g., MORPHINE) for this binding site; they either block the binding of other drugs to the μ receptor (i.e., competitive antagonists) or produce a much smaller effect than that of "full" agonists (i.e., they are only partial agonists). Therefore, these drugs block the effects of high doses of morphine-like drugs at μ opioid receptors, while producing partial agonist effects at κ (kappa) and/or δ (delta) opioid receptors. Some of the mixed opioid agonist-antagonists likely produce analgesia (pain reduction) and other morphine-like effects in the CENTRAL NERVOUS SYSTEM by binding as agonists to κ opioid receptors.

In many cases, however, there is an upper limit (ceiling) to some of the central nervous system effects of these drugs (e.g., respiratory depression). Furthermore, in people physically addicted to morphine-like drugs, the administration of a mixed opioid agonist-antagonist can produce an abstinence (WITHDRAWAL) syndrome by blocking the μ opioid receptor and preventing the effects of any μ agonists (i.e., morphine) that may be in the body. Pretreatment with these drugs can also reduce or prevent the euphoria (high) associated with subsequent morphine use, since the μ opioid receptors are competitively antagonized. Therefore, the mixed opioid agonist-antagonists are believed to have less ABUSE LIABILITY than full or partial opioid receptor agonists.

As more and more subtypes of receptors are discovered in other NEUROTRANSMITTER systems

(there are now more than five serotonin receptor subtypes and five dopamine receptor subtypes), it is quite likely that mixed agonist-antagonist drugs will be identified that act on these receptors as well.

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AIDS *See* Alcohol and AIDS; Complications: Route of Administration; Injecting Drug Users and HIV; Substance Abuse and AIDS; Needle and Syringe Exchanges and HIV/AIDS

AL-ANON Al-Anon is a fellowship very similar to ALCOHOLICS ANONYMOUS (AA), but it is for family members and friends of alcoholics. Although formally totally separate from the fellowship of AA, it has incorporated into its groups the AA Twelve Steps and Twelve Traditions and AA's beliefs and organizational philosophy, but it has directed them toward helping families of alcoholics cope with the baffling and disturbing experiences of living in close interaction with an active alcoholic. In this sense, it is a satellite organization of AA (Rudy, 1986). Proselytizing organizations, such as AA, of necessity attempt to reduce, even eliminate, the ties of newcomers with other significant persons and groups who are not members. Rather than attempt to sever those bonds for prospective AA members, AA evolved Al-Anon as a way to include families into a parallel organization, and thus also initiate them into the beliefs and practices of AA. In addition, as AA expanded and more alcoholics became "recovering" ones, close relatives became aware that their own personal problems could be reduced by applying AA principles to themselves and working the Twelve Step program, even though they were not alcoholic. In 1980, there were 16,500 Al-Anon groups worldwide, including 2,300 ALATEEN groups of children of alcoholics (Maxwell, 1980).

BRIEF HISTORY

Early in the 1940s, wives started attending AA meetings and soon began to informally meet together. By the late 1940s, there were so many family members at AA affairs that the AA Board of Trustees had to decide how to manage this valuable but perplexing influx. Since relatives of AA members had already begun to hold their own meetings, the board recommended that AA meetings be only for alcoholics but that whenever family members asked to participate they should be listed at the AA General Service office as a resource. Several AA wives began their own clearinghouse committee to coordinate the approximately ninety groups already in existence. Soon there was a separate network distinct and apart from AA itself. Because they were closely related to AA, however, they decided to shorten the first two letters of "alcoholic," and the first four letters of "anonymous" into Al-Anon. This has been their name ever since.

In 1950, the anonymous Bill W., a founder of AA, persuaded his wife Lois to get involved with the fledgling Al-Anon. The rapidly accumulating lists in the General Service office were turned over to her and to an associate, Anne B., who contacted those on the list, "and soon they had more work than they could handle" (Wing, 1992:136). For two years, the two conducted their activities at Stepping Stones, the suburban home of Bill W. and Lois. In 1952 they moved to New York City, where volunteer workers could be more easily recruited. By 1989, there were over 28,000 weekly Al-Anon Family Groups, which included Alateen, worldwide. "With each meeting averaging 12–15 members, an estimated total of 336,000–420,000 visits to Al-Anon meetings occur each week" (Cermak, 1988:92). Using data from a 1984 random sample of groups conducted by the Al-Anon fellowship, it was estimated that a quarter of a million people visit an Al-Anon meeting each week.

AL-ANON'S STRATEGY

Al-Anon strives to direct its members' attention away from the active alcoholic with whom they attempt to interrelate, and toward their own behavior and emotions. In many ways, their personalities resemble those of alcoholics: they repeatedly attempt to control the feelings and behaviors of the alcoholics in their midst by simple force of personal

will—much as alcoholics attempt to control their drinking by the sheer force of their individual will. In both instances, a denial syndrome emerges in their emotional makeup that protects their compulsive drive toward continued control. In sum, family members often become codependent—as obsessed with the alcoholics' behavior as he or she is with the bottle (Huppert, 1976).

For example, the alcoholic's spouse or partner has often vainly attempted to control the drinking. Except for brief periods, most pleas have been rejected and most promises have not been kept. Often, while the alcoholic has continued to drink and enjoy the brief emotional payoffs of intoxication, the spouse or other caretaker must try to cope for both of them by running the household, rearing the children, and working steadily to earn a living. If the alcoholic does show signs of improvement in a treatment center, the spouse or life partner may resent it deeply, since strangers have done more in a short period than all the partner's efforts over the years. To alcoholics' partners and relatives it appears as if they have not been wise enough, or determined enough, or superhuman enough, to get the alcoholics in their lives to stop drinking.

Al-Anon attempts to introduce the Twelve Steps of AA into the lives of family members as a way of minimizing the resentments and obsessive-control behavior they typically display. Al-Anon emphasizes an adaptation of AA's first step: "We admit we are powerless to control an alcoholic relative, that we are not self-sufficient." Such a step is an admission that it is a waste of time to try to control what is beyond their capacities. According to this strategy, it is no longer necessary for them to deny that their control efforts are powerless, and this relieves them from the enormous sense of accumulated burden and guilt. In addition, it allows acceptance of outsider treatment and its possible success.

(SEE ALSO: *Adult Children of Alcoholics; Codependence; Families and Drug Use; Treatment Types: Twelve Steps*)

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ALATEEN Alateen is a division of the Al-Anon Family Group. Its members typically are teenagers whose lives have been impacted by someone else's problem drinking. Roughly, 59 percent are age 14 or younger, while 26 percent are ages 15 to 16 and 15 percent are age 17 or more. The problem drinkers in their lives are predominantly one or both parents, but brothers and sisters are not uncommon.

The prevailing story about the origin of Alateen is quite straightforward. Legend has it that in 1957 a 17 year old in California was attending ALCOHOLICS ANONYMOUS (AA) and AL-ANON meetings with his parents. His father had just gotten sober in AA and his mother was an active member of Al-Anon. Although the teenager decided that the Twelve Steps of AA were helping him, his mother suggested that instead of attending AA meetings he start a teenaged group and pattern it after Al-Anon. The young man found five other teenaged children of alcoholic parents and, while the adult groups met upstairs, he goth them together downstairs.

As other teenagers came forward from Al-Anon groups, the idea spread and it is estimated that today about 3,500 Alateen groups meet worldwide. In formal terms, however, these groups are an important and an integral part of Al-Anon Family Groups. They are coordinated from the Al-Anon Family Group Headquarters in New York City and tied closely to their public-information programs. Thus, Alateen uses AA's Twelve Steps, but alters step twelve to simply read "carry the message to others," rather than "to other alcoholics." Alateen groups meet in churches and schoolrooms, often in

the same building as Al-Anon, but in a different room.

Although there are a few exceptions, an active, adult member of Al-Anon usually serves as a sponsor. Also, members of Alateen can choose a personal sponsor from other Alateen members or from Al-Anon members.

Alateen enables its members to openly share their experiences and to devise ways of coping with the problem of living closely with a relative who has a drinking problem. The strategy is to change their own thinking about the problem-drinking relative. Alateen teaches that alcoholism is like diabetes—it cannot be cured, but it can be arrested. Members learn that they did not cause it, and they cannot control it or cure it. Scolding, tears, or persuasion, for example, are useless. Rather, "they learn to take care of themselves whether the alcoholic stops or not" (Al-Anon Family Groups, 1991:5). They apply the Twelve Steps to themselves—to combat their often obsessive thinking about controlling alcoholic relatives and to help them stop denying those relatives' alcoholism. In addition, they adapt and apply AA's Twelve Traditions to the conduct of their groups. For example, they practice anonymity, defining it not as secrecy, but as privacy and the lowering of competitiveness among members. A 1990 survey of Alateen members indicated an increase in the number of black, Hispanic, and other minority members.

In essence, Alateen uses the strategy of AA itself to learn how to deal with obsession, anger, feelings of guilt, and denials. Newcomers, like newcomers in AA, gain hope when they bond with other teenagers to help one another cope with alcoholic parents and other relatives with drinking problems (Al-Anon Group Headquarters, Inc., 1973).

(SEE ALSO: *Adult Children of Alcoholics; Codependence; Families and Drug Use; Treatment Types: Twelve Steps*)

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ALCOHOL AND AIDS Alcohol and AIDS (acquired immunodeficiency syndrome) from infection with HIV (human immunodeficiency virus) are each separately agents that cause suppression of immune function. Therefore regular use of alcohol by people infected with HIV should be more suppressive than either alone. Some of the most interesting questions about infection with HIV including the following: Why does progression to AIDS after HIV infection vary in length of time from under 1 year to 15 years of more? Does inhibition of mental functions by heavy alcohol use increase risky sexual behaviors and thus the chance of becoming infected with HIV? Does alcohol use or abuse affect the production of the virus or the invasion of cells by the virus?

Conventional wisdom based on long experience is that alcohol's relaxation of sexual inhibition will increase risky sexual behaviors in those who use alcohol even in moderation (2–3 drinks a day). Risky behaviors include unprotected vaginal or anal intercourse with more than one partner, unprotected intercourse with intravenous drug users, who have a high risk of HIV infection, and unprotected oral sex with potentially protected infected partners. Anyone who has used intravenous drugs or had sex with more than one person is at significant risk of infection with HIV and other pathogens.

Cocaine and other drugs of abuse are commonly used along with alcoholic beverages. They appear to synergize in terms of affecting behavior, thereby enhancing the risk of HIV infection should one be exposed. In addition, studies in the laboratory have shown that alcohol use increases the susceptibility of cells to HIV, increasing the likelihood that an invading HIV virus will successfully cause disease. Animal studies involving mice with AIDS show that alcohol and cocaine clearly suppress immune function, moreso than AIDS alone. During alcohol and cocaine use, mice infected with the virus that causes AIDS are much more susceptible to cancer and pathogens, and die sooner.

While epidemiological studies in humans have been inconclusive, animal models and test tube studies with human cells clearly demonstarte that alcohol increases the immune damage resulting from retrovirus infection. Alcohol may lower resistance to disease and tumors in AIDS patients, may accelerate development of AIDS by those who are HIV infected, may increase HIV production by

cells, and may increase cellular susceptibility to HIV infection.

AIDS patients suffer from various opportunistic pathogens, which normally do not grow sufficiently to cause disease in immunologically normal people. Alcohol and cocaine exacerbate the immune dysfunction in studies with mice. They also increase the extent of colonization in mice with AIDS by two opportunistic parasites, Giardia and Cryptosporidium. Normal mice and humans are not as are essentially resistant to such parasites. Alcohol is a cofactor that accentuates immune damage resulting from the HIV retrovirus.

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ALCOHOL- AND DRUG-FREE HOUSING Alcohol- and drug-free (ADF) housing, also called *sober housing*, or *sober living environments*, or *alcohol-free living centers*, provides domestic accommodation for people who choose to live in an environment that is free of alcohol and/or drugs. ADF housing is ordinary housing, located in residentially zoned areas, distinguished only by the residents' shared commitment not to use alcohol or other drugs.

By definition, ADF housing excludes formal treatment or recovery services on the site. The philosophic premise of ADF housing is that the sober living environment is itself the "service" for residents. ADF housing provides a setting for daily living that supports residents' efforts to maintain sobriety among themselves.

As a practical matter, the presence of on-site human services would subject ADF residences to state or local licensing of staff and to certification of their facilities. Under such circumstances, residences would be treatment facilities and no longer ADF housing. As such, they would lose protections afforded to ADF housing by the Fair Housing Amendments Act of 1988 and become subject to zoning laws that prohibit treatment programs in residential neighborhoods. The result would be the systematic exclusion of such residences from the safe and economically stable areas most conducive to recovery. Under provisions of the Fair Housing Amendments Act, *no* regulation of any ADF residence is legal unless such requirements are imposed on *all* private residences in the surrounding community with the same zoning.

Such protections aside, ADF housing is a creature of the marketplace. For it to be affordable, public sponsorship of some sort must close the gap between market rents or mortgage costs and what residents can reasonably pay. A bewildering variety of affordability strategies for rent, property, and construction-cost subsidies has been worked out for individual ADF housing projects, but very few cities or other local jurisdictions have established formal policies to create and to sustain ADF housing.

Even so, for three principal reasons, interest in affordable ADF housing has increased remarkably in the last few years. First, local units of government, special boards, and districts, are under pressure to make provisions for low-income housing. Second, recent studies indicate that affordable ADF housing helps homeless and very low-income people maintain sobriety following initial successes in treatment/recovery programs. Third, ADF housing now figures prominently in discussions of using "social-model" recovery programs as vehicles for the cost-efficient deployment of treatment and recovery services. The search for economical ways to provide such services has led health-care system planners to reduce or eliminate the use of expensive residential-treatment programs. In conjunction with outpatient treatment and adjunct health and social services, affordable ADF housing increasingly is viewed as an alternative.

ADF housing follows three simple tenets: (1) residents must remain alcohol and drug free; (2) rent must be paid on time; and (3) residents must abide by provisions of the landlord-tenant agreement. This agreement may stipulate only that tenants must refrain from disruptive behavior that provides grounds for eviction for cause in local ordinances (typically these are violence or threats of violence, illegal activity, destruction of property, and perpetuation of undue nuisances). However, it may also impose house rules that dictate curfews, limit overnight guests, restrict automobile owner-

ship, delegate house chores, and so forth. As long as the tenant's participation in the regulated household is voluntary, and as long as the rules do not violate civil rights, ADF housing may be highly structured and closely governed.

An ADF residence can be program-affiliated or free-standing. Program-affiliated sober houses are tied to the treatment and recovery orientations of particular organizations. Residents are likely to come from the sponsoring program, and so will have been exposed to the sponsor's procedures and values. Accordingly, the residence will reflect the philosophy and practices of the parent organization. Free-standing houses operate more along the lines of conventional residences and rely exclusively on self-government.

Sober housing can be run by staff or residents. Staff-run houses operate under the direct management of owners, program operators, or housing management firms. Site managers are compensated and often are recovering people who have several more years of sobriety than the residents of the house. (It should be emphasized that for obvious legal reasons, they are not "treatment personnel" and their activities do not comprise formal service interventions.) In resident-run houses, residents take full responsibility for all aspects of house operation related to maintaining sobriety: admissions, maintenance of the house's social environment, disciplinary action, community relations, and physical maintenance.

Resident-run ADF houses may be democratic or oligarchic. Democratic houses may be highly egalitarian—the residents have equal votes and share equally in houses duties, as in OXFORD HOUSES—or they may be stratified, formally or informally, by residents' seniority in sobriety or by other measures of status. Some therapeutic communities, such as Delancey Street in San Francisco, California, are oligarchic. In general, larger resident-run programs tend to be more oligarchic in nature, though some provide many opportunities for resident participation in management and operation of the house, as does Beacon House in San Pedro (Los Angeles), California.

The interests of landlords, owners, and program operators notwithstanding, the rules of ADF households seek to protect a sober environment. For their own peace of mind, residents often prefer places that impose restrictions in the service of restraint, predictability, and good order. Entrance and eviction policies are critical in this respect.

ADF housing is not magically exempt from our meaner or more self-serving impulses toward exclusiveness. However, in well-run residences, entrance decisions focus only on the capacity of an individual to benefit from the house milieu. Such decisions, in which residents usually play an important part (if only to exercise rights of refusal), consider the structure and character of the house "program" in relation to the needs of a potential resident. Thus, a prospective resident with an expressed need or desire for a highly structured environment would be discouraged from entering a house with little structured activity. All recovering people can benefit from ADF housing, regardless of their treatment histories or other circumstances. But as ADF housing represents a spectrum of possibilities for group living, particularly concerning the extent to which the environment is regulated, its potential consumers must find a good fit. Variety in ADF housing is therefore essential.

Residents are free to live in sober housing as long as they follow house rules. Any fixed time limit on length of tenancy is contrary both to the spirit and (in most communities) to the laws of residency. However, those few who violate basic house rules must leave. Although management or a residents' council makes the final determination that basic house rules have been broken, violations usually are obvious in a well-run house. If formal proceedings are necessary, the only question to be settled is whether the violation actually occurred; thereafter, commencement of the eviction process is automatic. Residents usually understand well the penalties for violations; those who know they will be evicted often choose to leave immediately.

Violation of sobriety policy is the most common reason for a tenant's eviction or voluntary separation from ADF housing. ADF houses vary somewhat in their toleration of drinking and drug use. Nearly all take a "no-slip" approach, in which a single episode of drinking or using means that the person must leave. Some permit individuals to slip once or twice before being evicted. Residents generally find that firm no-drinking, no-using policies promote a sense of equity and maintain tranquility in the house.

Some houses prefer a "client-centered" policy that permits the drinker/user to remain in contact with counselors and otherwise receive help without

having to leave the residence. The risk in such a policy is that repeated drinking or using episodes among residents will disturb the social environment of the house. Some multicomponent programs with very large facilities handle this issue by asking the drinker/user to move from the sober housing component of the program to the detoxification or primary-recovery unit. Some ADF residences that permit off-site but not on-site drinking or using have found this policy to work well to reduce chronic intoxication among those for whom continuous sobriety is not a realistic expectation.

Eviction proceedings are time consuming and filled with legal procedures that have been designed to protect and extend the rights of the resident. A signed landlord-tenant agreement that specifically proscribes drinking and/or using offers the strongest starting point for eviction; but an eviction process can sometimes drag on for weeks or months even in the most clear-cut cases. Successful ADF houses rely on resident participation in management. (A residents' manual provides a model for peer-based response to a resident's drinking or drug use.)

ADF housing works best when residents themselves actively maintain the collective sobriety of their home. Management's task is to create a living environment in which sobriety is respected and maintained by the residents. Frequent contact between management and residents, both informally and through regular house meetings, provides a medium for interaction that quickly identifies a resident who has been drinking or using. Secretiveness and the absence of communication are important signs that something isn't right.

Architectural design plays a central role in creating an ADF housing environment that promotes both open social interaction and mutual accountability. The basic floor plan of the residence makes a critical contribution. "Open" circulation systems in buildings bring people into contact with one another. Examples are open-plan houses in which space flows from one room into another; areas that have nooks and side areas where people can sit or stop to chat; and centrally located corridors with wide openings directly into the rooms they serve.

Spaces that invite social contact are called "sociopetal." They subtly but powerfully encourage people to socialize, to greet each other, to notice one another during the day. Sociopetal spaces are lively, engaging places, in stark contrast with "sociofugal" spaces whose circulation systems emphasize separation and isolation. Sociofugal circulation systems keep people apart by using long corridors such as those found in hotels and rooms isolated by stairs and such. Sociofugal spaces are dull, depressing, and sometimes disorienting or frightening to anxious people who cannot easily see what is going on in the building. Developers of successful ADF residences understand the influences of architecture. Specialized design will play an important role in the future development of ADF housing, particularly affordable ADF housing for single parents or couples with children, who require functionally different and far more space than do single people.

It is not clear how quickly or in which specific directions ADF housing development will proceed. The Anti-Drug Abuse Act of 1988 provided that every state receiving federal block grant funds for alcohol and other drug programs establish a revolving fund of at least 100,000 dollars to make start-up loans for sober housing. This was a foot in the door for ADF projects, although the relatively paltry funds involved have not provided much leverage by themselves.

In addition, government at all levels—federal, state, or local—has a strong tendency to regulate and standardize the activities it supports and to demand accountability to its agencies rather than to other relevant constituencies, such as consumers of sober housing. Any governmental attempt to regulate the environment of sober housing raises far-reaching questions about the invasion of privacy, for ADF housing is by definition *ordinary* and protected from oversight not extended to other domestic households. The philosophy of ADF housing, and more practically, its necessary and salutary diversity, is not compatible with intrusive regulation.

Still, considerable potential exists for an explosion of interest and activity. As noted, ADF residences may play an important role in the reform of the system of services for people with alcohol and drug problems. Although interest in sober housing originated in the search for ways to support homeless and very low-income people completing residential treatment and recovery programs, the useful scope of sober housing may be much broader. Combined with various services in the surrounding community, perhaps it is a good alternative to expensive, traditional forms of residential treatment.

Private insurance companies and housing entrepreneurs already are developing sober living arrangements that are attractive to the middle and upper classes, as well as to low-income people.

It is also possible that sober residences appeal to many more people than those actively engaged in treatment or recovery programs. Just as some university dormitories have become sober housing for self-selected students, perhaps sober residences will become part of a larger trend to reconfigure domestic living arrangements to fit our changed family demography and our changing styles of life. In the heyday of the temperance movement, the United States was littered with dry hotels and boarding houses that catered to a preference of style, not merely or only to prohibitionist sentiment. It is not hard to imagine a future in which likeminded citizens cause "dry" households to reappear.

(SEE ALSO: *Treatment, History of*)

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ALCOHOL This section contains articles on some aspects of alcohol, and the following topics are covered: *Chemistry and Pharmacology; Complications; History of Drinking*; and *Psychological Consequences of Chronic Abuse*. For discussions of alcoholism, its treatment, and withdrawal symptoms, see the section entitled *Alcoholism; Treatment*; and *Withdrawal*. See also the articles *Alcoholics Anonymous (AA)* and *Treatment Types: Twelve Steps*. Other articles on related topics are listed throughout the Encyclopedia.

Chemistry and Pharmacology Chemical determination has discovered five separate forms of alcohol that have little molecular variation, but enough variation to produce substantial differences in their characteristics. Occurring naturally through the fermentation of fruits, vegetables and grains exposed to the bacteria in the air, alcohol production can be expedited by producing conditions conducive to the environmental needs of the alcohol producing organisms. The form of alcohol produced intentionally for use is ethyl alcohol, also called ethanol.

People do not drink pure ethanol. Most drinks with alcoholic content do not exceed an 8 percent concentration, such as beer. Most wines do not exceed 15 percent, and most liquors are still below 50 percent, or, in the terms of the United States, 100 proof by weight or volume. Furthermore, alcoholic beverages are often diluted by water before they are consumed.

CHEMISTRY

Ethanol has a very simple molecular structure, C_2H_6O. It is composed of only two carbon atoms, six hydrogen atoms, and one oxygen atom, yet its precise mechanism of action is not fully understood. Although it is commonly believed that ethanol is useful in a number of physical ailments (as medicinal alcohol, the medieval elixir of life), in reality its uses are not therapeutic—and its chronic use is toxic.

EFFECTS ON THE BODY AND THERAPEUTIC USES

Ethanol is a general central nervous system depressant, producing sedation and even sleep at higher doses. The degree of this depression is proportional to its concentration in the blood; however, this relationship is more predictable when ethanol levels are rising than three or four hours later, when blood levels are the same but ethanol levels are falling. This variance occurs because during the first fifteen or twenty minutes after an ethanol dose, the peripheral venous blood is losing ethanol to the tissues while the brain has equilibrated with arterial blood supply. Thus, brain levels are initially higher than the venous blood levels, and since all blood samples for ethanol determinations are taken from a peripheral vein, the ethanol con-

centrations are appreciably lower than a few hours later, when the entire system has achieved equilibrium.

The reticular activating system of the brain stem is the most sensitive area to ethanol's effects; this accounts for the loss of integrative control of the brain's higher functions. Anecdotal reports of a stimulating effect, especially at low doses, are likely due to the depression of the mechanisms that normally control speech and other behaviors that evolved from training or prior experiences. However, there may be a genetic basis for this initial stimulating effect, since rodents differing genetically show differences in the degree of initial stimulation or excitement. Upon drinking a moderate amount of ethanol, humans may quickly pass through the "stimulating" phase. Memory, the ability to concentrate, and insight are affected next whereas confidence often increases as moods swing from one extreme to another. If the dose is increased, then neuromuscular coordination becomes impaired. It is at this point that drinkers may be most dangerous, since they are still able to move about but reaction times and judgment are impaired—and sleepiness must be fought. The ability to drive an automobile or operate machinery is compromised. With higher doses, general (sleep) or surgical (unconsciousness) anesthesia may develop, but respiration is dangerously depressed.

Ethanol is believed by many to have a number of medicinal (therapeutic) uses; these are mostly based on anecdotal reports and have few substantiated claims. One example of a well-known but misguided use is to treat hypothermia—exposure to freezing conditions. Although the initial effects of an alcoholic beverage appear to "warm" the patient, ethanol actually dilates blood vessels, causing further loss of body heat. Another example is its effects on sleep— it is believed that a nightcap relaxes one and puts one to sleep. Acute administration of ethanol may decrease sleep latency, but this effect dissipates after a few nights. In addition, waking time during the latter part of the night is increased, and there is a pronounced rebound insomnia that occurs once the ethanol use is discontinued. Except as an emergency treatment to reduce uterine contractions and delay birth, the therapeutic use of oral ethanol is confined to treating poisoning from methanol and ethylene glycol. Most of ethanol's therapeutic benefits are derived from applying it to the skin, since it is an excellent skin disinfectant. Ethanol can lessen the severity of dermatitis, reduces sweating, cools the skin during a fever and, when added to ointments, helps other drugs penetrate the skin. These therapeutic uses for ethanol are for acute problems only.

Until recently, it had been felt that the chronic drinking of ethanol led only to organ damage. Recent evidence suggests that low or moderate intake of ethanol (1–2 drinks per day) can indirectly reduce the risk of heart attacks. The doses must be low enough to avoid liver damage. This beneficial effect is thought to be due to the elevation of high-density lipoprotein cholesterol (HDL-C) in the blood which, in turn, slows the development of arteriosclerosis and, presumably, heart attacks. This relationship has not been proven, but has been culled from the results of several epidemiological studies.

Several mechanisms have been proposed to explain how oral ethanol exerts its effects. One is thought to be its ability to alter the fluidity of cell membranes—particularly neurons. This disturbance alters ion channels in the membrane resulting in a reduction in the propagation of neuronal transmission. The anesthetic gases share this property with ethanol. Furthermore, it has been shown that the degree of membrane disordering is directly proportional to the drug's lipid solubility. It has also been argued that such membrane effects occur only at very high doses. More recently, scientists have reported that ethanol may augment the activity of the neurotransmitter GABA by its actions on a receptor site close to the GABA receptor. The effect of this action is to increase the movement of chloride across biological membranes. Again, this effect would alter the degree to which neuronal transmission is maintained.

PHARMACOKINETICS AND DISTRIBUTION

Ethanol is quickly and rapidly absorbed from the stomach (about 20%) and from the first section of the small intestines (called the duodenum). Thus the onset of action is related in part to how fast it passes through the stomach. Having food in the stomach can slow absorption because the stomach does not empty its contents into the small intestines when it is full. However, drinking on an empty stomach leads to almost instant intoxication because the ethanol not absorbed in the stomach

passes directly to the small intestines. Maximal blood levels are achieved about thirty to ninety minutes after ingestion. Ethanol mixes with water quite well, and so once it enters the body it travels to all fluids and tissues, including the placenta in a pregnant woman. After about twenty to thirty minutes for equilibration, blood levels are a good estimate of brain levels. Ethanol freely enters all blood vessels, including those in the small air sacs of the lungs. Once in the lungs, ethanol exchanges freely with the air one breathes, making a breath sample a good estimate of the amount of ethanol in one's body. A breathalyzer device is often used by police officers to detect the presence of ethanol in an individual.

Between 90 and 98 percent of the ethanol dose is metabolized. The amount of ethanol that can be metabolized per unit of time is roughly proportional to the individual's body weight (and probably the weight of the liver). Adults can metabolize about 120 mg/kg/hr which translates to about thirty ml (one ounce) of pure ethanol in about three hours. Women generally achieve higher alcohol blood concentrations than do men, even after the same unit dose of ethanol, because women have a lower percentage of total body water but also because they may have less activity of alcohol-metabolizing enzymes in the wall of the stomach. The enzymes responsible for ethanol and acetaldehyde metabolism—alcohol dehydrogenase and aldehyde dehydrogenase, respectively—are under genetic control. Genetic differences in the activity of these enzymes account for the fact that different racial groups metabolize ethanol and acetaldehyde at different rates. The best-known example is that of certain Asian groups who have a less active variant of the aldehyde dehydrogenase enzyme. When they consume alcohol, they accumulate higher levels of acetaldehyde than do Caucasian males, for example; this causes a characteristic response called "flushing," actually a type of hot flash with reddening of the face and neck. Some experts believe that the relatively low levels of alcoholism in such Asian groups may be linked to this genetically based aversive effect.

TOXIC EFFECTS

Chronic consumption of excessive amounts of ethanol can lead to a number of neurological disorders, including altered brain size, permanent memory loss, sleep disturbances, seizures, and psychoses. Some of these neuropsychiatric syndromes, such as Wernicke's encephalopathy, Korsakoff's psychosis, and polyneuritis can be debilitating. Other, less obvious problems also occur during chronic ethanol consumption. The chronic drinker usually fails to meet basic nutritional needs and is often deficient in a number of essential vitamins, which can also lead to brain and nerve damage.

Chronic drinking also causes damage to a number of major organs. Permanent alterations in brain function have already been discussed. By far, one of the most important causes of death in alcoholics (other than by accidents) is liver damage. The liver is the organ that metabolizes ingested and body toxins; it is essential for natural detoxification. Alcohol damage to the liver ranges from acute fatty liver to hepatitis, necrosis, and cirrhosis. Single doses of ethanol can deposit droplets of lipids, or fat, in the liver cells (called hepatocytes). With an accumulation of such lipid, the liver's ability to metabolize other body toxins is reduced. Even a weekend drinking binge can produce measurable increases in liver fat. It was found that liver fats doubled after only two days of drinking; blood ethanol levels ranged between twenty and eighty mg/dl, suggesting that one need not be drunk in order to experience liver damage.

Alcohol-induced hepatitis is an inflammatory condition of the liver. The symptoms are anorexia, fever, and jaundice. The size of the liver increases, and its ability to cleanse the blood of other toxins is reduced. Cirrhosis is the terminal and most dangerous type of liver damage. Cirrhosis results after many years of intermittent bouts with hepatitis or other liver damage, resulting in the death of liver cells and the formation of scar tissue in their place. Fibrosis of the blood vessels leading to the liver can result in elevated blood pressure in the veins around the esophagus, which may rupture and cause massive bleeding. Ultimately, the cirrhotic liver fails to function and is a major cause of death among alcoholics. Although only a small percentage of drinkers develop cirrhosis, it appears that a continuous drinking pattern results in greater risk than does intermittent drinking, and an immunological factor may be involved.

The role of poor nutrition in the development of some of these disorders is well recognized but not very well understood. Ethanol provides 7.1 kilocalories of energy per gram. Thus, a pint of whiskey

provides around 1,300 kilocalories, which is a substantial amount of raw energy, although devoid of any essential nutrients. These nutritional disturbances can exist even when food intake is high, because ethanol can impair the absorption of vitamins B_1 and B_{12} and folic acid. Ethanol-related nutritional problems are also associated with magnesium, zinc, and copper deficiencies. A chronic state of malnutrition can produce symptoms that are indistinguishable from chronic ethanol abuse.

Fetal alcohol syndrome (FAS) was recognized and described in the 1980s. Children of chronic drinkers are born deformed; the abnormality is characterized by reduced brain function as evidenced by a low IQ and smaller than usual brain size, slower than normal growth rates, characteristic facial abnormalities (widely spaced eyes and flattened nasal area), other minor malformations, and developmental and behavioral problems. Fetal malnutrition caused by ethanol-induced damage to the placenta can also occur, and fetal immune function appears to be weakened, resulting in the child's greater susceptibility to infectious disease. Depending on the population studied, the rate of FAS ranges from 1 in 300 to 1 in 2,000 live births; however, the incidence is 1 in 3 infants of alcoholic mothers. Even today, it is not known if there is a safe lower limit of ethanol that can be consumed by pregnant women without risk of having a child with FAS. The lowest reported level of ethanol that resulted in FAS was about 75 ml (2.5 oz.) per day during pregnancy. Among alcoholic mothers, if drinking during pregnancy is reduced, then the severity of the resulting syndrome is reduced.

TOLERANCE, DEPENDENCE, AND ABUSE

Tolerance, a feature of many different drugs, develops rather quickly to many of ethanol's effects after frequent exposure. When tolerance develops, the dose must be increased to achieve the original effect. Ethanol is subject to two types of tolerance: tissue (or functional) tolerance and metabolic (or dispositional) tolerance. Metabolic tolerance is due to alterations in the body's capacity to metabolize ethanol, which is achieved primarily by a greater activity of enzymes in the liver. Metabolic tolerance only accounts for 30 to 50 percent of the total response to alcohol in experimental conditions. Tissue tolerance, however, decreases the brain's sensitivity to ethanol and may be quite extensive. The development of tolerance can take just a few weeks or may take years to develop, depending on the amount and pattern of ethanol intake. As with other central nervous system depressants, when the dose of ethanol is increased to achieve the desired effects (e.g., sleep), the margin of safety actually decreases, as the dose comes closer to producing toxicity and the brain's control of breathing becomes depressed.

Like tolerance, dependence on ethanol can develop after only a few weeks of consistent intake. The degree of dependence can be assessed only by measuring the severity of the withdrawal signs and symptoms observed when ethanol intake is terminated. Victor and Adams (1953) provided perhaps one of the best descriptions of the clinical aspects of ethanol dependence. Patients typically arrive at the hospital with the "shakes," sometimes so severe that they cannot perform simple tasks by themselves. During the next twenty-four hours of their stay in the hospital, an alcoholic might experience hallucinations, which typically are not too distressing. Convulsions, however, which resemble those in people with epilepsy, may occur in susceptible individuals about a day after the last drink. Convulsions usually occur only in those who have been drinking extremely large amounts of ethanol. If the convulsions are severe, the individual may die. Many somatic effects, such as nausea, vomiting, diarrhea, fever, and profuse sweating are also part of alcohol withdrawal. Some sixty to eighty-four hours after the last dose, there may be confusion and disorientation; more vivid hallucinations may begin to appear. This phase of withdrawal is often called the delirium tremens, or DTs. Before the days of effective treatment, a mortality rate of 5 to 15 percent was common among alcoholics whose withdrawal was severe enough to cause DTs.

TREATMENT FOR ALCOHOL DEPENDENCE

The first step in treating alcoholics is to remove the ethanol from the system, a process called detoxification. Since rapid termination of ethanol (or any other central nervous system depressant) can be life threatening, people who have been using high doses should be slowly weaned from the ethanol by giving a less toxic substitute depressant. Ethanol itself cannot be used because it is eliminated from the

body too rapidly, making it difficult to control the treatment. Although barbiturates were once employed in this capacity, the safer benzodiazepines have become the drugs of choice. Not only do they prevent the development of the potentially fatal convulsions, but they reduce anxiety and help promote sleep during the withdrawal phase. New medications are constantly being tested for their abilities to aid in the treatment of alcohol withdrawal.

Once a person has become abstinent, various methods can be used to maintain abstinence and encourage sobriety—some are pharmacologic and others are through social-support networks or formal psychological therapies. One type of treatment involves making drinking an adverse toxic event for the individual, by giving a drug such as DISULFIRAM (Antabuse) or citrated CALCIUM CARBIMIDE, which inhibits the metabolism of acetaldehyde and causes facial flushing, nausea, and rapid heartbeat. When ethanol is ingested by someone on disulfiram, the acetaldehyde levels rise very high, very quickly. Disulfiram has not been successful in maintaining abstinence in all patients, however.

Many support groups are available to help people remain abstinent. ALCOHOLICS ANONYMOUS (AA) is one of the most widely known and available; it is structured around a self-help philosophy. The AA program emphasizes total avoidance of alcohol and any medication. Instead it relies on a "buddy" or "sponsor" system, providing support partners who are personally experienced with alcoholism and alcoholism recovery. A number of other types of psychological and behavioral approaches to treatment also exist.

(SEE ALSO: *Accidents and Injuries from Alcohol; Alcoholism; Fetus, Effects of Drugs on; Complications; Social Costs of Alcohol and Drug Abuse*)

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Complications Through their ethanol (alcohol) content, alcoholic beverages significantly affect the body's cellular function as well as its cognitive actions. Many of these effects are the consequence of a complex set of biochemical reactions, long-term exposure to ethanol with an accumulation of damage that is manifested in diverse ways, or the result of increased incidence or severity of major disease states, including AIDS, CANCER, or heart disease. However, some effects of ethanol are immediate and do not require prolonged exposure, nor are they induced as the end product of many physiological changes. For example, ethanol induces changes in cell membranes' fluidity by mixing with the lipids there. The membrane changes inhibit neurological functions and thus can cause car ACCIDENTS. All of these can occur with a single exposure and thus could be considered a direct effect of the ethanol in alcoholic beverages.

ALCOHOL METABOLISM

Ethanol Absorption and Metabolism. Because the ethanol molecule has a hydroxyl group, its metabolism involves dehydrogenase enzymes. After some metabolism in the stomach and intestine, it is transported to the liver for further metabolism. Alcohol dehydrogenase produces acetaldehyde, which causes many of the indirect effects attributed to ethanol. Because females metabolize alcohol less efficiently in the stomach wall than males, their exposure can be higher, with more direct consequences, from the same amount of alcohol consumption. Ethanol is also metabolized by the liver cells' MEOS system. Ethanol also affects the transportation of proteins across membranes in the cell. Thus aldehyde dehydrogenase's transportation into the mitochondria from the cell's cytoplasm is retarded. This reduces the oxidation of acetaldehyde to acetic acid, and increases ethanol's

indirect effects by altering its metabolism and that of its metabolites. Acetaldehyde is very reactive with proteins. Thus increased levels result in damage to proteins with which it reacts. As many are vital for cell function, cell death or dysfunction occurs. This damage persists for the life of the protein or cell.

Alcohol and Nutrition. Alcohol has major effects when consumed frequently or in high amounts by affecting the frequency and quality of foods consumed. This directly affects the amounts of vitamins and minerals that are consumed and available for absorption. The long-term consequences involve undernutrition, nutritional deficiencies, and ultimately malnutrition. Ethanol also directly affects the absorption of vitamin A, betacarotene (a vitamin A precursor), vitamin B_1 (thiamine), folate, vitamin E, vitamin D, and folate. Vitamins are critical for many enzymatic reactions, so ethanol causes indirect effects by altering vitamin levels. Acute alcohol ingestion changes many vitamin metabolic pathways. Folate and vitamin A metabolism can cause increased urinary excretion. Thiamine deficiency is responsible for a severe neurological consequence of excessive alcohol use—WERNICKE'S SYNDROME.

ACTIONS OF ALCOHOL ON THE BRAIN

The molecular site of alcohol's action on neurons is unknown. Alcohol may work by perturbing lipids in the cell membrane of the NEURON, interacting directly with the hydrophobic region of neuronal membrane proteins, or interacting directly with a lipid-free enzyme protein in the membrane. Ethanol alters the function of neuron-specific proteins. For example, evidence suggests that the activity of the chloride ion channel linked to the A-type receptor of the GABA NEUROTRANSMITTER increases during exposure to intoxicating amounts of alcohol. Acute exposure to alcohol effects the actions of GLUTAMATE, the major excitatory transmitter in the mammalian central nervous system. Chronic exposure to alcohol can result in TOLERANCE for and PHYSICAL DEPENDENCE on the drug. Tolerance is recognized as a chronic drinker's ability to consume increasing amounts of alcohol without displaying gross signs of intoxication. Alcohol's effects on stress may be regulated by the combination of its effect on information processing. Thus it can decrease internal conflicts and block inhibitions, thereby making social behaviors more extreme.

Free Radical Generation by Alcohol. Free radicals are a highly reactive oxygen species. They are important components of the body's host defense, yet in high levels can cause tissue damage. Cytochrome P-450 is an oxidizing system that generates free radicals from ethanol. The reactive oxygen species include superoxide and hydrogen peroxide. They react with DNA, protein, and lipids. Products of the free radical reactions include lipid peroxides; thus alcohol's production of free radicals indirectly initiates cancer, heart disease, and other major health problems. Free radicals are produced in higher levels when ethanol and acetaldehyde begin to accumulate in cells and saturate dehydrogenases. Then other products, such as free radicals and cocaethylene (when cocaine is present), are produced.

Cholesterol and Fatty-Acid Production from Alcoholic Beverages. Excessive ethanol intake leads to formation of ethanol- and fatty-acid-containing ethyl esters, produced by synthases. Thus tissues containing large amounts of synthases, such as the heart, would be more likely to be damaged. These products can adversely affect protein synthesis, alter cell membranes that contain large amounts of normal lipids, and suppress energy production by the cells' mitochondria. Cholesterol esterase connects cholesterol to fatty acids, thus producing fatty-acid cholesterol esters. When ethanol is present, the esterase produces fatty-acid ethyl esters with a reduction of cholesterol. Ethanol consumption modifies components of cell membranes, phospholipids, through the phospholipase D. The importance of these changes is poorly defined and understood.

Cocaethylene and Drug Metabolism. When alcohol and cocaine are ingested together, the "high" is accentuated. Ethanol can react with COCAINE via the enzyme cocaine esterase, producing a potentially toxic product, COCAETHYLENE. This enzyme inactivates cocaine in the absence of ethanol. Metabolism of cocaine and other drugs occurs in large part via cytochrome P-450 IIEI. It is increased by chronic alcohol consumption. This cytochrome oxidizes ethanol in the liver as well as many other compounds, including cocaine and the pain killer acetaminophen. Oxidative products of cytochrome P-450 are more toxic than the parent compounds, and thus can accentuate liver damage.

Metabolism of Protein. Consumption of alcoholic beverages affects the metabolism of ethanol and other alcohols, and alters the NADH/NAD ratio—the ratio of reduced nicotinamide adenine dinucleotide to oxidized nicotinamide adenine dinucleotide—which influences lipid, vitamin, and protein metabolism, membrane composition and function, and energy production. Such changes lead to indirect effects including cell damage, undernutrition, and weight loss. Chronic alcohol beverage use reduces type II muscle fibers, reducing the capacity for prolonged muscle activity and thus the ability to exercise, run, or do physical work. Loss of this fiber produces muscle pain, weakness, and damage. Reduced type II fibers may be due to lower RNA, which would indicate less protein synthesis.

Metabolism of Lipids and Fats. Fat and lipid functions and metabolism are altered by alcohol consumption. High alcohol intakes result in changes in the ratio of NADH/NAD +, which rduces breakdown of fats and lipids. The accumulated lipids are stored in the liver, producing a fatty liver. The NADH/NAD + ratio also inhibits synthesis of cholesterol and related steroid hormones. Thus production of progesterone and androstenedione are reduced by alcohol use. Such changes may be the cause of hypogonadism in males who consume alcohol chronically. Lipoprotein lipase is inhibited by ethanol, thus reducing removal of long acyl chains from lipids. In heart muscle this reduces available energy and could be a component of heart disease. Lipoproteins are transport molecules for fats, including cholesterol, in plasma fluids. Alcohol increases both low- and high-density lipoproteins, which could be beneficial and damaging, respectively, to the heart.

Lipids in the Function and Composition of Cell Membranes. Membranes have lipids and proteins as major components. Ethanol clearly affects lipids and membranes directly and indirectly. Alcohol affects cell membranes directly by its entry into them. Its physical characteristics modify arrangement of lipids in the cell membrane, and hence should affect cell function directly. For example, electrolyte balance within all cells is produced by sodium and potassium ion transportation. High alcohol intake reduces the ion transporters, which causes cells to take up water and thus to swell, affecting function. In addition, cells respond to hormones and other chemicals in the plasma outside the cell membrane by signal transduction. These signals regulate the functions of the various cell types, affecting overall physiology of the body. Important enzymes in this process include phospholipases. Ethanol acts like hormones and signal molecules, changing membrane phospholipases, which should modify cell function.

ALCOHOL TRAUMA, ACCIDENTS, AND BEHAVIORAL EFFECTS

Alcohol is directly involved in injuries by altering neurological function in ways that lead to motor vehicle ACCIDENTS, plane crashes, drownings, SUICIDE, and homicide. It appears to play a role in both unintentional and intentional injuries. Nearly one-fourth of suicide victims, one-third of homicide victims, and one-third of unintentional injury victims have high BLOOD ALCOHOL CONCENTRATIONS. Alcohol was a factor in half of fatal traffic crashes and 5 percent of all deaths. It causes premature mortality (not including deaths from indirect, biochemical changes induced by long-term exposure).

Alcohol and Auto Accidents. Alcohol consumption directly and promptly impairs many perceptual, cognitive, and motor skills needed to operate motor vehicles safely. Although in 1989 traffic fatalities involving at least one intoxicated driver or nonoccupant (pedestrian or other) decreased by half, 22,413 people were killed in alcohol-related motor vehicle crashes, representing approximately half of all traffic fatalities. The decrease in alcohol's involvement may be partially attributed to changes in MINIMUM DRINKING AGE LAWS.WOMEN drivers are involved in half as many alcohol-related car accidents as men. Impaired drivers arrested are significantly more hostile; they have greater psychopathic deviance, nontraffic arrests, and frequency of impaired driving, and they drink more than drunk drivers caught in roadblocks. Thus, impaired driving and alcohol-related accidents are part of problematic behaviors that can be directly modified by ethanol.

Alcohol and Airplane Accidents. Alcohol has not been shown to have caused a U.S. commercial airline accident. However, it plays a direct and prominent role in general aviation accidents. Pilot function is impaired by cognitive, perceptual, and psychomotor changes due to ethanol use. Positional

alcohol nystagmus may contribute to many aviation crashes involving spatial disorientation.

Alcohol and Water Accidents. Alcohol is associated with between half and two-thirds of adult drownings. Alcohol is also important in water-related spinal cord injuries.

Alcohol and Sexual Behavior. Via neurological changes, alcohol impairs rational thought, thus decreasing behavioral inhibitions. Alcohol is an excuse for behavior that violates social norms. Problem drinking behavior is associated with sexually transmitted disease.

Alcohol and Violence. High alcohol consumption reduces inhibition, impairs moral judgment, and increases aggression; thus there is greater likelihood of homicide or assault resulting from fights. Frequently, alcohol use has occurred in situations that emerge spontaneously from personal disputes. Alcohol is linked to a high proportion of violence, with perpetrators more often under the influence of alcohol than victims. Very high rates of problem drinking are reported among both property and violent offenders.

(SEE ALSO: *Accidents and Injuries from Alcohol; Complications*)

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History of Drinking The key to the importance of alcohol in history is that this simple substance, presumably present since bacteria first consumed some plant cells nearly 1.5 billion years ago, has become so deeply embedded in human societies that it affects their religion, economics, age, sex, politics, and many other aspects of human life. Furthermore, the roles that alcohol plays differ, not only from one culture to the next but even within a culture over time. A single chemical compound, used (or sometimes emphatically avoided) by a single species, has resulted in a complex array of customs, attitudes, beliefs, values, and effects. A brief review of the history of this relationship illustrates both unity and diversity in the ways people have thought about and treated alcohol. Special attention is paid to the United States as a case study of particular interest to many readers.

THE QUESTION OF ORIGINS

Ethanol, the form of alcohol desired for use to produce favorable effects, is both created naturally, in the fermentation of exposed fruits, vegetables, and grains that have become overripe, and through the intervention of people who accelerate the process by controlling the conditions of fermentation. If we assume that it is ethanol that produces a host of presumed favorable effects, as well as alcohol-related problems, then the logic of labeling some drinks "alcoholic" can be justified. It is important to remember, however, that labels are merely a social convention. No matter how great its alcohol content may be, wine is thought of as "food" in much of France and Italy—as is beer in Scandinavia and Germany. Similarly, in the United States, many people who regularly drink beer in considerable quantities do not think of themselves as using alcohol. Some fruit juices, candies, and desserts come close to having enough alcohol to be so labeled, but they are not. Thus many of the concerns that people have about alcohol relate more to their expectations than to the actual pharmacological or biochemical impact that the substance would have on the human body.

According to the Bible, one of the first things Noah did after the great Flood was to plant a vineyard (Genesis 9:21). According to the predynastic Egyptians, the great god Osiris taught people to make beer, a substance that had great religious as well as nutritional value for them. Similarly, early Greeks credited the god Dionysus with bringing them wine, which they drank largely as a form of worship. In Roman times, the god Bacchus was thought to be both the originator of wine and always present within it. It was a goddess, Mayahuel, with 400 breasts, who supposedly taught the Aztecs how to make pulque from the sap

Police and alleged moonshiners pose with one of the two giant stills taken during the raid at a downtown Pittsburgh office building, July 22, 1922. (© Bettmann/CORBIS)

of the century plant; that mild beer is still important in the diet of many Indians in Mexico, where it is often referred to as "the milk of our Mother." In each of these instances, whether the giver was male or female, alcohol was viewed as supernatural, reflecting deep appreciation of its important roles in nourishing and comforting people.

Anthropologists often treat myths as if they were each people's own view of history, but clearly it would be difficult to take all myths at face value. We cannot know when or where someone first sampled alcohol, but we can imagine that it might well have been just an attempt to make the most of an overripe fruit or a soured bowl of gruel. The taste, or the feeling that resulted, or both, may have been pleasant enough to prompt repetition and then experimentation. Probably it happened not just once but various times, independently, at a number of different places, just as did the beginnings of agriculture.

PREHISTORY AND ARCHEOLOGY

Although it is impossible to say where or when *Homo sapiens* first sampled alcohol, there is firm evidence, from chemical analysis of the residues found in pots dating from 3500 B.C., that wine was already being made from grapes in Mesopotamia (now Iran). This discovery makes alcohol almost as old as farming, and, in fact, beer and bread were first produced at the same place at about the same time from the same ingredients. We know little about the gradual process by which people learned to control fermentation, to blend drinks, or to store and ship them in ways that kept them from souring, but the distribution of local styles of wine vessels serves as a guide to the flow of commerce in antiquity.

It would be misleading to think of early wines and beers as similar to the drinks we know today. In a rough sense, the distinction between them is that a wine is generally derived from fruits or berries, whereas a beer or ale comes from grain or a grain-based bread. Until as recently as A.D. 1700, both were often relatively dark, dense with sediments, and extremely uneven in quality. Usually handcrafted in small batches, home-brewed beers tend to be highly nutritious but to last only a few days before going sour (i.e., before all the fermenting sugars and alcohol are depleted and become vinegar). By contrast, homemade wines have relatively little in the way of vitamins or minerals but can last a long time if adequately sealed.

In Egypt between 2700 and 1200 B.C., beer was not only an important part of the daily diet; it was also buried in royal tombs and offered to the deities. Many of the paintings and carvings in Egyptian tombs depict brewing and drinking; early papyri include commercial accounts of beer, a father's warning to his student son about the danger of drinking too much, praises to the god who brought beer to earth, and other indications of its importance and effects.

The earliest written code of laws we know, from Hammurabi's reign in Babylon around 2000 B.C., devoted considerable attention to the production and sale of beer and wine, including regulations about standard measures, consumer protection, and the responsibilities of servers.

In ancient Greece and Rome (roughly 800 B.C.–A.D. 400), there was wider diffusion of grape-growing north and westward in Europe, and wine was important for medicinal and religious purposes, although it was not yet a commonplace item in the diet of poor people. The much-touted sobriety of the Greeks is presumably based on their custom of diluting wine with water and drinking only after meals, in contrast to neighboring populations who often sought drunkenness through beer as a transcendental state of altered consciousness. Certainly heavy drinking was an integral part of the religious orgies that, commemorating their deities, we now call "Dionysiac" (or, in the case of Rome, "Bacchic"). The temperate stereotype also overlooks the infamous chronic drunkenness of Alexander the Great. Born in Macedon, in 356 B.C., he

managed to conquer most of the known world in his time, by 325 B.C., bringing what are now Egypt and most of the Middle East under the rule of Greece before he died in 323 B.C.

Romans were quick to point out how their relative temperance contrasted with the boisterous heavy drinking of their tribal neighbors in all directions, whom they devalued as the bearded ones, "barbarians." To a remarkable degree, the geographic spread of Latin-based languages and grape cultivation coincided with the spread of the Roman Empire through Europe and the accompanying diffusion of the Mediterranean diet—rich in carbohydrates and low in fats and protein—with wine as the usual beverage. In striking contrast were non-Latin speakers, who were less reliant on bread and pasta and without olive oil; they drank beers and meads, with drunkenness more common. Plato considered wine an important adjunct to philosophical discussion, and St. Paul recommended it as an aid to digestion.

The Hebrews established a new pattern around the time of their return from the Babylonian exile, and the construction of the Second Temple (c. 500 B.C.). Related to a new systematizing of religious practices was a strong shift toward family rituals, in which the periodic sacred drinking of wine was accompanied by a pervasive ethic of temperance, a pattern that persists today and often marks drinking by religious Jews as different from that of their neighbors. Early Christians (many of whom had been Jews), praised the healthful and social benefits of wine while condemning drunkenness. A majority of the many biblical references to drinking are clearly favorable, and Jesus' choice of wine to symbolize his blood is perpetuated in the solemn rite of the *Eucharist*, which has become central to practice in many Christian churches as *Holy Communion*.

In the Iron Age in France (c. 600 B.C.), distinctive drinking vessels found in tombs strongly suggest that political leadership involved the redistribution of goods to one's followers, with wine an important symbol of wealth. Archeologists have learned so much about the style and composition of pots made in any given area that they can often trace routes and times of trade, military expansion, or migrations by noting where fragments of drink containers are found. Although we know little about Africa at that time, we assume that mild fermented home brews (such as banana beer) were commonplace, as they were in Latin America. In Asia, we know most about China, where as early as 2000 B.C. grain-based beer and wine were used in ceremony, offered to the gods, and included in royal burials. Most of North America and Oceania, curiously, appear not to have had any alcoholic beverages until contact with Europeans.

Alcohol in classical times served as a disinfectant and was thought to strengthen the blood, stimulate nursing mothers, and relieve various ills, as well as to be an ideal offering to both gods and ancestral spirits. Obviously, drink and drinking had highly positive meanings for early peoples, as they do now for many non-Western societies.

FROM 1000 TO 1500

The Middle Ages was marked by a rapid spread of both Christianity and Islam. Large-scale political and economic integration spread with them to many areas that had previously seen only local warring factions, and sharp social stratification between nobles and commoners was in evidence at courts and manors, where food and drink were becoming more elaborate. National groups began to appear, with cultural differences (including preferred drinks and ways of drinking) increasingly noted by travelers, of whom there were growing numbers. Excessive drinking by poor people was often criticized but may well have been limited to festive occasions. With population increases, towns and villages proliferated, and taverns became important social centers, often condemned by the wealthy as subverting religion, political stability, and family organization. But for peasants and craftspeople, the household was still often the primary economic unit, with home-brewed beer being a major part of the diet.

During this period, hops, which enhanced both the flavor and durability of beer, were introduced. In Italy and France, wine became even more popular, both in the diet and for expanding commerce. Distillation had been known to the Arabs since about 800, but among Europeans, a small group of clergy, physicians, and alchemists monopolized that technology until about 1200, producing spirits as beverages for a limited luxury market and for broader use as a medicine. Gradual overpopulation was halted by the Black Death (a pandemic of bubonic plague), and schisms in the Catholic church resulted in unrest and political struggles later in this period.

Across northern Africa and much of Asia, populations, among whom drinking and drunkenness had been lavishly and poetically praised as valuable ways of altering consciousness, became temperate and sometimes abstinent, in keeping with the tenets of Islam and the teachings of Buddha and of Confucius. China and India both had episodes of prohibition, but neither country was consistent. In the Hindu religion, some castes drank liquor as a sacrament, whereas others scorned it—vivid proof that a culture, in the anthropological sense (as a set of beliefs and practices that guide one through living), is often much smaller than a religion or a nation, although we sometimes tend to think of those larger entities as more homogenous than they really are.

As the Middle Ages gave way to the Renaissance, both the population and the economy expanded throughout most of Europe. Because the Arabs (who had ruled from 711 to 1492) had been expelled from Spain and Portugal, they cut off overland trade routes to Asia; European maritime exploration therefore resulted in increasing commerce all around the coasts of Africa. The so-called Age of Exploration led to the startling encounter with high civilizations and other tribal peoples who had long occupied North America, Central America, and South America. Ironically, alcoholic beverages appear to have been totally unknown north of Mexico, although a vast variety of beers, chichas, pulques, and other fermented brews were important in Mexico as foods, as offerings to the gods and to ancestral spirits, and as shortcuts to religious ecstasy—if we assume that Native Americans then lived much as those who were soon to be described by the European conquerors and missionaries.

Throughout sub-Saharan Africa, we assume, home-brewed beers were plentiful nutritious, and symbolically important, as they came to be described in later years.

During the Middle Ages, drinking was treated as a commonplace experience, little different from eating, and drunkenness appears to have been infrequent, tolerated in association with occasional religious festivals and of little concern in terms of health or social welfare. Alcoholic beverages themselves were becoming more diverse but still were thought to be invigorating to humans, appreciated by spirits, and important to sociability.

FROM 1500 TO 1800

Wealth and extravagance were manifest in the rapidly growing cities of Europe, but so were poverty and misery, as class differences became even more exaggerated. The Protestant Reformation, which set out to separate sacred from secular realms of life, seemed to justify an austere morality that included injunctions against celebratory drunkenness. If the body was the vessel of the spirit, which itself was divine, one should not desecrate it with long-term heavy drinking. Puritans viewed intoxication as a moral offense—although they drank beer as a regular beverage and appreciated liquor for its supposed warming, social, and curative properties. Public drinking establishments evolved, sometimes as important town meeting places and sometimes as the workers' equivalent of social clubs, with better heat and lighting than at home, with news and gossip, games and companionship. COFFEE, TEA, and CHOCOLATE were also introduced to Europe at this time, and each became popular enough to be the focus of specialized shops. But each was also suspect for a time, while physicians debated whether they were dangerous to the health; clergy debated their effects on morality; and political and business leaders feared that retail outlets would become breeding places of crime, labor unrest, and civil disobedience. Brandies (*brantwijns*, liquor distilled from wines to be shipped as concentrates) spread among the aristocracy, and champagne was introduced as a luxury beverage (wine), as were various cordials and liqueurs. Brewing and wine-making grew from cottage industries to major commercial ventures, incorporating many technical innovations, quality controls, and other changes.

The "gin epidemic" in mid-eighteenth-century London is sometimes cited as showing how urban crowding, cheap liquor, severe unemployment, and dismal working conditions combined to produce widespread drinking and dissolution, but the vivid engravings by William Hogarth may exaggerate the problem. At the same time, the artist extolled beer as healthful, soothing, and economically sound. In France, even peasants began to drink wine regularly. In 1760, Catherine the Great set up a state monopoly to profit from Russia's prodigious thirst, and Sweden followed soon after.

Throughout Latin America and parts of North America, the Spanish and Portuguese con-

quistadors found that indigenous peoples already had home brews that were important to them for sacred, medicinal, and dietary purposes. The Aztecs of Mexico derived a significant portion of their nutritional intake from pulque but reserved drunkenness as the prerogative of priests and old men. Cultures throughout the rest of the area similarly used chicha or beer made from maize, manioc, or other materials. The Yaqui (in what is now Arizona) made a wine from cactus as part of their rain ceremony, and specially made chicha was used as a royal gift by the Inca of Peru. Religious and political leaders from the colonial powers were ambivalent about what they perceived as the risks of public drunkenness and the profits to be gained from producing and taxing alcoholic beverages. A series of inconsistent laws and regulations, including sometime prohibition for Indians, were probably short-lived experiments, affected by such factors as local revolts and different opinions among religious orders.

As merchants from various countries competed to gain commercial advantage in trading with the various Native American groups of North America, liquor quickly became an important item. It has become popular to assume that Native Americans are genetically vulnerable to alcohol, but some tribes (such as Hopi and Zuni) never accepted it, and others drank with moderation. The Seneca, in New York state, are an interesting case study, because they went from having no contact with alcohol through a series of stages culminating in a religious ban. When brandy first arrived, friends would save it for an unmarried young man, who would drink it ceremoniously to help in his required ritual quest for a vision of the animal that would become his guardian spirit. In later years, drinking became secular, with anyone drinking and boisterous brawling a frequent outcome. In 1799, when a tribal leader, who was already alcoholic, had a very different kind of vision, he promptly preached abstention from alcohol, an end to warfare, and devotion to farming—all of which remain important today in the religion that is named after him, Handsome Lake.

Throughout the islands of the Pacific, local populations reacted differently to the introduction of alcohol, sometimes embracing it enthusiastically and sometimes rejecting it. Eskimos were generally quick to adopt it, as were Australian Aborigines, to the extent that some interpret their heavy drinking as an attempt to escape the stresses of losing valued parts of their traditional ways of life. Detailed information about the patterns of belief and behavior associated with drinking among the diverse populations of Asia and Africa vividly illustrates that alcohol results in many kinds of comportment—depending more on sociocultural expectations than any qualities inherent in the substance.

In what is now the United States, colonial drinking patterns reflected those of the countries from which immigrants had come. Rum (distilled from West Indies sugar production) became an important item in international trade, following routes dictated by the economic rules of the British Empire. In the infamous Triangle Trade, captive black Africans were shipped to the West Indies for sale as slaves. Many worked on plantations there, producing not only refined sugar, a sweet and valuable new faddish food, but also molasses, much of which was shipped to New England. Distillers there turned it into rum, which was in turn shipped to West Africa, where it could be traded for more slaves. During the American Revolution (1775/6–1783), however, that trade was interrupted and North Americans shifted to whiskey. Farmers along what was then the frontier, still east of the Mississippi, were glad to have a profitable way of using surplus corn that was too bulky to bring to distant markets. After the war, when the first federal excise tax was imposed (on whiskey) in 1790, to help cut the debt of the new United States, producers' anger about a tax increase was expressed in the Whiskey Rebellion of 1794. To quell the uprising, federal troops (militia) were used for the first time. At about the same time, Benjamin Rush, a noted physician and signer of the Declaration of Independence, started a campaign against long-term heavy drinking as injurious to health.

Evidently, alcohol plays many roles in the history of any people, and changes in attitudes can be abrupt, illustrating again the importance that social constructions of reality have in relation to drinking.

THE 1800s

The large-scale commercialization of beer, wine, and distilled liquor spread rapidly in Europe as many businesses and industries became international in scope. Large portions of the European proletariat were no longer tied to the land for subsistence, and new means of transportation facili-

tated vast migrations. The industrial revolution was not an event but a long process, in which, for many people, work became separated from home. The arbitrary pace imposed by wage work contrasted markedly with the seasonal pace of traditional agrarianism.

In some contexts neighbors still drank while helping each other—as, for example, in barn-raising or reciprocal labor exchange during the harvest. But for the urban masses, leisure and a middle class emerged as new phenomena. Drinking, which became increasingly forbidden in the workplace as dangerous or inefficient, gradually became a leisure activity, often timed to mark the transition between the workday and home life. As markets grew, foods became diverse, so that beers and ciders (usually hard) lost their special value as nourishing and energizing.

In Europe, political boundaries were approximately those of the twentieth century; trains and steamships changed the face of trade; and old ideas about social inequality were increasingly challenged. Alcohol lost much of its religious importance as ascetic Protestant groups, and even fervent Catholic priests in Ireland, associated crime, family disruption, unemployment, and a host of other social ills with it, and taxation and other restrictions were broadly imposed. In Russia, the czar ordered prohibition, but only briefly as popular opposition mounted and government revenues plummeted. Those who paid special attention to physical and mental illnesses were quick to link disease with long-term heavy drinking, although liquor remained an important part of medicine for various curative purposes. A few institutions sprang up late in the nineteenth century to accommodate so-called inebriates, although there was little consensus about how or why drinking created problems for some people but not for others, nor was there any systematic research.

A wave of mounting religious concern that has been called the "great awakening" swept over the United States early in the 1800s, and, by 1850, a dozen states had enacted prohibition. Antialcohol sentiment was often associated with opposition to slavery. The local prohibition laws were repealed as the Civil War and religious fervor abated, and hard drinking became emblematic of cowboys, miners, lumberjacks, and other colorful characters associated with the expanding frontier. Distinctions of wealth became more important than those of hereditary social status, and a wide variety of beverages, of apparatus associated with drinking, and even of public drinking establishments accentuated such class differences.

Near the end of the century, another wave of sentiment against alcohol grew, as large numbers of immigrants (many of them Catholic and anything but ascetic) were seen by Protestant Yankees as trouble—competing for jobs, changing the political climate, and challenging old values. Coupled with this attitude was enthusiasm for "clean living," with an emphasis on natural foods, exercise, fresh air and water, loose-fitting clothing, and a number of other fads that have recently reappeared on the scene.

Native American populations, in the meantime, suffered various degrees of displacement, exploitation, and annihilation, sometimes as a result of deliberate national policy and sometimes as a result of local tensions. The stereotype of the drunken Indian became embedded in novels, news accounts, and the public mind, although the image applied to only a small segment of life among the several hundred native populations. Some Indians remained abstinent and some returned to abstinence as part of a deliberate espousal of indigenous values—for example, in the Native American Church, using PEYOTE as a sacrament, or in the sun dance or the sweat lodge, using asceticism as a combined religious and intellectually cleansing precept.

From Asia, Africa, and Oceania, explorers, traders, missionaries, and others brought back increasingly detailed descriptions of non-Western drinking practices and their outcomes. It is from such ethnographic reports—often sensationalized—that we can guess about the earlier distribution of native drinks and can recognize new alcoholic beverages as major commodities in the commercial exploitation of populations. Although some of the sacramental associations of traditional beverages were transferred to new ones, the increasing separation of brewing from the home, the expansion of a money-based economy, and the apparent prestige value of Western drinks all tended to diminish the significance of home brews. In African mines, Latin American plantations, and even some U.S. factories, liquor became an integral part of the wage system, with workers required to accept alcohol in lieu of some of their cash earnings. In some societies where drinking had been unknown before Western colonization, the rapid spread of

alcohol appears to have been an integral part of a complex process that eroded traditional values and authority.

THE TWENTIETH CENTURY

It has been said that the average person's life in 1900 was more like that of ancestors thousands of years earlier than like that of most people today. The assertion certainly applies to the consumption of liquor. Pasteurization, mass production, commercial canning and bottling, and rapid transport all transformed the public's view of beer and wine in the twentieth century. The spread of ideas about individualism and secular humanism loosened the hold of traditional religions on the moral precepts of large segments of the population. New assumptions about the role of the state in support of public health and social welfare now color our expectations about drinking and its outcomes. Mass media and international conglomerates are actively engaged in the expansion of markets, especially into developing countries.

World War I prompted national austerity programs in many countries that curtailed the diversion of foodstuffs to alcoholic beverages but didn't quite reach the full prohibition for which the United States became famous. Absinthe was thought to be medically so dangerous that it was banned in several European countries, and Iceland banned beer but not wine or liquor. Sweden experimented with rationing, and the czar again tried prohibition in Russia. The worldwide economic depression of the 1930s appears to have slowed the growth of alcohol consumption, which grew rapidly during the economic boom that followed World War II. The Scandinavian countries, beset by a pattern of binge drinking, often accompanied by violence, tried a variety of systems of regulation, including state monopolies, high taxation, and severely restricted places and times of sale, before turning to large-scale social research.

While several Western countries were expanding their spheres of influence in sub-Saharan Africa, they agreed briefly on a multinational treaty that outlawed the sale of alcoholic beverages there, although they did nothing to curtail production of domestic drinks by various tribal populations. A flurry of scientific analyses of indigenous drinks surprised many by demonstrating their significant nutritional value, and more detailed ethnographic studies showed how important they were in terms of ideology, for vows, communicating with supernatural beings, honoring ancestors, and otherwise building social and symbolic credit—among native societies not only in Africa but also in Latin America and Asia. Closer attention to the social dynamics of drinking and other aspects of culture showed that the impact of contact with Western cultures is not always negative and that for many peoples the role of alcohol remained diverse and vital.

In the United States, a combination of religious, jingoistic, and unsubstantiated medical claims resulted in the enactment of nationwide prohibition in 1919. Often called "the noble experiment," the Eighteenth Amendment to the Constitution was the first amendment to deal with workaday behavior of people who have no important public roles. It forbade commercial transaction but said nothing about drinking or possession. Most authorities agree that, during the early years, there was relatively little production of alcoholic beverages and not much smuggling or home production. It was not long, however, before illegal sources sprang up. Moonshiners distilled liquor illegally, and bootleggers smuggled it within the U.S. or from abroad. Speakeasies sprang up as clandestine bars or cocktail lounges, and a popular counterculture developed in which drinking was even more fashionable than before prohibition. Some entrepreneurs became immensely wealthy and brashly confident and seemed beyond the reach of the law, whether because of superior firepower or corruption or both. The government had been suffering from the loss of excise taxes on alcohol, which accounted for a large part of the annual budget. The stock-market crash, massive unemployment, the crisis in agriculture, and worldwide economic depression aggravated an already difficult situation, and civil disturbances spread throughout the country. Some of the same influential people who had pressed strongest for prohibition reversed their stands, and the Twenty-first Amendment, the first and only repeal to affect the U.S. Constitution, did away with federal prohibition in 1933. Although the national government retained close control over manufacturing and distribution to maximize tax collection, specific regulations about retail sales were left up to the states. An odd patchwork of laws emerged, with many states remaining officially dry, others allowing local option by counties or towns, some imposing a state monopoly, some requiring that drinks be served

with food and others expressly prohibiting it, some insisting that bars be visible from the street and others the opposite, and so forth. The last state to vote itself wet was Mississippi, in 1966, and many communities remain officially dry today. The older federal law prohibiting sales to Indians was not repealed until 1953, and many Indian reservations and Alaska native communities remain dry under local option.

The experience of failed prohibition in the U.S. is famous, but a similar combination of problems with lawlessness, corruption, and related issues led to repeal, after shorter experiments, in Iceland, Finland, India, Russia, and parts of Canada, demonstrating again that such drastic measures seem not to work except where supported by consensus and religious conviction (e.g., Saudi Arabia, Iran, and Ethiopia). It is ironic that some Indian reservations with prohibition have more alcohol-related deaths than those without. A more salutary recent factor is the growth of culturally sensitive programs of prevention and treatment that are being developed, often by the communities themselves, for Indian and other minority populations.

In the middle decades of the twentieth century, a number of alcoholics formed a mutual-help group, modeled on the earlier Washingtonians, and ALCOHOLICS ANONYMOUS has grown to be an international fellowship of individuals whose primary purpose is to keep from drinking. At about the same time, scientists from a variety of disciplines started studying various aspects of alcohol, and our knowledge has grown rapidly. Because of the large constituency of recovering alcoholics, the subject has become politically acceptable, and the disease concept has overcome much of the moral stigma that used to attach to alcoholism. Establishment of a National Institute on Alcohol Abuse and Alcoholism in 1971 signaled a major government commitment to the field, and its incorporation among the National Institutes of Health in 1992 indicates that concerns about wellness have largely displaced theological preoccupations.

Consumption of all alcoholic beverages increased gradually in the U.S. from repeal until the early 1980s, with marked increase following World War II, although it never reached more than one-third of what is estimated for the corresponding period a century earlier. Around 1980, sales of spirits started dropping and have continued to do so. A few years later, wine sales leveled off and have gradually fallen since; beer sales also appear to have passed their peak even more recently. These reductions occurred, despite increasing advertising, along with a return of the "clean living" movement and another shift toward physical exercise, less-processed foods, and concern for health. Linked with the reduction in drinking, what some observers call a "new temperance movement" has emerged, in which individuals not only drink less but call for others to do the same; the decline would be enforced by laws and regulations that would increase taxes, index liquor prices to inflation, diminish numbers and hours of sales outlets, require warning labels, ban or restrict advertising, and otherwise reduce the availability of alcohol. Such a "public health approach" is by no means limited to the U.S.; its popularity is growing throughout Europe and among some groups elsewhere, even as alcohol consumption continues to rise in Asia and many developing countries.

CURRENT IMPLICATIONS

A quick review of the history of alcohol lends a fresh perspective to the subject. The vast literature on ethnographic variation among populations demonstrates the different ways in which peoples, widely separated geographically and historically, have used and thought about alcohol. The idea of alcohol as being implicated in a set of problems is peculiar to the recent past and is not yet generally accepted in many areas.

What some observers call the "new temperance movement" and others call "neoprohibitionism" is a recent phenomenon that grew out of Scandinavian social research. The conclusion, on the basis of transnational comparisons, was that there appeared to be some relationship between the amount of alcohol people drink and a broad range of what the researchers called "alcohol-related problems" (including spouse abuse, child neglect, social violence, psychiatric illness, a variety of organic damages, and traffic fatalities). The vague and general findings gradually came, through a process of misquotation and paraphrasing, to be treated as a pseudoscientific iron-clad law, to the effect that problems are invariably proportionate to consumption, so that the most effective way to diminish problems would be to cut drinking. This approach is sometimes called the "control of consumption model," or the "single distribution model" (refer-

ring to the fact that heavy drinkers are on the same distribution-of-consumption curve as low and moderate drinkers, with no clear points that would objectively divide the groups).

This movement is not restricted to the U.S. and Scandinavia, however. The World Health Organization of the United Nations called for a worldwide reduction, by 25 percent, of alcohol consumption during the last decade and a half of the twentieth century, recommending that member countries adopt similar policies. Throughout most of central and western Europe and North America, sales have fallen markedly, although the opposite trend can be seen in much of the third world. An ironic development has been recent loosening of controls in Scandinavian countries, traditionally the exemplars of that approach, while controls are being introduced and progressively tightened in southern Europe, where drinking has traditionally been an integral part of the culture.

The European Community standardization of tariffs may result in further changes soon. A more realistic way of lessening whatever problems may be related to alcohol consumption would appear to be the "sociocultural model" of prevention, emphasizing, on the basis of cross-cultural experience, that people can learn to drink differently, to expect different outcomes from drinking, and actually to find their expectancies fulfilled. This program would not be quick or easy, requiring intensive public education, but it seems more feasible than simply curtailing availability—in which case those who enjoy moderate drinking would be inconvenienced but those who insist on drinking heavily would continue to do so. Concern over policy is not only directed at helping individuals who may have become dependent; it also has the aim of making life safer and more pleasant for all. The history of alcohol indicates that problems are by no means inherent in the substance but, rather, are mediated by the individual user and by social norms.

(SEE ALSO: *Beers and Brews; Temperance Movement; Treatment, History of*)

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Psychological Consequences of Chronic Abuse Chronic alcohol abuse (heavy drinking over a long period) can lead to numerous adverse effects—to direct effects such as impaired attention, increased ANXIETY, depression, and increased risk-taking behaviors—and to indirect affects such as impaired cognitive abilities, which may be linked to nutritional deficiencies from long-term heavy drinking.

A major difficulty in describing the effects of chronic alcohol abuse is that many factors interact with such consumption, resulting in marked individual variability in the psychological consequences. In addition, defining both what constitutes chronicity and abusive drinking in relation to resulting behavioral problems is not simply a function of frequency and quantity of alcohol consumption. For some individuals, drinking three to four drinks per day for a few months can result in severe consequences, while for others, six drinks per day for years may not have any observable effects. One reason for this variability is related to genetic differences in the effects of alcohol upon an individual. While not all of the variability can be linked to genetic predispositions, it has been demonstrated that the interactions between individual genetic characteristics and environmental factors are important in determining the effects of chronic alcohol consumption.

Other factors to consider when assessing the effects of chronic drinking relate to the age and sex of the drinker. In the United States, heavy chronic drinking occurs with the greatest frequency in white men, ages nineteen to twenty-five. For the majority of individuals in this group, heavy drinking declines after age twenty-five to more moderate levels and then decreases to even lower levels after age fifty. As might be expected, the type and extent of psychological consequences depend on the age of the chronic drinker. Research has indicated that younger problem drinkers are more likely to perform poorly in school, have more arrests, and be more emotionally disturbed than older alcoholics. Also, younger drinkers have more traffic accidents, which may result from a combination of their heavy drinking and increased risk-taking behavior. Many of the more serious consequences of chronic alcohol use occur more frequently in older drinkers—individuals in their thirties and forties; these include increased cognitive and mental impairments, divorce, absenteeism from work, and suicide. Chronic drinking in women tends to occur more frequently during their late twenties and continuing into their forties—but the onset of alcohol-related problems appears to develop more rapidly in women than in men. In a study of ALCOHOLICS ANONYMOUS members, women experienced serious problems only seven years after beginning heavy

drinking, as compared to an average of more than eleven years for men.

Black and Hispanic men in the United States tend to show prolonged chronic drinking beyond the white male's reduction period during his late twenties. Thus, for many of the effects of chronic drinking discussed below, age, sex, and duration of drinking are important factors that mediate psychological consequences.

NEGATIVE CONSEQUENCES

In the early 1990s, it was estimated that between 7 and 10 percent of all individuals drinking alcoholic beverages will experience some degree of negative consequences as a result of their drinking pattern. Most people believe that chronic excessive drinking results in a variety of behavioral consequences, including poor work/school performance and inappropriate social behavior. These two behavioral criteria are used in most diagnostic protocols when determining if a drinking problem exists. Several surveys have found that heavy chronic drinking does produce a variety of school- and job-related problems. A survey of personnel in the U.S. armed services found that for individuals considered heavy drinkers, 22 percent showed job-performance problems. Health professionals also show high rates of alcohol problems, with a late 1980s British survey indicating that physicians experience such problems at a rate of 3.8 times that of the general population. A variety of surveys have consistently shown that chronic excessive drinking leads to loss of support by moderate-drinking family and friends. The dissolution of marriage in couples in which only one member drinks is estimated to be over 50 percent. Often the interpersonal problems that surround a problem drinker can lead to family violence; a 1980s study found that more than 44 percent of men with alcohol problems admitted to physically abusing their wives, children, or significant-other living partners. Survey data also indicate that people who use alcohol frequently are more likely to become involved with others who share their drinking patterns—particularly those who do not express concern about the individual's excessive and altered behavior that results from drinking. This increased association with fellow heavy drinkers as one's main social-support network can itself result in increased alcohol use.

The interaction between the social setting and the individual, the current level of alcohol intoxication, and past drinking history all play a role in the psychological consequences of chronic heavy drinking. It is impossible to determine which changes in behavior result only from the use of alcohol.

Depression. One major psychological consequence resulting from heavy chronic drinking for a subpopulation of alcohol abusers (predominantly women) is the feeling of loss of control over one's life, commonly manifested as depression. (While not conclusive, some studies suggest that the menstrual cycle may be an additional factor for this population.) In many cases, increased drinking occurs as the depression becomes more intense. It has been postulated that the increased drinking is an attempt to alleviate the depression. Unfortunately, since this "cure" usually has little success, a vicious drinking cycle ensues. While no specific causality can be assumed, research on suicide has indicated that chronic alcohol abuse is involved in 20 to 36 percent of reported cases. The level of suicide in depressed individuals with no alcohol abuse is somewhat lower—about 10 percent. At this time, it is not clear if the chronic drinking results in depression or if the depression is a pre-existing psychopathology, which becomes exacerbated by the drinking behavior. The rapid improvement of depressive symptoms seen in the majority of alcoholics within a few weeks of detoxification (withdrawal) suggests that, for many, depressive symptoms are reflective of toxic effects of alcohol. Regardless of the mechanism, it appears that the combination of depression and drinking can be a potent determinant for increasing the potential to commit suicide.

Aggression. For another subpopulation of chronic alcohol abusers (mainly young men), an increase in overall aggressive behaviors has been reported. Again, there is an indication that these individuals represent a group that has an underlying antisocial personality disorder, which is exacerbated by chronic alcoholic drinking.

Sex Drive. Although it is often assumed that alcohol increases sexual behavior, chronic excessive use has been found to decrease the level of sexual motivation in men. In some gay male populations, where high alcohol consumption is also associated with increased high-risk sexual activity, this decrease in sex drive does not appear to result; however, for many chronic male drinkers, a long-term

consequence of heavy drinking is reduced sexual arousal and drive. This may be the combined result of the decreased hormonal levels produced by the heavy drinking and the decline of social situations where sexual opportunities exist.

Cognitive Changes. Perhaps the best-documented changes in psychological function resulting from chronic excessive alcohol use are those related to cognitive functioning. While no evidence exists for any overall changes in basic intelligence, specific cognitive abilities become impaired by chronic alcohol consumption. These most often include visuo-spatial deficits, language (verbal) impairments, and in more severe cases, memory impairments (alcoholic amnestic syndrome). A specific form of dementia, alcoholic dementia, has been described as occurring in a small fraction of chronic alcohol abusers. The pattern and nature of the cognitive effects, as measured on neuropsychiatric-assessment batteries in chronic alcohol abusers, exhibit a wide variety of individual patterns. Also, up to 25 percent of chronic alcoholics tested show no detectable cognitive deficits. Although excessive alcohol use has been clearly implicated in such deficits, a variety of coexisting lifestyle behaviors might be responsible for the cognitive impairments observed. For example, poor eating habits leading to vitamin deficiencies result in cognitive deficits similar to those observed in some alcohol abusers. Head trauma from accidents, falls, and fights (behaviors frequent in heavy drinkers) may also produce similar cognitive deficits. Therefore, it is extremely difficult to determine the extent to which alcohol abuse is directly responsible for the impairments—or if they are a result of the many alterations in behaviors that become part of the heavy-drinker lifestyle.

The specific psychological consequences of chronic drinking are complex and variable, but there is clear evidence that chronic abuse of alcohol results in frequent and often disastrous problems for the drinker and for those close to him or her.

(SEE ALSO: *Aggression and Drugs: Research Issues; Complications*)

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ALCOHOL BEVERAGE CONTROL *See* Distilled Spirits Council

ALCOHOL DEPENDENCE SYNDROME *See* Diagnosis of Drug Abuse; Disease Concept of Alcoholism and Drug Abuse

ALCOHOLICS ANONYMOUS (AA) This is a fellowship of problem drinkers, both men and women, who voluntarily join in a mutual effort to remain sober. It was started in the United States in the 1930s and has been maintained by alcohol-troubled people who had themselves "hit bottom"—they had discovered that the troubles associated with their drinking far outweighed any pleasures it might provide. AA serves, without professional guidance, a significant minority of the population of alcoholics in the United States. Various professionally oriented treatments serve other significant minorities of alcoholics.

AA is not the only hope for alcoholics; nor is it everything they need. Nevertheless, its program and meetings have restored thousands of alcoholics to abstinence, both in the United States and in many other countries. In 1992, the General Service Office of AA, located in New York City, reported a worldwide total of 87,403 AA groups, 48,747 of them in the United States, with an additional 1,783 in U.S. correctional facilities, and 5,173 in Canada, leaving 31,700 in other countries. The report estimated there were almost 2 million individual members in these groups worldwide; over half (1,079,719) lived in the United States.

THE TWELVE STEPS OF ALCOHOLICS ANONYMOUS

AA's program for remaining sober is called the Twelve Steps. They are:

1. We admitted we were powerless over alcohol—that our lives had become unmanageable.
2. Came to believe that a Power greater than ourselves could restore us to sanity.
3. Made a decision to turn our will and our lives over to the care of God *as we understood Him*.
4. Made a searching and fearless moral inventory of ourselves.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. Were entirely ready to have God remove all these defects of character.
7. Humbly asked Him to remove our shortcomings.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.
10. Continued to take personal inventory and when we were wrong promptly admit it.
11. Sought through prayer and meditation to improve our conscious contact with God *as we understood Him*, praying only for knowledge of His will for us and power to carry that out.
12. Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics and to practice these principles in all our affairs.

The steps are based on suggestions gleaned from the collective experiences of members about how they achieved sobriety—and then maintained it. In this sense, AA is a collectivity of mutual help groups more than it is discrete individuals engaging in self-help. At meetings both open to the public and "closed" (for members only), the Twelve Steps are closely examined, and members frankly tell their own versions of their drinking histories—their AA "stories"—and describe how the AA program helped them to achieve sobriety.

Membership in AA depends on an individual's declaration of intention to stop drinking. An AA group comes into being when two or more "drunks" join together to practice the AA program. "Loners" are relatively few, but some exist. There are no dues or fees for membership; AA is self-supporting and is not associated with any sect, denomination, political group, or other organization. It neither endorses nor opposes any causes. These points, and other basic descriptions of AA, appear on the first page of AA's monthly magazine, *The Grapevine*. Although AA is not set up as a centralized organization, a commonly shared set of traditions guides their meetings and treatment strategies. For example, one of the Twelve Traditions sets forth AA's singleness of purpose—to help alcoholics achieve and sustain sobriety; another tradition underscores the necessity for the anonymity of members, as a way to avoid personality inflation and to promote humility. Over time, the Twelve Traditions have come to be as vital a part of AA as the Twelve Steps. They are:

1. Our common welfare should come first; personal recovery depends upon A.A. unity.
2. For our group purpose there is but one ultimate authority . . . a loving God as He may express Himself in our group conscience. Our leaders are but trusted servants. . . . They do not govern.
3. The only requirement for A.A. membership is a desire to stop drinking.
4. Each group should be autonomous except in matters affecting other groups or A.A. as a whole.
5. Each group has but one primary purpose . . . to carry its message to the alcoholic who still suffers.
6. An A.A. group ought never endorse, finance, or lend the A.A. name to any related facility or outside enterprise, lest problems of money, property and prestige divert us from our primary purpose.
7. Every A.A. group ought to be fully self-supporting, declining outside contributions.
8. Alcoholics Anonymous should remain forever nonprofessional, but our service centers may employ special workers.
9. A.A., as such, ought never be organized; but we may create service boards or committees directly responsible to those they serve.
10. Alcoholics Anonymous has no opinion on outside issues; hence the A.A. name ought never be drawn into public controversy.
11. Our public relations policy is based on attraction rather than promotion; we need always

maintain personal anonymity at the level of press, radio, and films.

12. Anonymity is the spiritual foundation of all traditions ever reminding us to place principles before personalities.

Written in 1939, *Alcoholics Anonymous* (Alcoholics Anonymous World Services, 1939, 1955, 1976) is the basic text that outlines the experiences of the first 100 members in staying sober. It is fondly referred to as "The Big Book."

ORIGINS

The unlikely coming together of the two cofounders of AA, "Bill W." and "Dr. Bob" (William Griffith Wilson, a stockbroker, and Robert Holbrook Smith, a surgeon)—both pronounced alcoholics—is probably the most concrete event in AA's origins. Anonymity was basic, but there were other factors, among them Bill W.'s experiences prior to contact with Dr. Bob. Had it not been for the readiness these experiences generated in Bill W. to interact in a unique way with Dr. Bob, their initial meeting might have turned out to be the fifteen-minute encounter the doctor had initially planned.

First, Bill W. had reached a point of profound hopelessness. Second, he had had a chance encounter with an old drinking friend—Ebby T.—who despite strong and similar feelings of hopelessness had achieved considerable sobriety. Third, Ebby T. attributed this accomplishment to the Oxford Group, a nondenominational movement with no membership lists, rules, or hierarchy. It embraced specific ideas that would soon find their way into AA practice: For example, members alone were powerless to solve their own problems; they must carefully examine their behavior and try to make restitution to others they had damaged; and they practiced helping others, resisting personal prestige in the process.

Next, a severe relapse had forced Bill W. into a hospital, where he had visits from Ebby T. and where Bill W. longed for the sobriety Ebby seemed to have. Following a cry of lonely desperation and agony, he reports that "the result was instant, electric, beyond description. The place lit up, blinding white . . . came the tremendous thought. 'You are a free man'" (W.W., 1949, p. 372). As a result of these accumulated experiences, Bill W. decided on two strategies. One was to take his story to other alcoholics and the other was to become an evangelist, because of his spiritual experience. Even though he brought many alcoholics home with him and preached at them, he utterly failed and almost returned to drinking himself. But his account underscores how he came to realize that "to talk with another alcoholic, even though I failed with him, was better than to do nothing" (W.W., 1949, p. 374).

Two other experiences accumulated. He discovered that some medical authorities considered alcoholism a disease. Almost instantly, he replaced evangelism with science. Finally, in an effort to recoup some financial losses, he pursued a slim business opportunity in Akron, Ohio, on May 11 and 12, 1935. An Episcopalian minister there put him in contact with an Oxford Group member who, in turn, arranged for a meeting the next day with Dr. Bob. Abandoning his evangelical approach, he used his newly found scientific/disease approach with the doctor. An immediate rapport developed between the two men, and they talked until late into the night. Dr. Bob was, in effect, Bill W.'s first follower (Trice & Staudenmeier, 1989). Dr. Bob had only one "slip" during the next month, but soon thereafter the two began working together on other alcoholics, using the sickness/scientific approach. By August 1936, AA meetings, within an Oxford Group context, were being held both in Akron and New York City. Soon, however, both Bill W. and Dr. Bob decided to sever their relationship with the Oxford Groups, and the small AA groups were on their own. By 1940, their newly formed board of trustees listed twenty-two cities in which groups were well established and holding weekly meetings. Soon thereafter, *The Saturday Evening Post*, a popular magazine with a wide circulation, published an article simply entitled "Alcoholics Anonymous." It proved to be a compelling media event for the AA program, and a flood of positive responses and new groups resulted. Ever since then, AA growth has steadily expanded.

Two coordinating groups have acted to link together the thousands of AA local groups in the United States and abroad. In AA's first year the founders, along with members of the first New York City group, formed a tax-free charitable trust with a board of trustees composed of both alcoholic and nonalcoholic members. It acted as a mechanism for the collection and management of voluntary contri-

butions and as a general repository of the collective experience of all AA groups.

Today the board of trustees consists of fourteen alcoholic and seven nonalcoholic members who meet quarterly. At an annual conference, specific regions elect the alcoholic board members for four-year terms. The board appoints the nonalcoholic members for a maximum of three terms of three years each. An annual conference was established in 1955 at AA's Twentieth Anniversary Convention. It expresses to the trustees the opinions and experiences of AA groups throughout the movement. A General Service office (GSO) in New York City interprets and implements the deliberations of these two groups on a daily basis.

THE PROCESS OF AFFILIATION

Affiliation is a process, not a single, unitary happening within AA. Its elements and phases act to select and make ready certain alcoholics and problem drinkers for affiliation, leaving behind others with less readiness. The process begins before the problem drinker ever goes to a meeting (Trice, 1957). If the person has heard favorable hearsay about AA; if long-time drinking friendships have faded; if no will-power models of self-quitting have existed in the immediate background; and if the drinker has formed a habit of often sharing troubles with others—the stage is set for affiliation. It is further enhanced if, upon first attending meetings, the person has had experiences leading to the decision that the troubles associated with drinking far outweigh the pleasures of drinking (i.e., "hitting bottom"). Typically, this means that affiliates, contrasted with nonaffiliates, had a longer and more severe history of alcoholism—and those with more severe alcohol problems are more likely to make consistent efforts to affiliate than are those with less severe problems (Trice & Wahl, 1958; Emerick, 1989b).

Five other specific phases follow from those forces that make for commitment to the AA program: (1) first-stepping, (2) making a commitment, (3) accepting one's problem, (4) telling one's story, and (5) doing twelfth-step work (Rudy, 1986). First-stepping involves the initial contact with AA; it often entails orientation meetings that dwell on the group's notions of alcoholism as a disease and on step one in the twelve-step program: "We admitted we were powerless over alcohol . . . that our lives had become unmanageable." The newcomers also will probably become associated with an AA guide, who may soon become the newcomer's sponsor. Quick action by the AA group—closeness of initial contact—to include the newcomer increases the likelihood of affiliation (Sonnenstuhl & Trice, 1987). This expresses itself in pressing any obviously interested newcomer into a challenge of "ninety meetings in ninety days." In effect, the receiving group seeks to keep a close watch over the newcomer, gently forcing the person to forego other commitments and increase commitments to the AA program.

Decisive third and fourth phases are the acceptance of telling one's drinking story, with the beginning phrase, "I'm Chris X and I'm an alcoholic." Throughout the initial weeks and months, newcomers are gently and sometimes bluntly pressed to realize that they *are* alcoholics. They are encouraged to "go public" and tell their stories before the entire group at an open meeting. In numerous instances, newcomers may already have decided that they are alcoholic. In other cases, it may be a lengthy process of self-examination before this identity transformation occurs. In still others, it may never transpire, making them suspect as real AAs. In any event, the public telling of one's story is an act of commitment that symbolizes a conversion of self into a genuine AA member. Members counsel newcomers on the appropriate first time to tell their stories, and their narrations are cause for many congratulations. Much applause typically attends this open act of commitment.

A final phase involves the literal execution of the program's twelfth step: "Having had a spiritual awakening as a result of these Steps, we tried to carry this message to alcoholics, and to practice these principles in all our affairs." In essence, doing twelfth-step work exemplifies one of AA's basic philosophies, namely, one is never recovered from the disease; one is only "recovering." As a consequence, a member can maintain sobriety only by remaining active in AA and by steadily engaging in carrying the program to those who are still active alcoholics. In short, by doing twelfth-step work, members reinforce their membership and the new definition of self.

Throughout this affiliation process, another dynamic is also at work—"slipping,"—a relapse into drinking by a recovering AA member. After reviewing six relevant studies, plus a summary of his

own fieldwork with AA, Rudy (1980, p. 728) reported that among both newly committed and longer-term members "slipping is a common occurrence, but it is possible that it serves a function in A.A. . . . [It is] a deviant behavior and the function of this deviance is boundary maintenance." The response of most AA members to another's slipping is sympathy and understanding, sentiments that in turn enhance group solidarity. In essence, "their abstinence is dependent on interaction with those who slip" (Rudy, 1980, p. 731).

WHO AFFILIATES?

What are the characteristics of those who do undergo the affiliation processes, contrasted with those who do not, even though exposed to the possibility? Demographic variables such as age, social class, race, employment status, and parental socio-economic status have been consistently found to be *unrelated* to membership (Trice & Roman, 1970; Emerick, 1989). These findings provide considerable certainty about the existence of often-alleged demographic barriers to AA affiliation—they, in effect, fail to deter affiliation.

Less certainty can be attributed to significant psychological characteristics that have been found to encourage affiliation. As in evaluations of psychotherapies in general (Eysenck, 1952; Rachman & Wilson, 1980), researchers cannot predict with any certainty who will affiliate. Despite this, certain personality features have been systematically found to distinguish between affiliates and nonaffiliates (Ogborne & Glaser, 1981; Ogborne, 1989).

> Several studies have suggested that, among other things, A.A. members can be distinguished from other heavy drinkers with respect to personality and perceptual characteristics. . . . The authors suggest that A.A. affiliation is associated with authoritarianism and conformist tendencies, high affiliation needs, proneness to guilt, religiosity, external control and field dependency [Ogborne, 1989, p. 59].

Ogborne (1989) also reports on two additional studies that support the belief that AA attracts individuals with certain emotional makeups. Ogborne's overall findings were that alcohol-troubled persons who expressed group adherence, extroversion, submissiveness, and conservatism were attracted to AA and its program. Overall, these findings appear to be consistent with the role demands made on members. For example, the sociability and affiliativeness themes found among those who do affiliate, as compared to nonaffiliates, seem to match the heavy group interactions expected of members.

All things considered, affiliation with AA is a distinctively selective process that fits only a distinct minority of those in the alcohol-abusing population. Although the exact proportion of the population helped by AA is unknown, even AA's critics recognize that it is substantial. Other specific types of therapies may do proportionally somewhat better or worse, but a reasonable estimate would be that AA is associated with fairly typical improvement rates.

Current studies strongly suggest that AA appeals to a highly specific and select segment and, by doing so, further suggest that other therapies are also selective as to their appeal. These points underscore the need for service providers to be aware of the diverse makeup of the problem-drinking population. Assessment and services need to be far more individualized than they have been, so that assignments may be made to the most appropriate organizations, institutions, or therapies.

THERAPEUTIC MECHANISMS

As with psychotherapies in general, the effectiveness of AA has not been convincingly established. For example, some problem drinkers drop out after the first two or three AA meetings. Nevertheless, for those who remain, AA has unique and distinctive features that contribute to its therapeutic effectiveness.

By definition, as problem drinkers move into addiction, alcohol comes to be central to their lives. How can this centrality be reduced and a new conception of alcohol be put in its place? AA experiences provide a new orientation, not only to alcohol, but to self and to others. "One of A.A.'s great strengths lies in the quality of its social environment: the empathetic understanding, the acceptance and concern which alcoholics experience there which, along with other qualities, make it easier to internalize new ways of feeling, thinking, and doing" (Maxwell, 1982). Even brief exposure to AA introduces the alcoholic to the idea that self-regulation seems to be rarely achieved alone by

self-reliance and willpower. Its basic premise describes the compelling sense of ego powerlessness—but immediately offers the potent substitute of a viable community that provides individual attention, an explanation of alcoholism, and simple prescriptions for sobriety. "In a community that shares the same distresses and losses, accepts its members' vulnerabilities and applauds and rewards successes, A.A. provides a stabilizing, sustaining, and ultimately, transforming group experience" (Khantzian & Mack, 1989, 76).

Within the AA community, there are group-based specific therapeutic strategies unlikely to exist in professionally directed psychotherapy. Examples include (1) empirically based hope; (2) direct attack on denials; (3) practical guidelines for achieving sobriety; and (4) one-to-one sponsorship. When problem drinkers first attend meetings they are immediately aware of others who have confronted their very problem, and they hear these people speak about dramatic improvements in their lives via the AA program. Moreover, the AA program consistently reminds them that denial of the realities surrounding their drinking is a major barrier to any change. Telling one's story, either publicly or in closed sessions, helps to dissolve the entrenched denial systems that ward off therapeutic changes.

The Twelve Steps are structured phases that provide an organized approach to the confusions and frustrations of an individual's attempt to cope with alcohol problems. This is especially so when members reach the developmental stage where twelfth-step work is indicated. They dramatically see themselves as they once had been, and this reinforces their need to "work the program." In addition, simple, practical guidelines are repeated as group-tested ways to avoid using alcohol as an adjustment technique: "First things first," "one day at a time," "easy does it," and practical advice about how to work the program.

AA typically arranges for the informal sponsorship of newcomers—who often identify with and closely relate to their sponsors. Sponsors are recovering alcoholics typically available at all hours of the day and night for phone or in-person discussions and crises. These valuable treatment strategies are voluntary and free of any monetary cost or financial obligation. Drinkers who drop out or who reject active membership in AA may nevertheless be substantially helped by primary or secondary exposure to AA and its unique but widely publicized or modified therapeutic mechanisms. (Several organizations in the alcohol or drug recovery field have been working along similar but modified lines.) It is impossible to estimate the numbers of those helped by such exposure, but they are surely numerous and should not be discounted.

CRITICAL EVALUATIONS

Probably the most widespread and long-standing critical assessment of AA centers around the question of the selective nature of membership. Thus, critic Stanton Peele (1989, 57) bluntly insists: "In fact, research has not found A.A. to be an effective treatment for general populations of alcoholics." Again, however, neither has any given professional psychotherapeutic method been found to be effective for general populations of the psychoneurotic. In 1981, Ogborne and Glaser predicted that evidence will soon be found for the effectiveness of AA, but "it will be limited to a particular, identifiable subgroup of persons with alcohol problems." This concern had been expressed since the 1950s (Trice & Staudenmeier 1989), and more evidence of AA's selective nature came from Walsh et al. (1991).

Walsh and her colleagues randomized 227 employed problem drinkers into compulsory inpatient treatment, compulsory attendance at AA, and a choice of options. During a two-year follow up, the researchers used measures of performance with drinking and drug use to gauge effectiveness. They concluded that the hospital group fared best and that the group assigned to AA fared least well. Many of those randomly assigned to AA probably lacked the readiness and the emotional makeup that appear to be required for affiliation. Matching patients to specific treatments has been advocated for many years (Ogborne & Glaser 1981; Pattison 1982), and the Walsh study certainly indicates the importance of matching in the case of AA.

Emerick (1989) has broadened the array of criticisms of AA to include (1) that AA has denounced in the media scientific discoveries that contradict its "formal ideology" and dogma: (2) AA has brought pressures to bear in an effort to suppress various psychological findings. Emerick quickly acknowledges, however, that "it is these very characteristics . . . that provide for AA's strength and effectively preserve its boundaries and identity" (p. 5). This

criticism boils down to charges that AA is anti-intellectual and antiprofessional. Second, harmful effects may come to those who "do not fit comfortably in the organization" (p. 9). These harmful effects include beliefs that "slipping" inevitably leads to loss of control, making for more problems than otherwise. Some members may despair and lose hope when they discover they do not mesh with AA's norms and beliefs. Third, there is a risk of becoming "AA addicts" who spend so much time and energy on the AA program that they neglect other areas of life such as family and job. Fourth, AA groups may contain alcohol-troubled persons who also suffer from other psychiatric disorders—i.e., schizophrenia and anxiety disorders—that should be directly treated, but are covered up by AA ideologies. Finally, AA members may develop dual overlapping relationships inside AA that are ultimately harmful, i.e., a newcomer becomes the lover of an established member, or a sponsor enters into an unfortunate business partnership with a sponsoree.

Other negative judgments of AA that have been voiced at one time or another are that it is tilted toward being a religion by too much emphasis on a "higher power"; local groups are not nearly as accepting of drunks as advertised; it suffers from too much adulation and consequently often becomes a "dumping ground" at which companies and courts require compulsory attendance; and it insists that members come to accept the label of "alcoholic"—a label that continues to be highly stigmatic outside AA and tends to repel many of those who are inclined to affiliate.

Understandably, these criticisms have fueled alternative groups that claim to help members cope with alcohol problems but without many of the beliefs and rituals of AA. Apparently, many of the members of these groups are AA dropouts. Beyond this observation, no systematic research efforts have been mounted to determine how affiliation is achieved, and with what kinds of problem drinkers these alternatives to AA are effective. For example, RATIONAL RECOVERY (RR), SECULAR ORGANIZATION FOR SOBRIETY (SOS), and WOMEN FOR SOBRIETY (WFS) have all claimed to be alternative self-help groups for alcoholics. They tend to reject the notion that alcoholism is a disease and advocate instead personal responsibility. Also, they underscore individual willpower rather than AA's belief in a higher power (Gelman, Leonard, & Fisher, 1991). Regarding the charge that AA is overly religious, numerous close observers, including the present writer, have concluded that religion plays a minor role in the practical day-to-day effort of AAs to "work the program."

ADAPTATION OF AA TO OTHER DISORDERS

Despite the criticisms that have been directed against AA, its format and beliefs have nevertheless been applied to a wide variety of other addictions and behavior disorders. For example, NARCOTICS ANONYMOUS (NA) (estimated in 1979 to have about 700 groups in practically every U.S. state and in several other countries) first applied the AA pattern to drug addicts at the U.S. PUBLIC HEALTH SERVICE HOSPITAL at Lexington, Kentucky, in 1947. In 1948, and in 1953, groups of AA members who were also drug addicts formed an independent NA group in New York City and in Sun Valley, California. The resemblance to AA was made even more remarkable by the fact that the magazine that had made AA well-known (*The Saturday Evening Post*) also gave NA a national audience through a lengthy piece that played up similarities with AA (Ellison, 1954).

Similarly, AA's beliefs and strategies have been adapted to help people with a broad spectrum of other problems, including excessive buying, sexual excesses or deviations, gambling, child abuse, overdependence on others, eating disorders, and excessive shame and guilt. In addition, AL-ANON family groups and ALATEEN groups have adapted AA's philosophy to family, children, and friends of problem drinkers. Many others could be cited. Veteran AA members point to this great proliferation as evidence that AA's influence goes well beyond its impact on AA members. They argue that this widespread adaptation to other disorders demonstrates the essential value and appeal of the AA program.

(SEE ALSO: *Alcoholism; Gambling as an Addiction; Treatment*)

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HARRISON M. TRICE

ALCOHOLISM This section contains articles on some aspects of chronic drinking: *Abstinence versus Controlled Drinking* and *Origin of the Term*. For further information on this subject, see *Disease Concept of Alcoholism and Drug Abuse* and the sections on *Complications*, on *Treatment* and on *Withdrawal*.

Abstinence versus Controlled Drinking

The position of ALCOHOLICS ANONYMOUS (AA) and the dominant view among therapists who treat alcoholism in the United States is that the goal of treatment for those who have been *dependent* on alcohol is total, complete, and permanent abstinence from alcohol.

Abstinence was at the base of Prohibition (legalized in 1919 with the Eighteenth Amendment) and is closely related to prohibitionism—the legal proscription of substances and their use.

Although temperance originally meant moderation, the nineteenth-century TEMPERANCE MOVEMENT's emphasis on complete abstinence from alcohol and the mid-twentieth century's experience of the ALCOHOLICS ANONYMOUS movement have strongly influenced alcohol- and drug-abuse treatment goals in the United States. Moral and clinical issues, however, have been irrevocably mixed.

The disease model of alcoholism and drug addiction, which insists on abstinence, has incorporated new areas of compulsive behavior—such as

overeating and sexual involvements. In these cases, redefinition of *abstinence* from total avoidance to "the avoidance of excess" (what we would otherwise term moderation) is required.

Abstinence can also be used as a treatment-outcome measure, as an indicator of its effectiveness. In this case, abstinence is defined as the number of drug-free days or weeks during the treatment regimen—and measures of drug in urine are often used as objective indicators.

CONTROLLED DRINKING

By extension, for all those treated for alcohol *abuse*, including those with no dependence symptoms, moderation of drinking (termed *controlled drinking*, or CD) as a goal of treatment is rejected (Peele, 1992). Instead, providers claim, holding out such a goal to an alcoholic is detrimental, fostering a continuation of denial and delaying the alcoholic's need to accept the reality that he or she can never drink in moderation. One painful example of this is the case of Audrey Kishline, author of *Moderate Drinking: The New Option for Problem Drinkers*, and founder of the group Moderation Management. In the summer of 2000, Kishline pleaded guilty to a vehicular homicide incident in which she killed a father and his twelve-year-old daughter when she drove the wrong way on a Washington State highway. Her blood alcohol level at the time of the accident was 0.26—three times the legal limit.

In Britain and other European and Commonwealth countries, controlled-drinking therapy is widely available (Rosenberg et al., 1992). The following six questions explore the value, prevalence, and clinical impact of controlled drinking versus abstinence outcomes in alcoholism treatment; they are intended to argue the case for controlled drinking as a reasonable and realistic goal.

1. *What proportion of treated alcoholics abstain completely following treatment?*

At one extreme, Vaillant (1983) found a 95 percent relapse rate among a group of alcoholics followed for eight years after treatment at a public hospital; and over a four-year follow-up period, the Rand Corporation found that only 7 percent of a treated alcoholic population abstained completely (Polich, Armor, & Braiker, 1981). At the other extreme, Wallace et al. (1988) reported a 57 percent continuous abstinence rate for private clinic patients who were stably married and had successfully completed detoxification and treatment—but results in this study covered only a six-month period.

In other studies of private treatment, Walsh et al. (1991) found that only 23 percent of alcohol-abusing workers reported abstaining throughout a two-year follow-up, although the figure was 37 percent for those assigned to a hospital program. According to Finney and Moos (1991), 37 percent of patients reported they were abstinent at all follow-up years 4 through 10 after treatment. Clearly, most research agrees that most alcoholism patients drink at some point following treatment.

2. *What proportion of alcoholics eventually achieve abstinence following alcoholism treatment?*

Many patients ultimately achieve abstinence only over time. Finney and Moos (1991) found that 49 percent of patients reported they were abstinent at four years and 54 percent at ten years after treatment. Vaillant (1983) found that 39 percent of his surviving patients were abstaining at eight years. In the Rand study, 28 percent of assessed patients were abstaining after four years. Helzer et al. (1985), however, reported that only 15 percent of all surviving alcoholics seen in hospitals were abstinent at 5 to 7 years. (Only a portion of these patients were specifically treated in an alcoholism unit. Abstinence rates were not reported separately for this group, but only 7 percent survived and were in remission at follow-up.)

3. *What is the relationship of abstinence to controlled-drinking outcomes over time?*

Edwards et al. (1983) reported that controlled drinking is more unstable than abstinence for alcoholics over time, but recent studies have found that controlled drinking increases over longer follow-up periods. Finney and Moos (1991) reported a 17 percent "social or moderate drinking" rate at six years and a 24 percent rate at ten years. In studies by McCabe (1986) and Nordström and Berglund (1987), CD outcomes exceeded abstinence during follow-up of patients fifteen and more years after treatment (see Table 1). Hyman (1976) earlier found a similar emergence of controlled drinking over fifteen years.

TABLE 1
Selected Alcoholism Outcome Studies

Study	*Years of Follow-up*	*No. of Assessed Subjects*	*Percent Abstinent*	*Percent Controlled Drinking*	*Percent Remission Survivors*[a]
Untreated					
Goodwin, Crane, & Guze (1971)	8	93	8	33[b]	41
1989 Canadian National Survey	≥1	497	49	51	100[c]
Treated					
Rand (Polich, Armor, & Braiker 1981)	4	548	28	18[d]	46
Vaillant (1983)	8	100	39	6	45
Helzer et al. (1985)[e]	5–7	387	15[f]	18[g]	33
McCabe (1986)	16	31	26	35	61
Nordström & Berglund (1987)	18–24	55	20	38	58
Rychtarik et al. (1987)	5–6	43	23	21	44
Wallace et al. (1988)	0.5	169	68[h]	—[h]	68[h]
Finney and Moos (1991)	10	83	54	24	78
Walsh et al. (1991)	2	200	23 (37)[i]	10 (7)[i]	33 (44)[i]

[a]Since only survivors are included, successful remission rates are overstated.
[b]Nonabstinence remission included 18 percent "moderate drinkers," 9 percent getting "drunk about once a week," 6 percent "switched from whiskey to beer, . . . drank almost daily and sometimes excessively, [but] had experienced no problems from drinking since making the change."
[c]The Canadian National Survey data concern only recovered alcohol abusers.
[d]Defined as nonproblem drinking, with either low quantities of consumption (8%) or some heavy drinking (10%).
[e]Although all subjects in this study were hospital patients, only one group was treated in an alcohol unit. This group had the worst outcomes of any group, but these outcomes were not reported separately.
[f]Reported data were weighted by Helzer et al.
[g]Controlled drinking outcomes include occasional drinkers (4.6%), moderate drinkers (1.6%), and heavy, nonproblem drinkers (12%).
[h]Wallace et al. reported 57 percent continuous abstinence over 6 months and an additional 11 percent currently abstinent. Although Wallace et al. reported no controlled drinking, a small group (4%) had "one brief, contained return to drinking or drug use" in the 180-day period.
[i]Figures are for all treated groups, with assigned hospital patients in parentheses. No controlled-drinking category was included, but this column comprises those in the study who drank without ever becoming intoxicated during the 2-year follow-up (the latter data are not fully reported in the published article).

4. *What are legitimate nonabstinent outcomes for alcoholism?*

The range of nonabstinence outcomes between unabated alcoholism and total abstinence includes (1) "improved drinking" despite continuing alcohol abuse, (2) "largely controlled drinking" with occasional relapses, and (3) "completely controlled drinking." Yet some studies count both groups (1) and (2) as continuing alcoholics and those in group (3) who engage in only occasional drinking as abstinent. Vaillant (1983) labeled abstinence as drinking less than once a month and including a binge lasting less than a week each year.

The importance of definitional criteria is evident in a highly publicized study (Helzer el al., 1985) that identified only 1.6 percent of treated alcoholism patients as "moderate drinkers." Not included in this category were an additional 4.6 percent of patients who drank without problems but who drank in fewer than thirty of the previous thirty-six months. In addition, Helzer

et al. identified a sizable group (12%) of former alcoholics who drank a threshold of seven drinks four times in a single month over the previous three years but who reported no adverse consequences or symptoms of alcohol dependence and for whom no such problems were uncovered from collateral records. Nonetheless, Helzer et al. rejected the value of CD outcomes in alcoholism treatment.

While the Helzer et al. study was welcomed by the American treatment industry, the Rand results (Polich, Armor, & Braiker, 1981) were publicly denounced by alcoholism treatment advocates. Yet the studies differed primarily in that Rand reported a higher abstinence rate, using a six-month window at assessment (compared with three years for Helzer et al.). The studies found remarkably similar nonabstinence outcomes, but Polich, Armor, and Braiker (1981) classified both occasional and continuous moderate drinkers (8%) and sometimes heavy drinkers (10%) who had no negative drinking consequences or dependence symptoms in a nonabstinent remission category. (Rand subjects had been highly alcoholic and at intake were consuming a median of seventeen drinks daily.)

The harm-reduction approach seeks to minimize the damage from continued drinking and recognizes a wide range of improved categories (Heather, 1992). Minimizing nonabstinent remission or improvement categories by labeling reduced but occasionally excessive drinking as "alcoholism" fails to address the morbidity associated with continued untrammeled drinking.

5. *How do untreated and treated alcoholics compare in their controlled-drinking and abstinent-remission ratios?*

Alcoholic remission many years after treatment may depend less on treatment than on post-treatment experiences, and in some long-term studies, CD outcomes become more prominent the longer subjects are out of the treatment milieu, because patients unlearn the abstinence prescription that prevails there (Peele, 1987). By the same token, controlled drinking may be the more common outcome for untreated remission, since many alcohol abusers may reject treatment because they are unwilling to abstain.

Goodwin, Crane, & Guze (1971) found that controlled-drinking remission was four times as frequent as abstinence after eight years for untreated alcoholic felons who had "unequivocal histories of alcoholism" (see Table 1). Results from the 1989 Canadian National Alcohol and Drug Survey confirmed that those who resolve a drinking problem without treatment are more likely to become controlled drinkers. Only 18 percent of five hundred recovered alcohol abusers in the survey achieved remission through treatment. About half (49 percent) of those in remission still drank. Of those in remission through treatment, 92 percent were abstinent. But 61 percent of those who achieved remission without treatment continued drinking (see Table 2).

TABLE 2
Controlled Drinking and Abstinence in Relation to Treated and Untreated Remission: The 1989 Canadian National Alcohol and Drug Survey (Data Weighted to Represent National Population)

	Treated Remission (n = 89) %	*Untreated Remission (n = 408)* %
Abstinent (51%)	92	39
Nonabstinent (49%)	8	61

Data presented by L. C. Sobell & M. B. Sobell (November 1991). Cognitive mediators of natural recoveries from alcohol problems: Implications for treatment. Paper presented as part of a symposium, "Therapies for Substance Abuse: A View towards the Future," 25th Annual Meeting of the Association for the Advancement of Behavior Therapy, New York.

6. *For which alcohol abusers is controlled-drinking therapy or abstinence therapy superior?*

Severity of alcoholism is the most generally accepted clinical indicator of the appropriateness of CD therapy (Rosenberg, 1993). Untreated alcohol abusers probably have less severe drinking problems than clinical populations of alcoholics, which may explain their higher levels of controlled drinking. But the less severe problem drinkers uncovered in nonclinical studies are more typical, outnumbering those who "show major symptoms of alcohol dependence" by about four to one (Skinner, 1990).

TABLE 3
Relapse Rates at 4-Year Follow-up According to Remission Category at Eighteen Months, by Alcohol Dependence Category, Marital Status, and Age

	Age < 40 at Admission		*Age > 40 at Admission*	
	Abstaining 18 Months	*Nonproblem Drinking 18 Months*	*Abstaining 18 Months*	*Nonproblem Drinking 18 Months*
High Dependence Symptoms				
Married	12	17	14	50
Single	21	7	24	28
Low Dependence Symptoms				
Married	16	7	19	28
Single	29	3	32	13

J. M. Polich, D. J. Armor, & H. B. Braiker (1981). *The course of alcoholism: Four years after treatment.* New York: Wiley.

Despite the reported relationship between severity and CD outcomes, many diagnosed alcoholics do control their drinking, as Table 1 reveals. The Rand study quantified the relationship between severity of alcohol dependence and controlled-drinking outcomes, although, overall, the Rand population was a severely alcoholic one in which "virtually all subjects reported symptoms of alcohol dependence" (Polich, Armor, and Braiker, 1981).

Polich, Armor, and Braiker found that the most severely dependent alcoholics (eleven or more dependence symptoms on admission) were the least likely to achieve nonproblem drinking at four years. However, a quarter of this group who achieved remission did so through nonproblem drinking. Furthermore, younger (under 40), single alcoholics were far more likely to relapse if they were abstinent at eighteen months than if they were drinking without problems, even if they were highly alcohol-dependent (Table 3). Thus the Rand study found a strong link between severity and outcome, but a far from ironclad one.

Some studies have failed to confirm the link between controlled-drinking versus abstinence outcomes and alcoholic severity. In a clinical trial that included CD and abstinence training for a highly dependent alcoholic population, Rychtarik et al. (1987) reported 18 percent controlled drinkers and 20 percent abstinent (from fifty-nine initial patients) at 5 to 6 year follow-up. Outcome type was not related to severity of dependence. Nor was it for Nordström and Berglund (1987), perhaps because they excluded "subjects who were never alcohol dependent."

Nordström and Berglund, like Wallace et al. (1988), selected high-prognosis patients who were socially stable. The Wallace et al. patients had a high level of abstinence; patients in Nordström and Berglund had a high level of controlled drinking. Social stability at intake was negatively related in Rychtarik et al. to consumption as a result either of abstinence or of limited intake. Apparently, social stability predicts that alcoholics will succeed better whether they choose abstinence or reduced drinking. But other research indicates that the pool of those who achieve remission can be expanded by having broader treatment goals.

Rychtarik et al. found that treatment aimed at abstinence or controlled drinking was not related to patients' ultimate remission type. Booth, Dale, and Ansari (1984), on the other hand, found that patients did achieve their *selected* goal of abstinence or controlled drinking more often. Three British groups (Elal-Lawrence, Slade, & Dewey, 1986; Heather, Rollnick, & Winton, 1983; Orford & Keddie, 1986)

have found that treated alcoholics' beliefs about whether they could control their drinking and their commitment to a CD or an abstinence-treatment goal were more important in determining CD versus abstinence outcomes than were subjects' levels of alcohol dependence. Miller et al. (in press) found that more dependent drinkers were less likely to achieve CD outcomes but that desired treatment goal and whether one labeled oneself an alcoholic or not independently predicted outcome type.

SUMMARY

As of 2000, there is no conclusive evidence to show that one single method of treatment is consistently more successful than another (Project MATCH). One of the largest (U.S.) clinical experiments—sponsored by the National Institute on Alcohol Abuse and Alcoholism—shows that of the three major treatments studied (cognitive-behavioral therapy [CBT], twelve-step facilitation [TSF], and motivational enhancement therapy [MET]), none emerged the clear "winner."

A group of 952 outpatients (some still drinking) and 774 patients (previously receiving residential/day hospital treatment) participated in the study, that spanned over twelve weeks. The two groups were each divided into three separate groups, each group receiving one of the three treatments.

Particpants were polled immediately after treatment and again every three months for a year in an effort to document their subsequent progress. Patients of all three treatments exemplified good and sustained results. In all, only 10 percent of the participants dropped out of the study itself; two-thirds finished the treatment(s). The percentage of days abstinent (PDA) rose in all three groups by 60 percent (from 20 to 80 percent), and suffered only minimal decline in the next year.

Controlled drinking (CD) is one of a number of approaches with an important role to play in alcoholism treatment. CD, as well as abstinence, is an appropriate goal for the majority of problem drinkers who are not alcohol-dependent (though there is no proven scientific method to determine who can and who cannot stop drinking after one or two drinks). In addition, while controlled drinking becomes less likely the more severe the degree of alcoholism, other factors—such as age, values, and beliefs about oneself, one's drinking, and the possibility of controlled drinking—also play a role, sometimes the dominant role, in determining successful outcome type. Finally, reduced drinking is often the focus of a harm-reduction approach, where the likely alternative is not abstinence but continued alcoholism.

(SEE ALSO: *Alcohol; Disease Concept of Alcoholism and Drug Abuse; Relapse Prevention; Treatment*)

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Stanton Peele
Revised by Kimberly A. McGrath

Origin of the Term The term *alcoholism* is of relatively recent date; knowledge of the adverse effects of heavy alcohol (ethanol) consumption is not. A proverb describes alcohol as "both mankind's oldest friend and oldest enemy." Alcohol occurs in nature, and humans have long known how to ferment plants to create it; both its moderate and excessive use have therefore occurred since prehistory. The Bible cautions: "Do not look at wine when it is red, when it sparkles in the cup and goes down smoothly. At the end it bites like a serpent and stings like an adder" (Proverbs 23:31–32). A drunken Noah (Genesis 9:20–28) is one of a long line of such literary descriptions. In the classical era of the Greeks and the Romans we have drunks in the *Character Sketches* of Theophrastus, in the *Satyricon* of Petronius Arbiter, and in the *Epistles* of Seneca. In the 1600s, we have Shakespeare's porter in *Macbeth* (Act II, Scene 3) and others.

Viewing the long-term adverse effects of alcohol as a disease is a concept that also predates the term *alcoholism*. Benjamin Rush (1745–1813) and Thomas Trotter (1760–1832), both physicians, wrote extensively in this vein, using words such as *drunkenness*; their elder contemporary Benjamin Franklin (1706–1790) produced a glossary of 228 synonyms in use in 1737 for "being under the influence of alcohol." It was not until 1849 that the Swedish physician and temperance advocate Magnus Huss (1807–1890) first used the word *alcoholism* in his book *Alcoholismus Chronicus (The Chronic Alcohol-disease)*. Huss's term, used originally in a descriptive sense to denote the consequences *of* the prolonged consumption of large quantities of alcohol, has come to connote a

disease, believed by some to result *in* such consumption.

Huss meant by the term *chronic alcoholism* "those pathologic symptoms which develop in such persons who over a long period of time continually use wine or other alcoholic beverages in large quantities" and stated that it "corresponds with chronic poisoning." His book is filled with detailed case histories illustrating the various symptoms that might occur. Sweden was at that time highest in the list of countries that consumed liquors, and Huss, as attending physician to the Serafim Clinic In Stockholm, had ample opportunity to observe cases. The London *Daily News* of December 8, 1869, carried a story on "the deaths of two persons from alcoholism," which according to the *Oxford English Dictionary* was the first popular use of the word in English. From that time on, its use in both the professional and the popular literature greatly expanded. This is partly because of the natural process that popularizes usage of certain words and partly because of deliberate activities on behalf of the term *alcoholism*.

The period of national prohibition in the United States (1919–1933) was accompanied by a lack of attention to the consequences of alcohol consumption, for understandable reasons. Such consumption was illegal—permanently, it was assumed—and as a result, it was thought that there would be little in the way of consequences. Indeed, such consequences as cirrhosis of the liver did decline abruptly during this period. But as enthusiasm for prohibition waned, and especially after it was repealed, a need to promote treatment became increasingly evident. One group involved in this promotion used *alcoholism* as the key word in their efforts, and accordingly were called *the alcoholism movement* by sociologists who subsequently studied their work. In an early statement of this movement, Anderson (1942) predicted that "When the dissemination of these ideas is begun through the existing media of public information, press, radio, and platform, which will consider them as news, a new public attitude can be shaped." It was also felt that the term, together with the disease connotations attached to it, would encourage the involvement of physicians in its study and treatment. The medical profession was viewed as critical to the success of the effort to increase the nation's concern about the consequences of alcohol consumption. The formation of the NATIONAL COUNCIL ON ALCOHOLISM, the largest public interest group in this area, was a project of the same movement. Their successful efforts may be the reason that the term *alcoholism* developed and sustained a popularity in the United States beyond anything it achieved in Europe and even in Scandinavia, where it was first used.

MEANING BROADENS

As the term *alcoholism* became widely used, its meaning broadened. In a 1941 review of treatment, ten definitions of chronic alcoholism and sixteen definitions of alcohol addiction were collected from the international literature. Originally used by Huss to refer to a disease that consisted of the *consequences* of alcohol consumption, *alcoholism* came in time to represent a disease that *caused* high levels of alcohol consumption (Jellinek, 1960). A variant theory attempts to preserve the original meaning: High levels of alcohol consumption resulted in consequences of various kinds, particularly in terms of damage to the central nervous system, which damage in turn caused the high levels of consumption to continue (Vaillant, 1983). That is, the term *alcoholism* evolved over time from a primarily descriptive term to a largely explanatory concept. An example of a definition of *alcoholism* with clear explanatory intent is one that R. C. Rinaldi and colleagues produced in 1988 through an elaborate consensus exercise (a Delphi process) among eighty American experts, who defined the term as "a chronic, progressive, and potentially fatal biogenetic and psychosocial disease characterized by tolerance and physical dependence manifested by a loss of control, as well as diverse personality changes and social consequences." As a counterpoint to this line of development, a growing and increasingly influential literature holds that problems developing in the context of alcohol consumption do not constitute a disease at all (Fingarette, 1988).

The greater interest taken in alcohol consumption and its consequences as a result of the popularization of the term *alcoholism* has been gratifying as well as useful. But the broadening of meaning of the term, with much attendant controversy among the advocates of various definitions, has become problematic. For example, in a review of alternative definitions, Babor & Kadden (1985) concluded: "Clearly, the past and present lack of

consensus concerning the definition of alcoholism and the criteria for its diagnosis does not provide a solid conceptual basis to design screening procedures for early detection or casefinding.'' Because of its imprecise meaning, the term *alcoholism* has for some time now been dropped from the two major official systems of diagnosis of diseases, the INTERNATIONAL CLASSIFICATION OF DISEASES of the World Health Organization and the DIAGNOSTIC AND STATISTICAL MANUAL *of Mental Disorders* of the American Psychiatric Association. A recent comprehensive study of treatment deliberately avoided the use of the word *alcoholism* as too narrow in its focus, while suggesting that the word was not incompatible with the phrase that it chose to use—*alcohol problems*—to refer to any problem occurring in the context of alcohol consumption (Institute of Medicine, 1990, pp. 30–31).

COMPLEX PROBLEMS

These recent attempts to be precise in the use of words represent a return to the more straightforward, descriptive use of *alcoholism* by its originator, Huss. Two major realities contributing to this change of direction have been widely recognized since Huss first used the term in the 1840s. One is that the problems people experience are complex, including those that may arise in the context of alcohol use. Although alcohol may be a factor in some such problems—even an important factor—it is not often the full explanation for them. Multiple factors, including heredity, early environment, cultural factors, personality factors, situational factors, and others, contribute to the development of human problems and must be considered in their resolution. This formulation should not be taken to minimize the important role of alcohol in such problems or to say that the reduction or elimination of alcohol consumption may not be a critical factor in the resolution of problems in particular individuals. The other reality has to do with the extremely broad spectrum of problems that arise in the context of alcohol consumption. Although a substantial proportion of these problems arise from those who drink too much over a long period of time and who usually have multiple problems (those to whom the term *alcoholism* is usually applied), an even greater burden of problems arises from those who drink too much over *short* periods of time, and who have only a *few* problems. The simple reason is that there are more of the latter than of the former (Institute of Medicine, 1990, chapter 9). To reduce the burden upon society effectively, both kinds of populations must be dealt with. An exclusive concentration on *alcoholism* may cause this reality to be overlooked.

Costly Consequences. The term *alcoholism* retains, and probably will always retain, its place in general, nontechnical speech as an indicator of serious problems that are the consequences of prolonged heavy alcohol consumption. Its continued popularity has some advantages, for the public-health consequences of such alcohol consumption are horrendous. The presence of a convenient shorthand term for this fact in the public consciousness—*alcoholism*—serves as a continuing reminder of this major unfinished item on the public-health agenda. Certainly, there is a legitimate place in Western society for the use of alcohol. But with equal certainty, too many individuals fail to use alcohol wisely or well.

The ravages that prolonged exposure to alcohol produces in the human body are manifold, as Huss well understood; they include neurological problems (damage to the central and peripheral nervous systems), cirrhosis (fibrosis and shrinking) of the liver, hypertension (high blood pressure), and many forms of cancer, particularly of the digestive tract, to name but a few. If to these are added the consequences of short-term but intense exposure to alcohol and the intoxication it produces, one can include a high proportion of all accidents, burns, all types of violence including suicide, and especially automobile crashes, as well as the common behavioral effects of intoxication with which we are all too familiar. Small wonder that almost 30 percent of all admissions to hospitals in the United States are of persons with severe alcohol problems; yet most of these problems go unrecognized, and the individuals go untreated. About 50 percent of American women have or have had a parent, blood relative, or spouse to whom they would apply the term *alcoholism*; the figure is closer to 40 percent for men. The difficulties that this creates are legion—and its remediation would be a remarkable step forward.

(SEE ALSO: *Addiction: Concepts and Definitions; Disease Concept of Alcoholism; Treatment, History of*)

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FREDERICK B. GLASER

ALKALOIDS This is the general term for any number of complex organic bases that are found in nature in seed-bearing plants. These substances are usually colorless but bitter to the taste. Alkaloids often contain nitrogen and oxygen and possess important physiological properties.

Examples of alkaloids include not only quinine, atropine, and strychnine but also CAFFEINE, NICOTINE, MORPHINE, CODEINE, and COCAINE. Therefore, many drugs that are used by humans for both medical and nonmedical purposes are produced in nature in the form of alkaloids. Naturally occurring receptors for many alkaloids have also been identified in humans and other animals, suggesting an evolutionary role for the alkaloids in physiological processes.

NICK E. GOEDERS

ALLERGIES TO ALCOHOL AND DRUGS In addition to ALCOHOL, OPIATES, and BARBITURATES, some street drugs have been reported to induce allergic reactions. These allergic phenomena are most frequently mediated by reactions of the immune system known as immediate hypersensitivity and delayed hypersensitivity. *Immediate hypersensitivity* is mediated by the serum protein immunoglobulin E(IgE), whereas *delayed hypersensitivity* is mediated by thymus-derived lymphocytes (the white blood cells called T cells).

Immediate Hypersensitivity. The symptoms and signs associated with IgE-mediated immune reactions are urticaria (hives); bronchospasm that produces wheezing; angioedema (swelling) of face and lips or full-blown *anaphylaxis* (a combination of all the above symptoms and lowering of blood pressure). Abdominal pain and cardiac arrhythmias (irregular heartbeat) may also occur with anaphylaxis. Any or all of these symptoms occur when IgE, which has previously been synthesized by a sensitized lymphocyte, fixes to mast cells or ba-

sophils in the skin, bronchial mucosa, and intestinal mucosa. This cell-fixed IgE then binds the antigen that triggers the release of the following—the histamine, the slow-reacting substance of anaphylaxis (SRSA), the bradykinin, and the other mediators that induce these symptoms. Examples of this type of allergic reaction are the allergic responses to either bee stings or to penicillin.

Similar symptoms may also occur when mediators are released by mast cells in response to chemical or physical stimuli. This is called an *anaphylactoid reaction*. In this instance, the mast cell or basophil is directly activated by the chemical to release mediators without having to bind to IgE. Examples of this type of reaction are responses to intravenous contrast material, such as IVP dye, or the hives induced by exposure to cold.

Delayed Hypersensitivity. Reactions occur when antigenic chemicals stimulate T lymphocytes and induce their proliferation. T effector cells are then recruited into the tissue site. These effector cells bind the antigen and subsequently release effector molecules, such as the interleukins, the chemotactic factors, and the enzymes. These effector molecules induce an inflammatory response in the area and may also induce formation of granuloma (a mass of inflamed tissue) by macrophages and inflammatory cells. Symptoms of delayed hypersensitivity reactions are skin rashes, which may be red, pruritic (itchy), or bullous (blistered) in nature. Granulomas can cause lymph node enlargement and nodules in the skin or in organs. Examples of this response are poison ivy, cosmetic allergies, Erythema Nodosum or Sarcoidosis.

ALLERGIC RESPONSE TO ALCOHOL

True anaphylactic or anaphylactoid reactions to ALCOHOL (ethanol) are rare. Most reactions to ingested alcoholic beverages are secondary to other chemicals in the beverage such as yeasts, metabisulfite, papain, or dyes. However, there are reports of true allergic reactions in which the offending agent was shown to be the ethanol itself.

Symptoms of anaphylaxis have been reported to occur in several subjects following ingestion of beer and/or wine, and these symptoms were reproduced in one patient by administration of 95 percent ethanol. Hives have been reported with ethanol ingestion, and hives on contact with ethanol have been reported for some Asian patients. Bronchospasm was precipitated in some asthmatic patients by administration of ethanol, and contact hypersensitivity to 50 percent ethanol solution was produced in 6 percent of subjects tested. These allergic responses differ from the "flush" reaction exhibited in individuals (especially Asians) with acetaldehyde dehydrogenase abnormalities.

ALLERGIES TO OPIATES, BARBITURATES, AND STREET DRUGS

There have been reports of MORPHINE-induced hives in some people, and studies show that morphine can cause histamine release directly from cells without binding to specific receptors on cells. Anaphylaxis may also occur with either morphine or CODEINE, and IgE antibodies against morphine and codeine have been found in patients experiencing anaphylaxis. Thus, the OPIATES can mediate allergic reaction by either mechanism, and the antagonist drug NALOXONE will not reverse these reactions. There are also reports of HEROIN causing bronchospasm.

Some instances of anaphylaxis associated with the medical administration of opiates or local anesthesia during surgery are due to the often included preservative methylparaben, rather than to the opiate itself. Anaphylaxis may occur with more than one local anesthetic and/or analgesic compound in the same patient because of the methylparabens.

Numerous reports exist for anaphylactoid reactions following the use of BARBITURATES for the induction of anesthesia. The drugs themselves may induce histamine release. This may also be mediated through a true allergic IgE mediated response in some patients. Skin rashes also occur frequently following barbiturate usage. This may be a hypersensitivity reaction, or it may be a pseudo-allergic reaction.

Street drugs have been reported to induce asthma and or anaphylaxis. Bronchospasm may occur in patients smoking COCAINE or in those injecting heroin. This may occur more often in patients who have a previous history of asthma. The asthma may persist after the subjects have stopped smoking cocaine. Pulmonary edema (fluid in the lungs) may also occur with FREEBASING cocaine. These side effects are not likely to be mediated by the immune system. However, a hypersensitivity pneumonitis to cocaine has been described and is associated with elevated levels of IgE. MARIJUANA

does not appear to increase the incidence of either asthma or anaphylaxis.

(SEE ALSO: *Complications*)

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MARLENE ALDO-BENSON

AMANTADINE Amantadine is a medication (Symmetrel) that is believed to be an indirect DOPAMINE agonist; this means that it releases the neurotransmitter dopamine from nerve terminals in the brain. Since some of the symptoms of COCAINE withdrawal and cocaine dependence are thought to be related to abnormalities in the dopamine systems of the brain, and these are thought to contribute to relapse, amantadine has been examined as a treatment possibility.

After chronic cocaine use, many patients' dopamine systems either fail to release sufficient dopamine or are insensitive to the dopamine that is released. This relative dopamine deficit is believed to be responsible for the dysphoria of cocaine withdrawal. It was hoped that amantadine would relieve their dysphoria and reduce relapse back to cocaine abuse by increasing the release of dopamine in the brains of cocaine-dependent patients. Amantadine has been effective in reducing depressive symptoms in patients with neurological disorders such as Parkinson's disease, which is due to the death of dopamine-producing cells in the brain; however, no solid evidence exists that it is helpful in preventing continued cocaine use or relapse to cocaine use after detoxification.

(SEE ALSO: *Treatment: Cocaine; Withdrawal: Cocaine*)

THOMAS R. KOSTEN

AMERICAN ACADEMY OF ADDICTION PSYCHIATRY (AAAP) The American Academy of Addiction Psychiatry (7301 Mission Road, Suite 252, Prairie Village, KS 66208; 913-262-6161; http://www.aaap.org/) is a not-for-profit scientific and professional organization whose members specialize in the clinical treatment of addictive disorders and related research and education. The AAAP originated in 1985 as the American Academy of Psychiatrists in Alcoholism and Addictions (AAPAA). In 1996, the organization renamed itself the American Academy of Addiction Psychiatry.

The AAAP strives to provide psychiatrists, mental health professionals, and students a close-knit environment where members have opportunities to collaborate, network, and educate one another. The AAAP also seeks to increase public awareness of addiction psychiatry in the treatment and prevention of substance-use disorders by publishing policy statements, working with lawmakers, and cosponsoring programs such as

National Alcohol Screening Day. In an effort to further promote the field of addiction psychiatry, the AAAP will assist medical schools that are applying to the Accreditation Council on Graduate Medical Education for accreditation of their addiction psychiatry subspecialty programs. Other stated goals of the organization include promoting availability of the highest-quality treatment for all who need it; promoting excellence in clinical practice in addiction psychiatry; providing continuing education for addiction professionals; disseminating new information in the field of addiction psychiatry; and encouraging research on the etiology, prevention, identification, and treatment of addictions.

Membership in the AAAP exceeds one thousand and is organized into nine national and international geographic areas. Psychiatrists who work with alcoholism and addiction in their practices and are members of the American Psychiatric Association and/or the Canadian Psychiatric Association are eligible to become General Members in the AAAP. Faculty members and nonpsychiatrist professionals who have contributed to the field of addiction psychiatry may join as Affiliate Members. There are additional membership categories for medical students, residents, retired psychiatrists, and international members. Psychiatrists who have made significant contributions to the field through clinical work, teaching, research, or administration may, upon recommendation of the executive committee, be elected Fellows.

Physicians already certified as specialists in psychiatry by the American Board of Psychiatry and Neurology (ABPN) can earn further credentials in the subspecialty of addiction psychiatry by passing the ABPN Examination for Subspecialty Certification in Addiction Psychiatry. The AAAP offers addiction psychiatry review courses to assist in preparing for certification.

The official journal of the AAAP is the *American Journal on Addictions*, a peer-reviewed clinical publication that is published quarterly. The organization also publishes a quarterly newsletter, *AAAP News*. Subscriptions to both the journal and newsletter are included with membership.

The AAAP hosts an annual meeting and symposium, which includes workshops, presentations of papers, poster sessions, committee meetings, and area meetings designated by geographic region, as well as the annual business meeting and an awards ceremony.

(SEE ALSO: *American Society of Addiction Medicine*)

FAITH K. JAFFE
REVISED BY NANCY FAERBER

AMERICAN INDIANS AND DRUG USE
See Ethnicity and Drugs

AMERICAN SOCIETY OF ADDICTION MEDICINE (ASAM) The American Society of Addiction Medicine (4601 North Park Avenue, Arcade Suite 101, Chevy Chase, MD 20815; 301-656-3920; http://www.asam.org) is a not-for-profit organization of physicians in all medical specialties and subspecialties who devote a significant part of their practice to treating patients addicted to, or having problems with, alcohol, nicotine, and other drugs. The society strives to have addiction recognized as a medical disorder by health insurance and managed care providers, and the medical community at large. Many of its members are actively involved in medical education, research, and public policy issues concerning the treatment and prevention of addiction.

ASAM's roots can be traced to the early 1950s, when Dr. Ruth Fox organized regular meetings at the New York Academy of Medicine with other physicians interested in alcoholism and its treatment. These meetings led to the establishment, in 1954, of the New York City Medical Society on Alcoholism, which eventually became the American Medical Society on Alcoholism (AMSA). Another state medical society devoted to addiction as a subspecialty, the California Society for the Treatment of Alcoholism and Other Drug Dependencies, was established in the 1970s. By 1982, the American Academy of Addictionology was incorporated, and all these groups united within AMSA the following year. Because the organization was concerned with all drugs of addiction, not only alcohol, and was interested in establishing addiction medicine as part of mainstream medical practice, the organization was renamed the American Society on

Alcoholism and Other Drug Dependencies, which was soon changed to the American Society of Addiction Medicine (ASAM) in 1989. In 1988, the American Medical Association (AMA) House of Delegates admitted ASAM as a voting member and in 1990, the AMA recognized addiction medicine as a medical specialty. In the late 1990s, ASAM and the American Managed Behavioral Healthcare Association (AMBHA) began an ongoing collaboration to publish statements of consensus on various issues such as the effective treatment of addictive disorders and credentialing of clinical professionals, among others.

The stated mission and goals of ASAM are to increase access to and improve the quality of addictions treatment; educate physicians, medical students, and the public; promote research and prevention; and establish addiction medicine as a specialty recognized by the American Board of Medical Specialties.

By the late 1990s, membership in the society exceeded 3,500, with chapters in all 50 states, as well as overseas. Membership consists of private- and group-practice physicians, corporate medical directors, residents, and medical students, as well as retired physicians. ASAM-certified members with at least 5 years' active participation in the society, as well as involvement in related organizations and activities, may become Fellows.

Educational activities of the society are conducted through publications, courses, and clinical and scientific conferences. Publications include, among others, a bimonthly newsletter, *ASAM News*; the *Journal of Addictive Diseases*, published quarterly; the *ASAM Principles of Addiction Medicine*, a comprehensive reference guide; and the *Patient Placement Criteria for the Treatment of Substance-Related Disorders*, a clinical guide for matching adult and adolescent patients to appropriate levels of care. Courses include the Ruth Fox Course for Physicians; Medical Review Officer (MRO) certification training; and in-depth studies of addiction medicine. An annual medical-scientific conference includes scientific symposia, clinically oriented courses and workshops, and presentations of submitted papers.

In its continued effort to establish the legitimacy of addiction medicine as a subspecialty within medicine, ASAM administers a 6-hour certification examination, is a primary sponsor of medical postgraduate fellowships in alcoholism and drug abuse, and has developed guidelines for the training of physicians in this area of medical practice.

MARC GALANTER
JEROME H. JAFFE
REVISED BY NANCY FAERBER

AMERICAN SOCIETY FOR THE PREVENTION OF TEMPERANCE *See* Temperance Movement; Women's Christian Temperance Movement (WCTM)

AMOBARBITAL Amobarbital (Amytal) is one of the many different members of the BARBITURATE family of central nervous system depressants used to produce relaxation, sleep, anesthesia, and anticonvulsant effects. In terms of the duration of its effects, it is considered an intermediate-acting barbiturate. When taken by mouth, its sedating effects take about 1 hour to develop and last about 6 to 8 hours, although it takes considerably longer for all the drug to leave the body.

In addition to its use as a sedative, amobarbital is occasionally used in psychiatric evaluation in so-called "Amytal interviews," to relax patients in order to help them recall memories or information that has been repressed due to trauma. This technique was sometimes called narcoanalysis or narcotherapy.

DOSAGE AND ADMINISTRATION

Amobarbital may be given orally, intramuscularly, or intravenously for the treatment of insomnia or anxiety. The adult dosage for sedation is 15 to 50 milligrams but 65 to 200 milligrams for sleep. For treating convulsions, the adult dose is 65 to 200 milligrams, with a maximum dose of 500 milligrams.

Amobarbital should not be given to patients with a history of addiction; personal or family history of porphyria; severe kidney, liver, or lung disease; or hypersensitivity to barbiturates.

Amobarbital is incompatible with a number of medications, including dimenhydrinate, phenytoin, hydrocortisone, insulin, morphine, cimetidine, pancuronium, streptomycin, tetracycline, vancomycin, and penicillin G. It may decrease the

effectiveness of birth control pills containing estrogen. It has also been shown to increase the risk of birth defects if taken during pregnancy.

PSYCHIATRIC USE

The use of amobarbital in "Amytal interviews" has declined since the mid–1990s because of its relatively low success rate. One medical text published in the mid–1990s noted that the amount of clinically useful information obtained by this method is quite limited. Amobarbital interviews appear to be useful primarily in distinguishing between psychosis and delirium. Psychotic patients usually improve with amobarbital, whereas delirious patients get worse.

DEPENDENCY AND ABUSE

Amobarbital has been largely replaced by benzodiazepine medications as a sedative because of the high risk of abuse. It has been dropped from the 1999 edition of the *Physicians' Desk Reference*, which implies that it is no longer manufactured in the United States. As of 2000, it is still available in Canada. Although amobarbital has been less popular with addicted patients than the more rapidly acting barbiturates (secobarbital and pentobarbital), it is still sold on the street as "blues" or "rainbows" (combinations of amobarbital and secobarbital). A daily dose of 500 to 600 milligrams is considered sufficient to produce dependence. The time necessary to produce dependence is estimated at 30 days. It has often been noted that the symptoms of barbiturate dependence resemble those of chronic alcoholism, though barbiturate withdrawl is more often associated with life-threatening complications than alcohol withdrawl.

EMERGENCY TREATMENT

Overdose. Although the toxic dose of amobarbital varies according to height, weight, and other factors, 1 gram taken by mouth usually produces serious poisoning in an adult. Two to 10 grams are usually a fatal dose. Emergency treatment is supportive, including oxygen administration if necessary, fluid therapy and other standard treatment for shock, and forced diuresis if the patient has normal kidney function. This procedure speeds the excretion of the barbiturate in the urine.

Withdrawal. The symptoms of withdrawal from amobarbital or any barbiturate may be severe or even fatal if the patient has been taking the drug in large doses (800 mg/day). The barbiturate withdrawal syndrome is similar to delirium tremens. Within 12 to 20 hours after withdrawal, the patient becomes restless and weak. During the second and third days, 75 percent of patients develop convulsions, which may progress to status epilepticus and death. From the third to the fifth day, untreated withdrawal syndrome is marked by delirium, hallucinations, insomnia, fever, and dehydration. To prevent withdrawal syndrome, patients are treated with a dose of phenobarbital equivalent to one-third of the daily dose of amobarbital on which they are dependent. This initial dose of phenobarbital is decreased by 30 milligrams per day until the patient's system is clear of drugs.

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SCOTT E. LUKAS
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AMOTIVATIONAL SYNDROME This term refers to a hypothetical effect produced by drugs, especially MARIJUANA, whereby individuals lose interest or the ability to engage in activities motivated by normal psychological processes. It is associated with lethargy, a severe reduction in activities, unwillingness to work, failure to meet responsibilities, and neglect of personal needs including hygiene and nutrition (despite efforts by others to help and despite statements by the indi-

viduals that they wish they could get started in these activities).

The experimental evidence of the existence of this syndrome has been mixed, but it is generally believed that amotivation is rarely caused by a drug alone; it is instead the result of a complex interaction among the effects of the drug, the personality and experience of the individual, and the context in which the drug is repeatedly administered. In addition, there is some confusion as to whether this syndrome is seen only during drug intoxication and is therefore transient or whether it is a more permanent consequence that persists for a long period of time following cessation of drug use.

(SEE ALSO: *Cannabis sativa; Complications*)

CHRIS-ELLYN JOHANSON

AMPHETAMINE Amphetamine was first synthesized in 1887, but its central nervous system (CNS) stimulant effects were not noted at that time. After rediscovery, in the early 1930s, its use as a respiratory stimulant was established and its properties as a central nervous system stimulant were described. Reports of abuse soon followed. As had occurred with cocaine products when they were first introduced in the 1880s, amphetamine was promoted as being an effective cure for a wide range of ills without any risk of addiction. The medical profession enthusiastically explored the potentials of amphetamine, recommending it as a cure for everything from alcohol hangover and depression to the vomiting of pregnancy and weight reduction. These claims that it was a miracle drug contributed to public interest in the amphetamines, and they rapidly became the stimulant of choice—since they were inexpensive, readily available, and had a long duration of action.

Derivatives of amphetamine, such as METHAMPHETAMINE, were soon developed and both oral and intravenous preparations became available for therapeutic uses. Despite early reports of an occasional adverse reaction, enormous quantities were consumed in the 1940s and 1950s, and their liability for abuse was not recognized. During World War II, the amphetamines, including methamphetamine, were widely used as stimulants by the military in the United States, Great Britain, Germany, and Japan, to counteract fatigue, to increase alertness during battle and night watches, to increase endurance, and to elevate mood. It has been estimated that approximately 200 million Benzedrine (amphetamine) tablets were dispensed to the U.S. armed forces during World War II. In fact, much of the research on performance effects of the amphetamines was carried out on enlisted personnel during this period, as the various countries sought ways of maintaining an alert and productive armed force. Although amphetamine was found to increase alertness, little data were collected supporting its ability to enhance performance.

Since 1945, use of the amphetamines and COCAINE appears to have alternated in popularity, with several stimulant epidemics occurring in the United States. There was a major epidemic of amphetamine and methamphetamine abuse (both oral and intravenous) in Japan right after the war. The epidemic was reported to have involved, at its peak, some half-million users and was related to the release with minimal regulatory controls of huge quantities of surplus amphetamines that had been made for use by the Japanese military. Despite this experience, there were special regulations governing their manufacture, sale, or prescription in the United States until 1964 (Kalant, 1973).

The first major amphetamine epidemic in the United States peaked in the mid-1960s, with approximately 13.5 percent of the university population estimated, in 1969, to have used amphetamines at least once. By 1978, use of the amphetamines had declined substantially, contrasting with the increase of cocaine use by that time. The major amphetamine of concern in the United States in the 1990s is methamphetamine, with pockets of "ice" (smoked methamphetamine) abuse.

Amphetamines are now controlled under Schedule II of the CONTROLLED SUBSTANCES ACT. Substances classified within this schedule are found to have a high potential for abuse as well as currently accepted medical use within the United States. Amphetamine, methamphetamine, cocaine, METHYLPHENIDATE, and phenmetrazine are all stimulants included in this schedule.

MEDICAL UTILITY

Amphetamines are frequently prescribed for the treatment of narcolepsy, obesity, and for childhood ATTENTION DEFICIT DISORDER. They are clearly ef-

ficacious in the treatment of narcolepsy, one of the first conditions to be successfully treated with these drugs. Although patients with this disorder can require large doses of amphetamine for prolonged periods of time, attacks of sleep can generally be prevented. Interestingly, tolerance does not seem to develop to the therapeutic effects of these drugs, and most patients can be maintained on the same dose for years.

Although the amphetamines have been used extensively in the treatment of obesity, considerable evidence exists for a rapid development of tolerance to the anorectic (appetite loss) effects of this drug, with continued use having little therapeutic effect. These drugs are extremely effective appetite suppressants, but after several weeks of use the dose must be increased to achieve the same appetite-suppressant effect. People remaining on the amphetamines for prolonged periods of time to decrease food intake can reach substantial doses, resulting in toxic side effects (e.g., insomnia, irritability, increased heart rate and blood pressure, and tremulousness). Therefore, these drugs should only be taken for relatively short periods of time (4–6 weeks). In addition, long-term follow-up studies of patients who were prescribed amphetamines for weight loss have not found any advantage in using this medication to maintain weight loss. Data indicate that weight lost under amphetamine maintenance is rapidly gained when amphetamine use is discontinued. In addition to the lack of long-term efficacy, the dependence-producing effects of amphetamines make them a poor choice of maintenance medication for this problem.

The use of amphetamines in the treatment of attention deficit disorders in children, remains extremely controversial. It has been found that the amphetamines have a dramatic effect in reducing restlessness and distractibility as well as lengthening attention span, but there are side effects. These include reports of growth impairment in children, insomnia, and increases in heart rate. Those promoting their use point to their potential benefits and they advocate care in limiting treatment dose and duration. Opponents of their use, while agreeing that they provide some short-term benefits, conclude that these do not outweigh their disadvantages. Amphetamine therapy has also been attempted, but with little success, in the treatment of Parkinson's disease, and both amphetamine and cocaine have been suggested for the treatment of depression, although the evidence to support their efficacy does not meet current standards demanded by the U.S. Food and Drug Administration.

PHARMACOLOGY

The amphetamines act by increasing concentrations of the neurotransmitters DOPAMINE and NOREPINEPHRINE at the neuronal synapse, thereby augmenting release and blocking uptake. It is the augmentation of release that differentiates amphetamines from cocaine, which also blocks uptake of these transmitters. Humans given a single moderate dose of amphetamine generally show an increase in activity and talkativeness, and they report euphoria, a general sense of well-being, and a decrease in both food intake and fatigue. At higher doses repetitive motor activity (i.e., stereotyped behavior) is often seen, and further increases in dose can lead to convulsions, coma, and death. This class of drugs increases heart rate, respiration, diastolic and systolic blood pressure, and high doses can cause cardiac arrhythmias. In addition, the amphetamines have a suppressant effect on both rapid eye movement sleep (REM)—the stage of sleep associated with dreaming—and total sleep. The half-life of amphetamine is about ten hours, quite long when compared to a stimulant like cocaine, which has a half-life of approximately one hour, or even methamphetamine which has a half-life of about five hours.

The amphetamine molecule has two isomers: the *d*-(+) and *l*-(−) isomers. There is marked stereoselectivity in their biological actions, with the *d*-isomer (dextroamphetamine) considerably more potent. For example, it is more potent as a locomotor stimulant, in inducing stereotyped behavior patterns, and in eliciting central nervous system excitatory effects. The isomers appear to be equipotent as cardiovascular stimulants. The basic amphetamine molecule has been modified in a number of ways to accentuate various of its actions. For example, in an effort to obtain appetite suppressants with reduced cardiovascular and central nervous system effects, structural modifications yielded such medications as diethylproprion and fenfluramine, while other structural modifications have enhanced the central nervous system stimulant effects and reduced the cardiovascular and anorectic actions, yielding medications such as

methylphenidate and phenmetrazine. These substances share, to a greater or lesser degree, the properties of amphetamine.

TOXICITY

A major toxic effect of amphetamine in humans is the development of a schizophrenia-like psychosis after repeated long-term use. The first report of an amphetamine psychosis occurred in 1938, but the condition was considered rare. Administration of amphetamine to normal volunteers with no histories of psychosis (Griffith et al., 1968) resulted in a clear-cut paranoid psychosis in five of the six subjects who received *d*-amphetamine for one to five days (120–220 mg/day), which cleared when the drug was discontinued. Unless the user continues to take the drug, the psychosis usually clears within a week, although the possibility exists for prolonged symptomology. This amphetamine psychosis has been thought to represent a reasonably accurate model of schizophrenia, including symptoms of persecution, hyperactivity and excitation, visual and auditory hallucinations, and changes in body image. In addition, it has been suggested that there is sensitization to the development of a stimulant psychosis—once an individual has experienced this toxic effect, it is readily reinitiated, sometimes at lower doses and even following long drug-free periods.

Amphetamine abusers taking repeated doses of the drug can develop repetitive behavior patterns which persist for hours at a time. These can take the form of cleaning, the repeated dismantling of small appliances, or the endless picking at wounds on the extremities. Such repetitive stereotyped patterns of behavior are also seen in nonhumans administered repeated doses of amphetamines and other stimulant drugs, and they appear to be related to dopaminergic facilitation. Cessation of amphetamine use after high-dose chronic intake is generally accompanied by lethargy, depression, and abnormal sleep patterns. This pattern of behavior, opposite to the direct effects of amphetamine, does not appear to be a classical abstinence syndrome. The symptoms may be related to the long-term lack of sleep and food intake that accompany chronic stimulant use as well as to the catecholamine depletion that occurs as a result of chronic use.

Animals given unlimited access to amphetamine will self-administer it reliably, alternating days of high intake with days of low intake. They become restless, tremulous, and ataxic, eating and sleeping little. If allowed to continue self-administering the drug, most will take it until they die. Animals maintained on high doses of amphetamines develop tolerance to many of the physically and behaviorally debilitating effects, but they also develop irreversible damage in some parts of the brain, including long-lasting depletion of dopamine. It has been suggested that the prolonged anhedonia seen after long-term human amphetamine use may be related to this, although the evidence for this is not very strong.

BEHAVIORAL EFFECTS

Nonhumans. As with all PSYCHOMOTOR STIMULANT drugs, at low doses animals are active and alert, showing increases in responding maintained by other reinforcers, but often decreasing food intake. Higher doses produce species-specific repetitive behavior patterns (stereotyped behavior), and further increases in dose are followed, as in humans, by convulsions, hyperthermia, and death. Tolerance (loss of response to a certain dose) develops to many of amphetamine's central effects, and cross-tolerance among the stimulants has been demonstrated in rats. Thus, for example, animals tolerant to the anorectic effects of amphetamine also show tolerance to cocaine's anorectic effects. Although there is tolerance development to many of amphetamine's effects, sensitization develops to amphetamine's effects on locomotor activity. Thus, with repeated administration, doses of amphetamine that initially did not result in hyperactivity or stereotypy can, with repeated use, begin to induce those behaviors when injected daily for several weeks. In addition, there is cross-sensitization to this effect, such that administration of one stimulant can induce sensitization to another one. In contrast to cocaine, however (in which an increased sensitivity to its convulsant effects develops with repeated use), amphetamines have an anticonvulsive effect.

Learned behaviors, typically generated by operant schedules of reinforcement, are generally affected by the amphetamines in a rate-dependent fashion. Thus, behaviors that occur at relatively low rates in the absence of the drug tend to be increased

at low-to-moderate doses of amphetamine, while behaviors occurring at relatively high frequencies tend to be suppressed by those doses of amphetamine. In addition, with high doses most behaviors tend to be suppressed. As is seen with other stimulants, such as cocaine, environmental variables and behavioral context can play a role in modulating these effects. For example, behavior under strong stimulus control shows tolerance to repeated amphetamine administration much more rapidly than does behavior under weak stimulus control. In addition, if the amphetamine-induced behavioral disruption has the effect of interfering with reinforcement delivery, tolerance to that effect develops rapidly. Tolerance does not develop to the amphetamine-induced disruptions when reinforcement density is increased or remains the same.

Amphetamines can serve as reinforcers in nonhumans and, as described above, can produce severely toxic consequences when available in an unlimited fashion. However, when available for a few hours a day, animals will take them in a regular fashion, showing little or no tolerance to their reinforcing effects.

Humans. A substantial number of studies have been carried out evaluating the effects of amphetamines on learning, cognition, and other aspects of performance. The data indicate that under most conditions the amphetamines are not general performance enhancers. When there is improvement in performance associated with amphetamine administration, it can usually be attributed to a reduction in the deterioration of performance due to fatigue or boredom. Attention lapses that impair performance after sleep deprivation appear to be reduced by amphetamine administration; however, as sleep deprivation is prolonged, this effect is reduced. A careful review of the literature in this area (Laties & Weiss, 1981) concluded that improvement is more obvious with complex, as compared with simple, tasks.

In addition, in trained athletes, whose behavior shows little variability, only very small improvements can be seen. Laties and Weiss have argued persuasively, however, that the small changes in performance induced by amphetamines can result in the 1 to 2 percent improvement that may make the difference in a close athletic competition. Although the facilitation in performance after amphetamine does not appear to be substantial, it is sufficient to "spell the difference between a gold medal" and any other. Unfortunately, such data have led athletes to take stimulants prior to athletic events, particularly those in which strenuous activity is required over prolonged periods (e.g., bicycle racing), leading to hyperthermia, collapse, and even death in some cases.

The mood-elevating effects of the amphetamines are generally believed to be related to their abuse. Their use is accompanied by reports of increased self-confidence, elation, frequently euphoria, friendliness, and positive mood. When amphetamine is administered repeatedly, tolerance develops rapidly to many of its subjective effects (such that the same dose no longer exerts much of an effect). This means that the user must take increasingly larger amounts of amphetamine to achieve the same effect. As with nonhuman research subjects, there is however, little or no evidence for the development of tolerance to amphetamine's reinforcing effects.

Experienced stimulant users, given a variety of stimulant drugs, often cannot differentiate among cocaine, amphetamine, methamphetamine, and methlyphenidate—all of which appear to have similar profiles of action. Since these drugs have different durations of action, however, it becomes easier to make this differentiation over time.

ABUSE

In the United States in the 1950s, nonmedical amphetamine use was prevalent among college students, athletes, truck drivers, and housewives. The drug was widely publicized by the media when very little evidence of amphetamine toxicity was available. Pills were the first form to be widely abused. Use of the drug expanded as production of amphetamine and methamphetamine increased significantly, and abusers began to inject it. An extensive black market in amphetamines developed, and it has been estimated that 50 to 90 percent of the quantity commercially produced was diverted into illicit channels. In the 1970s, manufacture of amphetamines was substantially curtailed, amphetamines were placed in Schedule II of the Controlled Substances Act, and abuse of these substances was substantially reduced. Perhaps only by coincidence, as amphetamine use declined, cocaine use increased.

The amphetamines, as with other stimulants, are generally abused in multiple-dose cycles (i.e.,

binges), in which people take the drug repeatedly for some period of time, followed by a period in which they take no drug. Amphetamines are often taken every three or four hours for periods as long as three or four days, and dosage can escalate dramatically as tolerance develops. Like cocaine binges, these amphetamine-taking occasions are followed by a "crash" period in which the user sleeps, eats, and does not use the drug. Abrupt cessation from amphetamine use is usually accompanied by depression. Mood generally returns to normal within a week, although craving for the drug can last for months.

There is little evidence for the development of physical dependence to the amphetamines. Although some experts view the "crash" (with lethargy, depression, exhaustion, and increased appetite) that can follow a few days of moderate-to-high dose use as meeting the criteria for a withdrawal syndrome, others believe that the symptoms can also be related to the effects of chronic stimulant use. When using stimulants people do not eat or sleep very much and, as well, catecholamine depletion may well be contributing to these behavioral changes.

TREATMENT

As of the mid-1990s, little information is available about the treatment of amphetamine abusers, and no reports of successful pharmacological interventions exist in the treatment literature. As with cocaine abuse, the most promising nonpharmacological approaches include behavioral therapy, RELAPSE PREVENTION, rehabilitation (e.g., vocational, educational, and social-skills training), and supportive psychotherapy. Unlike cocaine, however, minimal clinical trials with potential treatment medications for amphetamine abuse have been carried out. The few that have been attempted report no success in reducing a return to amphetamine use.

(SEE ALSO: *Amphetamine Epidemics; Pharmacokinetics; Treatment*)

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MARIAN W. FISCHMAN

AMPHETAMINE EPIDEMICS Amphetamine, METHAMPHETAMINE, and related compounds have relatively brief abuse histories, dating from the 1930s and 1940s. Similar to the other major PSYCHOMOTOR STIMULANT of abuse, COCAINE, the amphetamines are addictive, and a number of cycles of epidemic use have occurred in the United States and in other countries. Unlike cocaine, however, the amphetamines do not occur in nature and can only be synthesized in a laboratory—a distinction that significantly influences the manufacture, distribution, and abuse patterns of the drugs.

EARLY USE IN THE UNITED STATES

Amphetamines were initially synthesized in 1887, with methamphetamine being developed approximately thirty years later. The rise in the popularity of the amphetamines parallels that experienced during the introduction of cocaine. Exaggerated publicity and fallacious claims about amphetamines, combined with medical optimism concerning potential uses and a lack of understanding of abuse, contributed to a dramatic increase in public interest in amphetamines. In 1933, Central Nervous System stimulant actions of amphetamines were reported, about the same time that reports of their effectiveness in treating narcolepsy and Parkinson's disease were released. When the American Medical Association (AMA) approved the use of amphetamines for these disorders, a mild warning was added that "continuous doses higher than recommended" might cause "restlessness and sleeplessness," but physicians were assured that

"no serious reactions had been observed." Between 1932 and 1946 the pharmaceutical industry developed more than three dozen generally accepted clinical uses for amphetamines, among them the treatment of schizophrenia, morphine and codeine addiction, tobacco smoking, heart block, head injuries, infantile cerebral palsy, radiation sickness, low blood pressure, seasickness, and persistent hiccups. It was not until several decades later that the addictive properties and psychiatric complications of amphetamines were fully recognized by the medical community.

U.S. PATTERNS AND TRENDS

In the 1940s and 1950s amphetamines were prescribed liberally and soon surpassed cocaine as an illicit stimulant widely available on the street. The increase in the popularity of amphetamines was influenced by easy availability, low cost, and long duration of effect (eight to twelve hours). Between the 1930s and the 1970s the public could obtain amphetamines, such as Benzedrine, in a variety of over-the-counter (OTC) nasal inhaler preparations. Abuse involved breaking open the inhalers and ingesting directly or soaking the fillers in alcohol or coffee. Although inhaler use may have introduced hundreds of thousands of Americans to amphetamine abuse, this type of abuse was most prevalent in prison populations and among deviant groups. The ability to cause euphoria, dysphoria, and psychic stimulation resulted in removal of amphetamine-like drugs from OTC inhaler preparations in 1971. However, amphetamine products remained available in pill, capsule, or injectable form.

During World War II, methamphetamine and amphetamine were widely used by the American, British, German, and Japanese military as insomniacs and as stimulants to increase alertness during battle and night watches; they were used as well by war-related industries to enhance worker productivity. Perhaps as many as 200 million tablets and pills were supplied to American troops during the war. The U.S. armed services authorized the issue of amphetamines on a regular basis beginning with the Korean conflict, escalating to well over 225 million standard-dose tablets dispensed between 1966 and 1969.

R. R. Monroe and A. H. Drell (1947) reported that at the end of World War II, some soldiers who had used amphetamines returned home with drug habits. In addition, during the 1940s and 1950s enormous quantities of these drugs were prescribed in the civilian population without concern for any addictive effects, as the drugs continued to be marketed to treat obesity, narcolepsy, hyperkinesis, and depression. College students, athletes, truck drivers, and housewives began using amphetamines for nonmedical purposes, primarily to increase energy, decrease the need for sleep, lose weight, and elevate mood. Pharmaceutical production reached 3.5 billion tablets (about 20,000 standard dosage units per thousand U.S. residents) in 1958 and 10.0 billion tablets by 1970. In comparison, the medical use of amphetamines in 1996-98 averaged approximately 1.8 standard dosage units per thousand U.S. population.

One consequence of excessive production and widespread popularity of amphetamines was the diversion of pharmaceutical-grade drugs to illegal traffic and use. Drugs sold on the black market came from or would otherwise have gone to pharmaceutical companies, wholesalers, druggists, and physicians. Probably over half (and potentially 90 percent) of the total commercial product was diverted into the black market. In 1966, the Food and Drug Administration (FDA) estimated that more than 25 tons of amphetamine were illegally distributed (Fischman, 1990). One market for the product was composed of long-distance truck drivers who found that amphetamines allowed them to work for extended periods without resting. The all-night restaurants and truck stops served as a distribution network that spanned the entire country.

By the mid-1960s, the need for intervention and legislative controls over amphetamine production and distribution was clear. The Drug Abuse Control Amendments of 1965, passed by Congress, required increased record keeping throughout the system of manufacture, distribution, prescription, and sale. However, diversion of pharmaceutical amphetamine to illicit use continued. The Controlled Substances Act (CSA) was passed in 1970. It further established the legal foundation of the government's fight against drug abuse, and placed amphetamine and some related stimulant drugs in Schedule II—acknowledging the drugs' high potential for abuse, development of psychological or physical dependency, and restricted medical use. In 1971 the Justice Department began imposing quotas on legal amphetamine production.

A significant shift from abuse of oral preparations to abuse of the intravenous form occurred during the 1960s. Intravenous methamphetamine abuse, described by S. M. Pittel and R. Hofer (1970), was particularly prevalent in the Haight-Ashbury district of San Francisco, where "speed," the street name for amphetamine and methamphetamine, began to replace hallucinogenic drugs, such as LSD, in popularity. Escalating doses of methamphetamine were taken, often as a series of injections over several days or weeks—what came to be known as a "speed run." Exhaustion, then depression, accompanied the end of a run, followed by readministration of the drug to mitigate the unpleasant side effects and regain the previous euphoria and high—thus the cycle of high to low to high. Initially the drugs were diverted from pharmaceutical supplies. Later, some unscrupulous physicians who were already prescribing intravenous methamphetamine to treat heroin addiction became involved in illegal prescriptions. In 1963, injectable ampules of methamphetamine were voluntarily removed by manufacturers from sale to retail pharmacies in California.

Speed use escalated during the 1960s, with the Haight-Ashbury district serving as a focal point. With this escalation came an increase in violence and the diffusion of manufacturing and distribution of speed from Haight-Ashbury to other areas along the West Coast (Smith, 1970). Outlaw motorcycle gangs became heavily involved in methamphetamine manufacture and gained control of its clandestine production and distribution. Within the subculture, serial speed users became known as "speed freaks." A public campaign was initiated to inform users of the hazards associated with speed use. Partly as a result of the "speed kills" campaign, amphetamine and methamphetamine use dropped sharply after 1972. From 1972 through 1977, the characteristics of the drug-taking population changed from heavy users to predominantly light-to-moderate users, and a growing proportion were women.

Changes in Clandestine Manufacture. Because of increasing controls on the prescribing and marketing of amphetamine, the clandestine manufacture of methamphetamine became more widespread. The availability of illicitly synthesized methamphetamine varied greatly during the 1970s and 1980s. Analyses of street samples of drugs purported to be methamphetamine revealed that until 1974, specimens were on average less than 30 percent methamphetamine. From 1975 through 1983 the composition of methamphetamine in samples increased from 60 to over 95 percent. For the street samples submitted as stimulants, including those submitted as amphetamine, methamphetamine, or speed, methamphetamine made up a relatively small percentage between 1972 and 1979, but increased to approximately 60 percent in 1983. These data demonstrate the increasing predominance of methamphetamine in the speed market during this time period. Prior to the increase in quality of street speed, the products sold as methamphetamine or speed were usually a combination of phenylpropanolamine hydrochloride, ephedrine, and caffeine and referred to as "look-alike" speed. The term referred to the similarity of appearance of these drugs and of central nervous system effects. Other constituents also found in products purported to be speed included pseudoephedrine and cocaine.

Since the mid-1980s, virtually all substances marketed illicitly as amphetamine or by street terms, such as "speed," "crystal," "crank," "go," "go-fast," "zip," or "cristy," contain methamphetamine. By analyzing contaminants found in street methamphetamine samples, researchers have determined that clandestine manufacture of methamphetamine, rather than diversion of pharmaceutical products, now supplies the illicit marketplace. According to the U.S. Drug Enforcement Administration (DEA), methamphetamine has been the most prevalent clandestinely manufactured controlled substance in the United States, and one of the only widely abused controlled substances that can be made in the home. Along with the increase in methamphetamine laboratory seizures was a localized resurgence of methamphetamine abuse—since the clandestine manufacture of the methamphetamine in a community facilitates the development of a market for the drug. Clandestine labs also create other hazards for the community since the materials used (precursors, reagents, and solvents) are hazardous in the hands of inexperienced chemists, who may cause explosions and fires. Also, each pound of methamphetamine produced creates up to five pounds of hazardous wastes, and the operators (who rarely own the property) commonly discard the wastes on or near the site, creating long-lasting chemical contamination of the area. The number of laboratories seized declined in the early

1990s, largely because of the passage and enforcement of the Chemical Diversion and Trafficking Act of 1988, which placed under federal control the distribution of twelve precursor and eight essential chemicals used in the production of illicit drugs, including phenyl-2-propanone, the major methamphetamine precursor in use at the time.

In the late 1980s, however, the clandestine methamphetamine chemists brought into production a more efficient synthesis process utilizing ephedrine or pseudoephedrine as the precursor chemical. As knowledge of this process spread, in some cases not only by word of mouth, but also via the growing medium of the Internet, the number of clandestine labs began to increase again. In 1997, 98 percent of all clandestine laboratories seized by the Drug Enforcement Administration (DEA) were producing methamphetamine and, in 1999 more than 7,000 clandestine methamphetamine labs were seized, along with over 2,250 kg of methamphetamine. Figure 1 shows that the amount of methamphetamine seized domestically increased substantially from 1990 through 1999. While most of the labs seized early in the 1990s were in California, Texas, or Oregon, in 1998 the DEA seized labs in almost every state in the nation, with 371 labs seized in Missouri.

Another factor increasing the spread of methamphetamine was a change in control of the manufacturing and distribution process. Although motorcycle gangs continued to control a share of the market, in 1995 well-established Mexico-based polydrug trafficking organizations began manufacturing and distributing methamphetamine. Importing precursor chemicals and reagents into or through Mexico, these organizations established "superlabs" in Mexico and in southern California that were capable of producing ten pounds or more of high-purity methamphetamine in one to two days. These superlabs are in marked contrast to the more numerous and widely distributed "mom-and-pop" labs producing several ounces that may be set up in a motel room, a car trunk, or on a kitchen counter. By 1999, it was estimated that superlabs were producing approximately 85 percent of the methamphetamine in the U.S. mainland. Portions of the Crime Control Act of 1990 and the Chemical Diversion Control Act of 1993, as well as the Comprehensive Methamphetamine Control Act of 1996, were all enacted to counter the changes in the synthetic process, the changes in the trafficking patterns, the emergence of superlabs, and the proliferation of mom-and-pop labs for the clandestine manufacture of methamphetamine.

TABLE 1
Drugs and Their Precursors

Controlled Substance	*Precursor*
Methamphetamine	Phenyl-2-propanone
Amphetamine	Phenylacetic acid
Methamphetamine	Ephedrine
Phencyclidine (PCP)	Piperidine
Methaqualone	Anthranilic acid
Lysergide (LSD)	Ergotamine tartrate

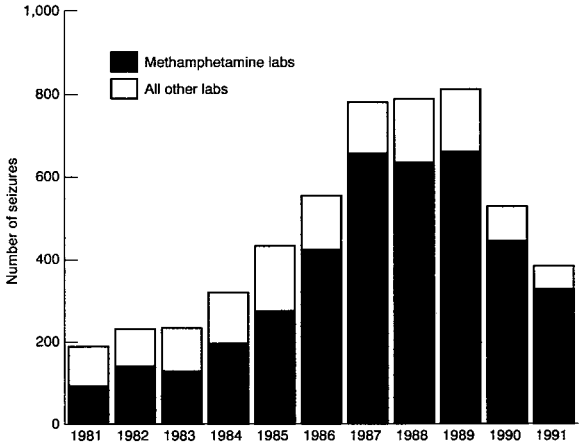

Figure 1
Number of Clandestine Methamphetamine Laboratories Seized in the United States, 1981–1991
SOURCE: U.S. Department of Justice (1992).

Changes in Indicators of Abuse. The DRUG ABUSE WARNING NETWORK (DAWN), a nationally based surveillance system that monitors emergency medical consequences and deaths related to drug use, reflected a stable trend across the United States from the mid-1970s until the mid-1980s. Over seven hundred hospitals in twenty-one metropolitan areas and a panel of hospitals outside of these areas report to DAWN. During the mid-1980s sharp increases in nonfatal emergency-room episodes began to appear, largely in metropolitan areas on the West Coast. Increases in drug-use indicators were also reported for methampheta-

mine through the Community Epidemiology Work Group (CEWG), a network of state and local drug-abuse experts representing twenty cities and metropolitan areas across the United States.

Total methamphetamine and amphetamine mentions in DAWN rose from earlier levels during 1988 and 1989, decreased during 1990 and 1991, then rose sharply during the early 1990s to reach an erratic higher plateau for the rest of the decade (see figure 2). Among DAWN emergency-room cases, the most common route of administration of methamphetamine was intravenous. Methamphetamine accounted for approximately 3.0 percent of the total DAWN drug mentions in 1994 and just under 2.5 percent in 1998, compared with 16.0 percent and 17.5 percent for cocaine during 1984 and 1998, respectively.

The Treatment Episode Data Set (TEDS) collects information nationwide on admissions to drug and alcohol treatment facilities that report to state administrative data systems. Data on Primary, secondary, and tertiary substances of abuse, their route of administration, frequency of use, and age at first use are among the data collected. In 1997, TEDS captured data on an estimated 67 percent of all U.S. drug and alcohol treatment admissions.

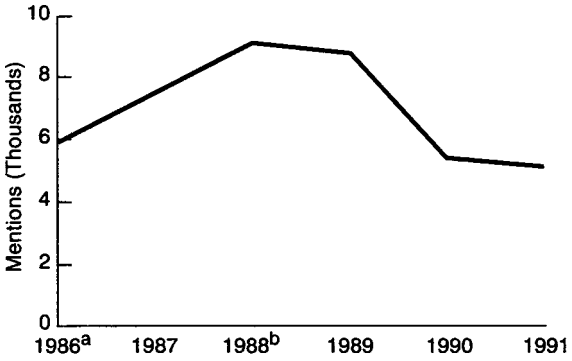

Figure 2
Trends in Methamphetamine-Related Emergencies, 1986–1991

SOURCE: The Drug Abuse Warning Network (DAWN).
NOTE: [a]Estimates for 1986–1987 are provisional. The estimates are based on a nonrandom sample of hospital emergency rooms in the coterminous U.S. [b]Estimates for 1988–1991 are based on a representative sample of nonfederal short-stay hospitals with 24-hour emergency rooms in the coterminous U.S.

From 1992 to 1997, both the absolute number of admissions reporting methamphetamine or amphetamine as the primary drug of abuse and the percentage of such admissions, relative to treatment admissions for all substances, more than tripled. The most common route was inhalation, but almost 30 percent of admissions reported injecting the drug.

The demographic profile of methamphetamine abusers in several studies that looked at different populations in the late 1980s and through the 1990s showed the majority of abusers to be predominantly Caucasian, low to middle income, high-school educated young adults generally ranging in age from 20 to 35, with slightly more males than females. However, by the end of the 1990s, there were indications of growing numbers of women and Hispanic abusers. Routes of administration tend to vary from locale to locale and from subgroup to subgroup, and include injecting intravenously, smoking (inhaling vaporized drug), and snorting. Methamphetamine abusers carry an increased risk of both Hepatitis B and HIV infections, predominantly through sharing of needles and increased unsafe sex practices.

Other U.S. Trends. At the same time that increases were being noted in methamphetamine use on the mainland of the United States, a new phenomenon was developing in Hawaii. Sharp rises in law-enforcement activity and in clients entering treatment because they smoked a new dosage form of methamphetamine were recorded between 1986 and 1989. The street names for this drug were *ice*, *crystal*, *shabu* (Japanese), and *batu* (Filipino for rock), and it looked like a large, usually clear crystal resembling broken fragments of glass or rock candy. Ice is of high purity (90 to 100 percent) and the d-isomer (the more psychoactive molecular form) of methamphetamine hydrochloride salt. In Hawaii it is almost always smoked in a glass pipe. The hydrochloride salt is sufficiently volatile to vaporize in a pipe so that it can be inhaled. This route of administration allows rapid absorption into the bloodstream, with onset of effects similar to those experienced with intravenous administration.

The use of ice was first detected by Hawaiian treatment programs during the summer of 1986, with more widespread use occurring into the 1990s. By 1997, The Treatment Episode Data Set reported that in Hawaiian drug and alcohol treat-

ment admissions, methamphetamine/amphetamine was the most commonly reported single primary substance of abuse, accounting for almost one quarter of all admissions. This epidemic, which was described in an outbreak investigation and follow-up field study conducted by the NATIONAL INSTITUTE ON DRUG ABUSE, involved a population varying widely in age and ethnic background and included both sexes. The ice-using treatment population was studied and reflected a younger population, with a higher representation of women and a larger proportion of Hawaiian/part Hawaiian than other drug users in treatment in the state. Ice was typically smoked in runs, or periods of continuous use, averaging three to eight days, with one or two days between runs, during which the user would "crash" into deep, prolonged sleep. Users reporting this pattern became rapidly addicted and experienced numerous adverse medical, social, and physiologic consequences. Precipitants of the epidemic included both a law enforcement campaign that effectively eradicated large portions of the Hawaiian marijuana or pakalolo crop, and well-orchestrated marketing campaigns by Asian ice distributors holding out ice as a replacement drug.

Until the late 1980s, the ice form of methamphetamine came only from Asia, specifically Hong Kong, Korea, Japan, Taiwan, Thailand, and the Philippines. Attempts to smuggle ice from Taiwan and Korea into Hawaii can be documented back to the mid-1980s by the Drug Enforcement Administration (DEA). The importation and distribution of ice in Hawaii has been linked to Asian and Hawaiian criminal organizations and gangs. By 1989, limited distribution of ice had occurred on the West Coast of the United States. In the following year, increased amounts of ice were found in California and subsequently in other limited locations. The increase in availability of ice was believed to stem from clandestine laboratories operating in California. During 1990, domestically manufactured ice began to be supplied to distributors in Hawaii and seven clandestine ice laboratories were seized nationwide, six of them in California. This domestic production was compensating for a disruption of the major Asian trafficking organizations smuggling ice from South Korea.

INTERNATIONAL PATTERNS AND TRENDS

The use and abuse of amphetamines has also occurred in countries outside the United States, although the absence of significant epidemiologic information in many countries and the lack of standardization in data collection and analysis make multinational comparisons difficult. Based on available data, amphetamine use and abuse appear to be an endemic problem worldwide and, as in the United States, other countries reflect patterns that are episodic, localized, population-specific, and rooted in multiple etiologies. Senay (1991) cites a number of studies conducted principally in the 1970s and first half of the 1980s that document this phenomenon. Amphetamine epidemics have been evident in several countries during the past fifty years and continue to be the primary concern in some. The experience of three of these, Japan, Sweden, and Thailand, are described briefly, but are described at greater length elsewhere (Kalant 1973, Klee 1997, Chaiyawong, 1999).

Japan. Methamphetamine has been the single most prevalent drug of abuse in Japan since the 1940s. Based on indicators from law-enforcement data, epidemic patterns appeared at several periods during that time.

The increase of methamphetamine abuse in Japan during the 1940s and early 1950s has been attributed to the wholesale appearance of the drug in the black market following World War II. Both amphetamine and methamphetamine were available to Japanese forces during the war and became widely used by the beleaguered civilian population following military defeat. In response to the escalation in abuse, Japan's Stimulants Drug Law of 1951 was enacted and the eventual decline in abuse was attributed to the effectiveness of the penal provisions of the law and subsequent control of the raw materials used to produce the drug. After a hiatus of fifteen years, Japan's methamphetamine abuse began to increase again and continued at relatively elevated levels through the mid-1980s. That epidemic was associated with illicit production of the drug and trafficking by criminal organizations—Yakuza and Boryokudan. The downturn in recent years is being attributed to the implementation of a stimulant-abuse prevention campaign that was begun in 1979.

Sweden. Amphetamines also have been at the forefront of the drug-abuse problem in Sweden since the 1930s. According to a 1988 report from the Swedish Council for Information on Alcohol and Other Drugs, abuse of amphetamines increased during the three decades following their introduction to the market as an over-the-counter (OTC) medication and their widespread promotion as a treatment for a variety of health conditions. While the prevalence of heavy, dysfunctional use during that era has been disputed, amphetamine abuse continues as a predominant problem in Sweden, especially among injection drug users.

Thailand. Thailand had a 1998 population of just over 60 million, with low unemployment (3.5 percent) and high literacy (94 percent). A major transit route for heroin from Laos and Burma, the country began experiencing a large shift in drug use patterns during the 1990s. The drug began to be more widely used in the late 1980s, as the synthesis from ephedrine came into practice. In the late 1990s, production had changed from being centralized in the hands of large crime syndicates running superlabs, to being shared with a multitude of small mom-and-pop labs—a pattern opposite that of the United States. Substantial increases have been seen in a number of use indicators. Figure 3 shows changes in the percentage of patients reporting drug use in the thirty days prior to admission for methamphetamine and heroin during the period 1993 to 1997.

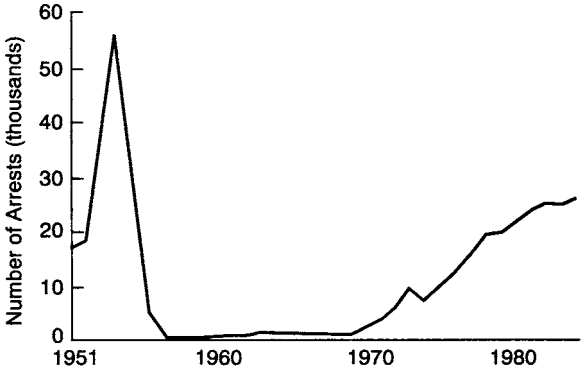

Figure 3
Methamphetamine Abuse in Japan, 1951–1985

CONCLUSION

In the United States, the overall magnitude of use and abuse of amphetamines, including methamphetamine, is relatively minor compared with the prevalence of other illicit drugs, such as marijuana and cocaine. The National Household Survey on Drug Abuse (NHSDA), a nationally representative survey of the household population age 12 and older, estimates that by 1998, 4.4 percent had ever used stimulants—including amphetamines, methamphetamine, and other prescription stimulants (U.S. Department of Health and Human Services, 1998). The NHSDA also estimated that 0.7 percent of the household population had used stimulants during 1998 and 0.3 percent had used them during the month prior to the interview. These numbers are in contrast to 35.8 percent estimated to have ever used any illicit drug, 33.0 percent who reported any use of marijuana, and 10.6 percent who had ever used cocaine. However, data from the national HIGH SCHOOL SENIOR SURVEY, Monitoring The Future, indicate that among twelfth graders surveyed each year since 1975, annual and past-30-day use of stimulants such as methamphetamines have always been substantially higher than use of cocaine. These differences in magnitude in no way diminish the impact of the health consequences and social problems, both at the individual and at the community level, resulting from amphetamine epidemics in the United States and in other countries.

In the United States, abuse of amphetamine and methamphetamine dates back to the early part of the twentieth century. During the ensuing years, abuse of amphetamine and methamphetamine has become endemic throughout this country, with focal problematic areas. Survey data and ethnographic information indicate a concentration of abuse in cities along the West Coast and in Hawaii that has been moving east and north across the United States. Historically, the typical composite methamphetamine user was white, male, young adult, and with a low to middle income, but this picture may be changing. As was experienced in the Hawaiian "ice" outbreak, and as seen recently in California, methamphetamine users can include diverse ethnic and socioeconomic groups. Methamphetamine is reported by the Drug Enforcement Administration (DEA) to be the most common product of illicit drug laboratories in the United

States. With extensive production and distribution systems in place—and potentially serious medical, psychological, and social consequence to abuse—these drugs continue to pose a significant public health threat.

The literature suggests that abuse of amphetamines has been and remains an endemic problem among diverse populations in countries throughout the world, at times reaching epidemic proportion. Measurement of the scope of drug abuse, trend analysis, and valid cross-cultural comparisons are fraught with difficulties. However, based on history, it is clear that the prevention of future epidemics requires the implementation of an effective program of international drug-abuse surveillance, communication, and early intervention.

(SEE ALSO: *Epidemics of Drug Abuse; Ethnicity and Drugs*)

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AMSTERDAM, DRUG USE IN *See* Netherlands, Drug Use in

AMYGDALA The amygdala, a region of the brain, is part of the limbic system. The limbic system is a group of similar brain structures related functionally. They provide the basis for emotion and motivated behaviors including REWARD-related events. The amygdala is located in the temporal lobe and consists of several different parts. It plays a role in various brain functions including epilepsy, emotion, learning and memory, and drug abuse.

In particular, the role of the extended amygdala has become an area of recent investigation. The extended amygdala refers to a group of brain structures that extend from the amygdala to the NUCLEUS ACCUMBENS; these brain regions are believed to participate in the general reward circuitry of the brain. The MESOLIMBIC DOPAMINE SYSTEM sends projections to the amygdala; these axons arise from the dopamine cells in the VENTRAL TEGMENTAL AREA.

The amygdala has long been established as an important area mediating stimulus-reward associations. This behavior is believed to play an important role in the seeking and using of drugs of abuse, especially COCAINE. An informative way to study drug abuse in animal models is through the SELF-ADMINISTRATION of drugs that are abused by humans. Rats can be trained to self-administer cocaine, and then the experimenter can interfere with the neurochemical transmission in the amygdala in particular, modulating DOPAMINE RECEPTORS and concentrations. The result of this manipulation is that the animals will increase or decrease their rate of administration of drugs. Thus, the amygdala makes a significant contribution to the study of cocaine-taking behavior.

The amygdala also contributes to the rewarding properties of ETHANOL. Studies have examined the effect of altering neurotransmission in the amygdala on ethanol self-administration. Similar to the findings reported for cocaine, modulation of the amygdala causes animals to change their rate of ethanol administration.

The amygdala is also involved in the effects of chronic drug exposure on the brain. Small changes in neurochemicals in the extended amygdala suggest that it may be mediating chronic drug action. These studies indicate that changes in the amygdala after long-term drug exposure may contribute to relapse.

Together, the amygdala and nucleus accumbens may be the main brain regions that underlie the brain changes associated with drug (particularly cocaine) addiction.

STEPHANIE DALLVECCHIA-ADAMS

AMYL NITRATE *See* Inhalants

AMYTAL *See* Amobarbital

ANABOLIC STEROIDS Anabolic steroids are synthetic versions of the naturally occurring male sex hormone, testosterone. They are more properly called anabolic-androgenic steroids (AASs), because they have both bodybuilding (anabolic) effects and masculinizing (androgenic) effects. The masculinizing effects of testosterone cause male characteristics to appear during puberty in boys, such as enlargement of the penis, hair growth on the face and pubic area, muscular development, and deepened voice. Females also produce natural testosterone, but ordinarily in much smaller amounts than males.

AASs are sometimes referred to simply as steroids. Steroid means only that a substance either resembles cholesterol in its chemical structure or is made from cholesterol in the body. Thus, AASs are

one kind of steroid. (They are not to be confused with an entirely different group of steroids called corticosteroids—of which prednisone and cortisone are examples—which are commonly used to treat illnesses such as arthritis, colitis, and asthma. In contrast to anabolic steroids, corticosteroids can cause muscle tissue to be wasted.) AASs are also referred to as ergogenic drugs, which means performance-enhancing. Street or slang terms for AASs include "roids" and "juice."

Soon after testosterone was first isolated and synthesized in the laboratory in 1935, a number of synthetics were created to be used as medicines. The synthetic forms were developed because natural testosterone did not work very long when given as a pill or injection (it is subject to rapid breakdown in the body). Bodybuilders may have begun using AASs to build muscle size and strength as early as the 1940s. Olympic athletes started to use these drugs in the 1950s. Most of this use went undetected, however, because the technology of drug testing did not allow reliable detection of AASs in the urine until the 1976 Olympic Games. Even so, anabolic steroids did not become a household word until Canadian sprinter, Ben Johnson, tested positive for AASs at the Seoul Olympic Games in 1988. In the same year, a study reported that 6.6 percent of American male high school seniors had tried AASs. This study made it clear that elite athletes were not the only ones taking

TABLE I
Anabolic Steroids Used by Bodybuilders

Generic Name	*Representative Brand Names*
Injectable testosterone esters[a]	
Testosterone cypionate	Depo-Testosterone (Slang name: Depo-T), Virlon IM
Testosterone enanthate	Delatestryl
Testosterone propionate	Testex, Oreton propionate
Other injectables	
Nandrolone decanoate	Deca-Durabolin (Slang names: Deca, Deca-D)
Nandrolone phenpropionate	Durabolin
Methenolone enanthate	Primobolan Depot
Veterinary injectables used by humans	
Trenbolone acetate	Finaject (Finajet) 30, Parabolan
Boldenone undecylenate	Equipoise
Stanozolol	Winstrol V
Pills (17-alkylated AASs)[b]	
Ethylestrenol	Maxibolan
Fluoxymesterone	Halotestin
Methandrostenolene	Dianabol (Slang names: D-bol, D-ball)
Methenolone	Primobolan
Methyltestosterone	Android (10 & 25), Metandren, Oreton Methyl, Testred, Virilon
Oxandrolone	Anavar
Oxymetholone	Adroyd, Anadrol-50
Oxymesterone	Oranabol
Stanozolol	Winstrol

[a]Least toxic to liver and cholesterol levels; cause estrogen levels to increase.
[b]Most toxic to liver and cholesterol levels.

these drugs. By 1991, AASs were added by federal law to the list of Schedule III of the Controlled Substances Act. Schedule III Controlled Substances are recognized to have value as prescribed medicines, but also have a potential for abuse that may lead to either low to moderate physical dependence or high psychological dependence. Table 1 lists the names of some AASs that bodybuilders have used. Hundreds of AASs have been synthesized, and more comprehensive lists exist (Wright & Cowart, 1990; Yesalis, 2000).

Two naturally occurring steroids, dehydroepiandrosterone (DHEA) and androstenedione, are used by the body to make testosterone and estrogen (Corrigan, 1999). The benefits and adverse effects of synthetic DHEA and androstenedione are mostly unknown, but they are commonly believed to have anabolic and androgenic effects. Unlike other AASs, DHEA and androstenedione are neither regulated by the Food and Drug Administration nor listed as controlled substances in the United States. DHEA has been sold over-the-counter as a nutritional supplement in the United States since 1994, even though the International Olympic Committee, many U.S. sports organizations, and some countries such as Australia ban it.

GENERAL CHEMICAL STRUCTURE

Testosterone has a four-ring structure composed of nineteen carbon atoms. Accordingly, the carbon atoms are labeled by number from one to nineteen (see Figure 1). Many synthetic forms of testosterone are made by adding either an alkyl group or an ester to the seventeen-carbon atom. An alkyl group is a chain of carbon and hydrogen atoms. An ester is formed by reacting an acidic chain of carbon and hydrogen atoms to the -OH group on the seventeen-carbon atom. In general, when an alkyl group is added to the seventeen-carbon atom, the resulting drug can be taken as a pill; however, these so-called seventeen-alkylated AASs are relatively toxic to the liver and are more likely to cause negative effects on cholesterol levels. By contrast, when an ester is formed at the seventeen-carbon atom, an injectable form of testosterone is created that is less toxic to the liver and cholesterol levels. Other AASs are created by making modifications at other carbon atoms.

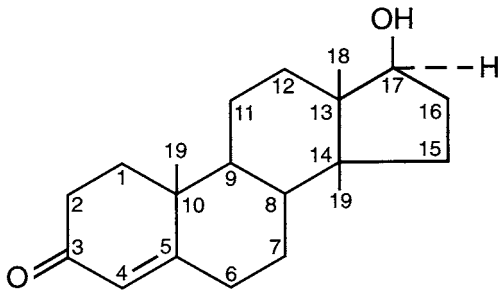
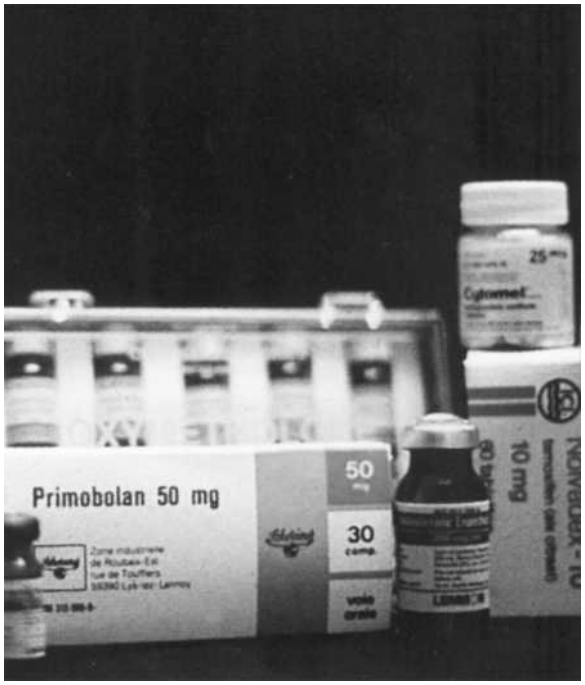


Figure 1
Testosterone Molecule.
The numbers refer to carbon atoms, and the hydrogen and hydroxyl groups are at carbon 17.

MEDICAL AND NONMEDICAL USES

AASs are prescribed by physicians to treat a variety of medical conditions (Bagatell & Bremner, 1996). The most accepted use is for treating boys and men unable to produce normal levels of their own testosterone, a condition known as testosterone deficiency or hypogonadism. AASs are also used to treat a rare skin condition called hereditary angioedema, certain forms of anemia (deficiency of red blood cells), advanced breast cancer, and endometriosis (a painful condition in females in which tissue usually found only in the uterus develops in other body parts). AASs are also combined with female hormones to treat distressing symptoms that can accompany menopause. Experimentally, AASs have been used to treat a condition in which bone loss occurs (osteoporosis), to treat impotency and low sexual desire, and as a male birth control pill. In addition, AASs have been used in the treatment of Acquired Immune Deficiency Syndrome (AIDS) to stimulate appetite, weight gain, strength, and improvements in mood. Most of these medical uses are uncommon, either because the conditions are rare (such as angioedema) or because other treatments are preferred (such as erythropoeitin for anemia). Nevertheless, AASs are important medicines to have available.

Nonmedically, AASs are used to enhance athletic performance, physical appearance, and fighting ability. Since society endows people who look physically fit and attractive with many benefits and recognition, some individuals see AASs as a means to those benefits. Three groups of AAS users have been described:



Most illegal anabolic steroids are sold at gyms, competitions, and through mail order operations; some of those commonly encountered are pictured. (Drug Enforcement Administration)

1. The athlete group aims to win at any cost. The athlete also believes, sometimes correctly, that the competition is using AASs. The anticipated rewards to the athlete are the glory of victory, social recognition and popularity, and financial incentives (college scholarships, major league contracts).
2. The aesthete group aims to create a beautiful body, as if to make the body into a work of art. Aesthetes may be competitive bodybuilders, or aspiring models, actors, or dancers. They put their bodies on display to obtain admiration and financial rewards.
3. This group of AAS users seeks to enhance their ability to fight or intimidate. They include body guards, security guards, prison guards, police, soldiers, bouncers, and gang members. These people depend on fighting for their very survival.

Whether AASs actually work to improve performance and appearance has been debated. Invariably, users believe AASs do work, but some scientific studies have failed to show an effect. However, there are serious limitations to how these studies were done and what they could show. In general, most researchers agree that AASs can work in some individuals to enhance muscle size and strength when combined with a proper exercise program and diet (Bagatell & Brenner, 1996; Bhasin et al., 1996). By contrast, AASs probably do not improve performance of aerobic or endurance activities (Yesalis, 2000).

CONSEQUENCES OF USE

AASs have been associated with a variety of undesirable effects. The most severe consequence attributed to their use is death. One study of mice given AASs revealed a shortened life span (Bronson & Matherne, 1997). In humans, the distinction between fatalities that occur among relatively healthy athletes who use AASs and patients with illnesses (such as anemia) who are prescribed AASs is important, because ill patients are already at a higher risk for an early death. Nevertheless, reported deaths in nonmedical steroid users (such as athletes and aesthetes) have occurred from liver disease, cancer, heart attacks, strokes, and suicide (Yesalis, 2000; Pope & Brower, 2000). Clearly, anyone using AASs should have their health monitored by a physician.

Psychiatric Effects. Another serious, life-threatening consequence has been violent aggression toward other people. Both the medical literature and newspapers contain reports of previously mild-mannered individuals who committed murder and lesser assaults while taking AASs (Thiblin et al., 1997). Although reports of severe violence generate both alarm and widespread attention, the total number of such reports is small. Moreover, the effects of AASs on violent behavior vary widely depending on the social circumstances and the characteristics of the individual. Nevertheless, most but not all studies in humans have found that high doses of AASs increase feelings and thoughts of aggressiveness (Yesalis & Cowart, 1998). Although an increase in feelings and thoughts of violence does not always lead to violent behavior, it can be very distressing to the individual and to those around him or her. ''Roid rage'' is a slang expression used to describe the aggressive feelings, thoughts, and behaviors of AAS users.

Other psychiatric effects of AASs include mood swings and psychosis (Pope & Brower, 2000). AAS

users commonly report that they feel energetic, confident, and even euphoric during a cycle of use. They may have a decreased need for sleep or find it difficult to sleep because of their high energy level. Such feelings may give way to feeling down, depressed, irritable, and tired between cycles of use. With continued use of high AAS doses, moods may shift suddenly, so that the user feels on top of the world one moment, irritable and aggressive the next, and then depressed or nervous. The appetite may also swing widely with cycles of use (Wright & Cowart, 1990). During a cycle on AASs, huge quantities of food may be consumed to support the body's requirements for muscle growth and energy. During the "off cycles," appetite may diminish.

The term "psychosis" means that a person cannot distinguish between what is real and what is not. For example, a person may believe that other people intend harm when no real threat exists; or a person may believe that an impossible, life-threatening stunt can be performed with no problem. Such false beliefs are called delusions. The psychotic person may also experience hallucinations, such as hearing a voice that is not there. Fortunately, most psychiatric effects of AASs tend to disappear soon after AASs are stopped, although a depressed mood may last for several months. Obviously, when suicides, homicides, or legal consequences from assault have occurred, they cannot be reversed simply by stopping one's use of AASs.

Effects on the Liver. AASs can affect the liver in various ways, but the seventeen-alkylated AASs are more toxic to the liver than other AASs. Most commonly, AASs cause the liver to release extra amounts of enzymes into the bloodstream that can be easily measured by a blood test. The liver enzymes usually return to normal levels when AASs are stopped. The liver also releases a substance called bilirubin, which in high amounts can cause the skin and eyes to turn yellow (a condition called jaundice). As many as 17 percent of patients treated with the seventeen-alkylated AASs develop jaundice (Yesalis, 2000). Nonmedical AAS users can also develop jaundice. Although untreated jaundice can be dangerous and even fatal, jaundice usually disappears within several weeks of stopping AASs. Jaundice can also signal other dangerous conditions of the liver, such as hepatitis, so a physician should always treat it. Another condition that occurs among patients treated with AASs is peliosis hepatis, in which little sacs of blood form in the liver. Death can occur from bleeding if one of the sacs ruptures. Finally, liver tumors may occur in 1 to 3 percent of individuals (including athletes) using high doses of the seventeen-alkylated AASs for more than two years (Yesalis, 2000). Rare cases of liver tumors have been reported with other types of AASs as well. Some of the tumors are cancerous, and although more than half of the tumors disappeared when AASs were stopped, others resulted in death.

Potential to Affect the Heart. AASs can cause changes in cholesterol levels (Yesalis, 2000). Low amounts of a certain kind of cholesterol (high-density lipoprotein cholesterol) in the blood are known to increase the risk of heart attacks. AASs, especially the seventeen-alkylated ones, cause a lowering of this so-called good form of cholesterol. When AASs are stopped, however, cholesterol levels return to normal. Another risk factor for heart attacks and strokes is high blood pressure. Studies have shown that AASs can cause small increases in blood pressure, which return to normal when AASs are stopped. As a result of strenuous exercise, many athletes develop an enlarged heart that is not harmful. Some, but not all studies, suggest that AAS users can develop a harmful enlargement of the heart. As noted previously, heart attacks and strokes have been reported in AAS users, but studies are needed to determine if AAS users have a higher risk of heart attacks and strokes than nonusers (Yesalis, 2000).

Sexual Side Effects. AASs can alter the levels of several sex-related hormones in the body, resulting in many adverse effects (Wright & Cowart, 1990; Yesalis & Cowart, 1998). In males, the prostate gland can enlarge, making it difficult to urinate; the testes shrink; and sterility can occur. The effects on the prostate, the testes, and sterility reverse when AASs are stopped; however, at least one case of prostate cancer has been reported, an exception to reversibility. Males can also develop enlarged breast tissue from taking AASs, an effect medically termed "gynecomastia" (it is referred to by male users as "bitch tits"). Gynecomastia occurs because testosterone is chemically changed in the body to the female hormone, estrogen. Thus, the male user experiences higher amounts of estrogen than normal. Painful lumps in the male breast may persist after stopping AASs, and they sometimes require surgical removal. Females, however, may undergo shrinkage of their breasts, as a response to

higher amounts of male hormone than normal. Menstrual periods become irregular and sterility can occur in females as well. Deepened voice and an enlarged clitoris are effects in females, which do not always reverse after stopping AASs. Women may also develop excessive hair growth in typically masculine patterns, such as on the chest and face. Finally, both males and females may experience increases and decreases in their desire for sex.

Other Effects. In children of both sexes before the onset of puberty, AASs can initiate the characteristics of male puberty and cause the bones to stop growing prematurely. The latter effect can result in shorter adult heights than would otherwise occur. AASs can cause premature baldness in some individuals, and it can cause acne. The acne is reversible with cessation of AASs. Other possible effects include small increases in the number of red blood cells, worsening of a condition called sleep apnea (in which afflicted persons stop breathing for short intervals during sleep), and worsening of muscle twitches (known as tics) in those who are predisposed.

Patterns of Illicit Use. AASs are commonly smuggled from countries where they are obtained over-the-counter without a prescription, and then sold illegally in the United States. Dealers and users typically connect in weight-lifting gyms. Users report that AASs are relatively easy to obtain.

Steroids are taken as pills, through skin patches, and by injection (Bagatell & Bremner, 1996). Injection occurs into large muscle groups (buttocks, thigh, or shoulder) or under the skin, but not into veins. Cases of acquired immune deficiency syndrome (AIDS) have been reported in steroid users due to needle sharing. Steroids are often taken in cycles of six to twelve weeks on the drugs, followed by six to twelve weeks off. At the beginning of a cycle, small doses are taken with the intent to build to larger doses, which are then tapered at the end of a cycle. Illicit users typically consume ten to one hundred times the amounts ordinarily prescribed for medical purposes, requiring them to combine or "stack" multiple steroid drugs. The actual dose cannot always be determined, however, because illicit steroids may contain both falsely labeled and veterinary preparations. (Drugs purchased on the illicit market do not always contain what the labels indicate, and law-enforcement officials have confiscated vials contaminated with bacteria.)

Steroid users commonly take other drugs, each with their own risks, to manage the unpleasant side effects of steroids, to increase the body-building effects, and/or to avoid detection by urine testing (Wright & Cowart, 1990). For example, estrogen blockers, such as tamoxifen or clomiphene, are taken to prevent breast enlargement. Water pills (diuretics) are taken both to dilute the urine prior to drug testing and to eliminate fluid retention so that muscles will look more defined. Human chorionic gonadotropin (HCG) is an injectable, nonsteroidal hormone that stimulates the testicles to produce more testosterone and to prevent them from shrinking. Human growth hormone is another nonsteroidal hormone that is taken to increase muscle and body size.

Addictive Potential. As with other drugs of abuse, dependence on AASs occurs when a user reports several of the following symptoms: Inability to stop or cut down use, taking more drugs than intended, continued use despite having negative effects, tolerance, and withdrawal. "Tolerance" refers to needing more of a drug to get the same effect that was previously obtained with smaller doses, or of having diminished effects with the same dose. In terms of the anabolic effects, tolerance was demonstrated in animals in the 1950s. In recent studies, 12 to 18 percent of nonmedical AAS users reported tolerance (Yesalis, 2000; Copeland et al., 2000). Whether tolerance develops to the mood-altering effects of AASs is unknown. *Withdrawal* refers to the uncomfortable effects users experience when they stop taking AASs. As noted previously, many of the undesirable effects reverse when AASs are stopped, however, others can begin—such as depressed mood, fatigue, loss of appetite, difficulty sleeping, restlessness, decreased sex drive, headaches, muscle aches, and a desire for more AASs (Copeland et al., 2000). The depression can become so severe that suicidal thoughts occur. The risk of suicide described previously is thought to be highest during the withdrawal period.

Studies indicate that between 14 and 57 percent of nonmedical AAS users develop dependence on AASs (Yesalis, 2000), and rare cases have been reported in women (Copeland et al., 2000). These studies support the addition of AASs to the list of Schedule III controlled substances. Nevertheless, AASs may differ from other drugs of abuse in several ways. First, neither physical nor psychological dependence on AASs has been reported to oc-

cur when AASs are prescribed for treating medical conditions. This differentiates the AASs from the opioid pain killers and the sedative-hypnotics. Second, dependence may develop primarily to the muscle-altering effects of AASs, rather than the mood-altering effects. Some researchers have questioned whether AASs produce dependence at all, because most definitions of dependence require that drugs be taken primarily for their mood-altering effects. Third, AAS users appear more preoccupied with their bodies and how they look than do users of other drugs of dependence.

SUMMARY

The anabolic-androgenic steroids are related to the male sex hormone, testosterone. They have both masculinizing and bodybuilding effects. AASs are useful to treat a variety of mostly uncommon medical conditions. They are sometimes used for the nonmedical purposes of enhancing athletic performance and physical appearance. Most researchers agree with users that AASs can increase muscle size and strength in some individuals when combined with a proper exercise program and diet. Many are also concerned about the potential for harmful effects with AASs, especially when the patterns of illicit use are considered. Drugs obtained on the illicit market may be contaminated, falsely labeled, or may contain substances not approved for human use. Multiple steroid and nonsteroidal drugs are combined, and AAS doses may exceed therapeutic doses by ten to one hundred times. Although the seventeen-alkylated AASs are commonly used because pills are more convenient than injections, they are more toxic to the liver and cholesterol levels than the injectable testosterone esters. Nevertheless, injections carry their own risks from improper injection techniques to dirty and shared needles.

The most serious side effects of AASs seem relatively uncommon, such as deaths or near-deaths from liver disease, heart attacks, strokes, cancer, suicide, and homicidal aggression. Most other side effects appear to be reversible when AASs are stopped, such as altered cholesterol levels, some liver effects, most psychiatric effects, testicular shrinkage, sterility, high blood pressure, and acne. Exceptions to reversibility include lumps in the male breast, deepened voice and enlarged clitoris in females, and cessation of bone growth in children. Moreover, some individuals may develop dependence on AASs, making it difficult for them to stop using. Stopping use can also produce distressing withdrawal symptoms, the worst of which is suicidal depression. Finally, studies of the long-term effects of using AASs are lacking, so safety cannot be assumed with the high-dose use of these drugs.

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Kirk J. Brower

ANALGESIC Analgesics are drugs used to control pain without producing anesthesia or loss of consciousness. Analgesics vary in terms of their class, chemical composition, and strength. Mild analgesics, such as aspirin (e.g., Bayer, Bufferin), ace-

tominophen (e.g., Tylenol), and ibuprofen (e.g., Advil), work throughout the body. More potent agents, including the OPIATES codeine and morphine, work within the central nervous system (the brain and spinal cord). The availability of the more potent analgesics is more carefully regulated than that of aspirin and other similar analgesic/anti-inflammatory agents that are sold in drugstores OVER-THE-COUNTER. The more potent opiate agents typically require prescriptions to be filled by pharmacists.

An important aspect of analgesics is that they work selectively on pain, but not on other types of sensation, such as touch. In this regard, they are easily distinguished from anesthetics which block all sensation. Local anesthetics, such as those used in dental work, make an area completely numb for several hours. General anesthetics typically are used to render patients unconscious for surgery.

(SEE ALSO: *Pain: Drugs Used in Treatment of*)

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GAVRIL W. PASTERNAK

ANESTHETIC *See* Inhalants

ANGEL DUST *See* Phencyclidine (PCP); Slang and Jargon

ANHEDONIA This term refers to a clinical condition in which a human or an experimental animal cannot experience positive emotional states derived from obtaining a desired or biologically significant stimulus. Generally, certain stimuli serve as positive reinforcers in normal individuals (e.g., food, water, the company of friends). "Positive reinforcement" is a descriptive term used by behavioral scientists to denote an increase in the probability of a behavior that is contingent on the presentation of biologically significant stimuli, such as food or water.

Anhedonia may be idiopathic (of unknown cause), may occur as a side effect of certain drugs (for example, the NEUROLEPTICS), which act as dopamine-receptor antagonists, or may be an aspect of certain psychiatric disorders, such as depression. It is conjectured that a state of anhedonia may occur during the "crash" that follows a prolonged bout of drug self-administration, particularly COCAINE or amphetamine-like stimulants.

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ANTHONY PHILLIPS

ANIMAL RESEARCH *See* Research, Animal Model

ANORECTIC This term derives from Greek (*a* + *oregein*, meaning "not to reach for"; later, *anorektos*) and it refers to a substance that reduces food intake. It came into use in English about 1900. Anorectic agents (also referred to as anorexics, anorexegenics, or appetite suppressants) fall into a number of categories according to the brain neurotransmitter system through which they work.

Central nervous system (CNS) stimulants that act through the noradrenergic and dopaminergic systems include COCAINE, amphetamine-like compounds, mazindol, and phenylpropanalamine. Serotonergic compounds include fenfluramine, fluoxetine, and sertraline. Several endogenous peptides (within the body) also have anorectic actions, in that they inhibit food intake—these include cholecystokinin, glucagon, and the bombesin-like peptides.

Not *all* agents that can suppress appetite are medically approved for such use. For example, cocaine is approved *only* as a local anesthetic.

(SEE ALSO: *Amphetamine*)

TIMOTHY H. MORAN

ANOREXIA This term means a "loss of appetite," especially when prolonged, and came into English in the 1620s from Latin usage, based on Greek stems (*a* [no] + *orexis* [appetite]). Anorexia generally leads to loss of weight due to a of loss of appetite; anorexia nervosa is an appetite disorder associated with severe weight loss. Eating disorders of this type and those associated with compulsive eating are, in some ways, behavioral equivalents of drug abuse.

(SEE ALSO: *Overeating and Other Excessive Behaviors*)

TIMOTHY H. MORAN

ANSLINGER, HARRY JACOB, AND U.S. DRUG POLICY For almost a third of a century, from 1930 until 1962, one man, Harry Jacob Anslinger (1892–1975), had the dominant role in shaping and enforcing U.S. policy about the use of drugs—other than alcohol and tobacco. Understanding his life and work is, therefore, a necessity for understanding the evolution of federal drug policies through the end of the twentieth century. Anslinger was Commissioner of the U.S. Treasury Department Bureau of Narcotics from 1930 to 1962, chief U.S. delegate to international drug agencies until 1970, and a leading proponent of repressive antidrug measures in the United States—and worldwide.

Anslinger was born in Altoona, Pennsylvania, May 20, 1892, the eighth of nine children in a Swiss immigrant family. At the age of twelve, he was sent by a neighbor to pick up a package of morphine from the drugstore. Anslinger said that he never forgot the screams of agony shrieked by the neighbor's wife because of her withdrawal symptoms, or how quickly she felt good upon getting a dose, or how easy it was for a twelve-year-old boy to buy morphine. Until the Harrison Act was passed in 1914—about a decade after this incident which haunted Anslinger for the rest of his life—there was simply no federal law against selling or using narcotics. They were inexpensive. Most persons started taking them as perfectly legal painkillers. A few of these persons became addicted, often without even realizing it. They merely used the narcotic so steadily that they didn't experience withdrawal. These addicts were typically white, lived in the countryside, continued to function satisfactorily at work or at home, could afford the cheap drugs they used, and caused no trouble to anyone else.

At the age of fourteen, Anslinger started working for the Pennsylvania Railroad while taking high school courses in his free hours. Without a high school diploma, he managed in 1913 to enter a Pennsylvania State College two-year program in engineering and business management, while continuing to work part-time for the railroad and also playing the piano for silent movies. By 1915, he was a railroad detective, and one summer day he had to assist an Italian railroad worker who had been beaten and left unconscious near the tracks by an organized-crime gangster.

In 1917, he volunteered to help the American effort in World War I and worked for the U.S. Army as assistant to the Chief of Inspection of Equipment. In 1918, Anslinger entered the U.S. diplomatic service. His First post was Holland, where he was assigned as liaison to deposed Kaiser Wilhelm's entourage; Wilhelm II was born in 1859, became Emperor (Kaiser, that is Caesar) of Germany and King of Prussia in 1888 upon the death of his father, Frederick III, reigned as emperor and king until 1918 when he fled to asylum in Holland, where he lived a comfortable life as a country gentleman until his death in 1940. Anslinger's assignment in Holland lasted three years, and then in the summer of 1921, he was sent to Hamburg, Germany. In 1923 he was reassigned from Germany to Venezuela for a frustrating three-year stint as U.S. vice-consul in La Guaira, the port for the capital city of Caracas (McWilliams, 1990).

THE FEDERAL BUREAU OF NARCOTICS

In 1920, the Prohibition Amendment had made the importing, manufacture, or sale of alcoholic beverages illegal throughout the United States and its possessions (a slight amount was permitted for sacramental and medicinal purposes). Of course, illegal liquor became an instant success. In 1926, Anslinger became U.S. consul in Nassau in the British Bahamas, which were then a principal location from which illegal alcohol was smuggled into

the United States. Consul Anslinger was quickly recognized for his effective work in persuading the British authorities to cooperate in curbing the flow of intoxicating beverages. The Volstead Act (1919) and the Harrison Act (1914), aimed respectively at enforcing Prohibition and controlling the distribution of narcotic drugs, were both tax measures, and hence came within the jurisdiction of the U.S. Treasury Department. Treasury soon borrowed Anslinger from the Department of State to serve in its Prohibition Bureau, which then enforced both acts. On July 1, 1930, three years before Prohibition ended, the drug-regulation functions were shifted to a new Bureau of Narcotics; Anslinger was named acting commissioner after other candidates were disqualified by scandal. President Herbert C. Hoover made Anslinger's appointment permanent on September 23, 1930.

REPRESSIVE POLICIES

Drug addiction, particularly genuine addiction to the OPIATES, had already been demonized when Anslinger came on the scene: Addicts had been labeled dope fiends in the public mind, and the illicit traffic had been attributed first to sinister German agents during World War I, then to terrifying Chinese tongs (secret societies). Clinics, set up to relieve the plight of addicts who had been cut off from their supplies by providing them with maintenance doses of HEROIN, were curbed by a series of U.S. Supreme Court rulings, interpreted by the Treasury Department to prohibit heroin maintenance as a form of medical treatment. The clinics were closed, and by 1925 doctors had stopped prescribing to addicts. A black market had begun to flourish parallel to that in alcohol.

Anslinger favored an extremely punitive approach from the outset. He cultivated members of Congress and other politicians, providing all who served his interests with material to portray themselves as fierce drug-fighters. He relentlessly opposed "education" about the realities of drug use—on the ground that it would encourage "youthful experimenters." His Bureau sounded one alarm after another: For example, drugs caused users to commit violent crimes (it was solemnly claimed, for a while, that dangerous criminals took drugs to sharpen their vicious courage in committing crimes) and that addicts induced others to become addicted (each addict made seven others in his career), so they were "infectious" in the community.

Sometimes the Bureau of Narcotics warned that an entire generation of American youth, including young children, was imperiled; then, suddenly, drugs would be revealed as responsible for a wave of juvenile delinquency and for the menace of "young hoodlums." The Bureau announced its enforcement success in terms of the total number of years of sentences imposed on drug offenders annually ("3,248 years, 10 months, 18 days" for 1933); SEIZURES OF DRUGS were announced at STREET VALUE, which grossly inflated their price. Because formal systems to measure levels of drug abuse were lacking, drug statistics could be and were manipulated—skyrocketing when Anslinger wanted support or appropriations, plummeting when he wanted credit and praise. Indeed, even before Anslinger became commissioner of narcotics in 1930, enforcement activities had resulted in an increase in federal prisoners and had led Congress to establish two U.S. Public Health Service Hospitals (one in Kentucky, one in Texas)to treat addicted inmates. They were authorized in January 1919, but it was 1935 before the first of the two actually opened. However, the major thrust of U.S. policy was control of supply and punishment of users.

MARIJUANA TAX ACT OF 1937

During his tenure as commissioner, Anslinger dominated the enactment of U.S. narcotics laws. In the mid-1930s, to puff the menace his Bureau was combating, he turned his attention to MARIJUANA (CANNABIS SATIVA [hemp]), used at the time by a few Spanish Americans, Caribbean Blacks, and in such limited circles as jazz musicians. A number of responsible studies of the effects of marijuana (such as one by the Hemp Commission in British India in 1895) and its more potent form, hashish, had pronounced it relatively harmless—but that gave Anslinger and a few other sensationalists of the day no pause. Shocking accounts of heinous crimes induced by marijuana began emanating from the Bureau; the theory that pot smoking was a dangerous "gateway" to other addictions gained credence; and a Bureau-sponsored film, *Reefer Madness*, was produced to popularize Anslinger's visions of the hazards of drug use. Viewed from the end of the

twentieth century, this film convulses audiences as classic kitsch.

Anslinger orchestrated the passage of a bill in Congress to place marijuana in the same highly restricted categories as HEROIN and COCAINE. President Franklin Delano Roosevelt signed the bill on August 2, 1937. The drug soon came to account for more enforcement activity than any other. Honest research on its toxic properties was stifled because the Bureau would not license its use by researchers outside of government. Although its therapeutic value in alleviating nausea due to chemotherapy for cancer patients or for treating glaucoma is generally recognized, it remains in the most strictly prohibited "dangerous drug" classification in the year 2000.

BOGGS ACT OF 1951

In the late 1940s, Anslinger launched an attack on judges, claiming that the drug problem was caused by too-lenient sentences imposed on drug offenders. This was picked up by Anslinger disciples in Congress and resulted in legislation—the Boggs Act, signed by President Truman on November 2, 1951, and amended by the Narcotics Control Act, signed by President Eisenhower on July 18, 1956. These acts introduced severe mandatory minimum punishments following conviction, at least two, five, and ten years in prison for repeated convictions, without probation or parole, and with a mandatory life sentence—or death, at a jury's discretion—for sale of heroin by an adult to a minor.

UNIFORM STATE LAWS

The Narcotics Bureau had similarly pressured state legislatures to enact extreme drug laws, promulgating a Uniform Narcotic Drug Act through the National Conference of Commissioners on Uniform State Laws in 1932, inducing the passage of tough marijuana measures after 1937, and promoting "Little Boggs Acts" in the late 1950s. Some of the latter laws contained penalties even more severe than the federal law, and it became common practice for Anslinger's men and local prosecutors to shuffle drug offenders into either state or federal courts, depending on where they would receive the harsher sentences.

Federal lawmakers were somewhat inhibited, in Anslinger's day, by the fact that federal drug laws were based solely on Congress's power to tax. States could enact penalties based on their general powers to punish crime, and some even prescribed punishment for the mere status of being an addict—until the Supreme Court ruled that practice out in Robinson v. California (1963). Little attention was paid to "treatment" or "rehabilitation"; addicts should either give up their wicked habits, and their spreading of the vice to others, or they should be isolated from society by "quarantine" somewhere. Toward the end of Anslinger's tenure, attention turned to "civil commitment," which sometimes in effect made the quarantine a life-sentence, at least when lawmakers provided scanty resources for rehabilitating those thus committed.

INTERNATIONAL DRUG POLICIES

By 1930, when Anslinger had become commissioner, the patterns of international controls had also been largely set, with the United States urging stringent repression and most of the rest of the world remaining indifferent or resistant. (The basic Hague Convention of 1912 would never have been ratified by more than a few nations had not the United States insisted upon its inclusion in the Paris peace treaties, which created the League of Nations in 1921.) Although the United States never joined the League of Nations, U.S. representatives were always given a voice in drug matters—and Anslinger dominated international deliberations, leading the U.S. delegations first to the drug-control agencies of the League of Nations and then to those of the United Nations (U.N.), even after his resignation as U.S. commissioner.

Anslinger's annual Bureau reports to the U.S. Treasury were also submitted as his official annual reports to the League Opium Advisory Committee and its successor, the U.N. Commission on Narcotic Drugs. He could thus push his views in the United States as recommendations endorsed by the international bodies and, simultaneously, present them to the latter as official statements of U.S. positions.

Anslinger participated in the drafting of the 1931 Narcotics Limitation Convention, which imposed controls on the production of drugs for legitimate medical uses; he pressed for the 1936 Convention for Suppression of Illicit Traffic, which sought to persuade other nations to impose

criminal sanctions on domestic distribution and consumption. When World War II isolated Geneva and ended most of the functions of the League of Nations based there, he arranged for moving the international drug agencies to New York City, where they continued to operate. After the war, he was the leading proponent of a Single Convention, finally approved in 1961, after ten years of drafting. It incorporated much of the U.S. law-enforcement orientation, including obligations upon members to control crops and production, to standardize identification and packaging, and to impose severe criminal penalties on drug offenders.

Although the Single Convention was widely ratified, many signatories ignored its requirements. Lacking enforcement sanctions, it had small effect. Some of Anslinger's more radical proposals, such as including the promotion of opium addiction in the definition of genocide, which he charged to enemies like the People's Republic of China and the Soviet Union, won no international support, but they played well at home.

THE ANSLINGER LEGACY

It is often asserted that Anslinger was forced out of the Federal Bureau of Narcotics by the Kennedy administration. Actually, he offered his resignation on his seventieth birthday, May 20, 1962, but was asked by the Kennedy administration to remain as acting commissioner until a successor could be found. He did so, and was pleased when his closest aide, Deputy Commissioner Henry L. Giordano, was appointed by President Kennedy as the new commissioner and promised that he would make no changes in policies established by Anslinger. Kennedy also permitted Anslinger to remain as U.S. representative to the United Nations, a post he continued to hold until 1970. Anslinger, for his part, spoke highly of the fact that Attorney General Robert Kennedy, the president's younger brother, went after top figures in the Mafia, not merely jailing addicts. After the Kennedy assassination in 1963, President Johnson (president from 1963 to 1969) moved much of the federal drug-control apparatus from Treasury to the Department of Justice.

Yet Anslinger's perspective seems to live on. Presidents Nixon (1969–1974), Reagan (1981–1989), and George Bush (1989–1993) intensified the drug war, justifying such efforts with arguments initially developed by Anslinger. Congress, too, continues to be influenced by Anslinger's views. Congressional rhetoric and penal statutes are more extreme in the year 2000 than those of the Boggs era. Marijuana is still lumped with heroin and cocaine, and new alarms—the discovery of a CRACK-baby epidemic and the menace of "ice"—are periodically trumpeted. Anslinger created and set a pattern of aggressive drug suppression—contrary to the initial purposes of the Harrison Act—and kept drug prohibition alive when the alcohol ban, Prohibition, which had truly been intended, was repealed in 1933. Anslinger may indeed have been, as critical Congressman John M. Coffee labeled him in 1938, "far and away the costliest man in the world."

Anslinger has often been portrayed as a racist who promoted and enforced extremely severe laws against marijuana once he realized that it was favored by blacks (as HASHISH, its use had been legal in Muslim Africa, and the Spanish permitted its importation to the Americas along with the slaves). It is a fact that Anslinger prevented the showing in the United States of a Canadian film *Drug Addict* on grounds that it would encourage youthful experimentation with drugs. Keys & Galliher (2000) claim: "The historical record paints a different picture of Anslinger's reasoning and demonstrates what was really unacceptable to Anslinger and the FBN [Federal Bureau of Narcotics]. Major themes of the film, addicts are recruited from all races and classes. . . . To emphasize this the film shows affluent whites injecting drugs. . . ." If Anslinger indeed objected to the film for showing affluent white addicts, one would logically suspect that he would object to the following passage for the same reason: "Many of the big dealers in the business of narcotic agony move in the most elite circles in both Europe and America. One notorious international trafficker, responsible for the addiction of millions, . . . with his edge of accent and his impeccable grooming, melted easily into the Park Avenue cocktail hour." But Anslinger did not object to this description of an affluent Parisian drug lord. Indeed, he is its author (Anslinger, 1961).

In hindsight, it is easy to say that in 1930 America would have benefited from a three-pronged approach to narcotics: punishment for organized crime when it imported drugs, medical treatment

for addicts, and honest education about the facts of drug use. But we must remember that Anslinger was basically a high school dropout. He never received a high school diploma or a degree from a four-year undergraduate college. He saw the world in one-dimensional black-and-white terms. He had seen the pain that addiction caused his neighbor's wife. He had seen the evil of organized crime. He had seen decisive American military force in World War I destroy the evil empires, as they were perceived, of Germany and Austria-Hungary. He had seen the Allies fight crime and exact severe punishment. (He could not have foreseen in 1930 that the severity of this punishment was misguided and would lead to Hitler and World War II.) He wanted to fight the evil of drug addiction. The solution seemed simple. Make everything connected with drug use—sale, use, importation, manufacture—illegal; lock up everyone involved in any way with any drug for as long as possible, preferably for life. He seems never to have realized that greater emphasis on treatment and education would have made drugs less profitable to organized crime, that his extreme criminalization policies created a niche for international criminals to fill. The years 1930 through 1962 in which he headed the FBN were not normal years, but rather consisted of three great crises in a row: the Depression, World War II, and the beginning of the Cold War—all three of which encouraged greater activism on the part of the federal government. They were also the years in which blacks migrated from the rural South to the urban North, where they were often tempted to ease their culture shock with narcotics that were expensive (as they had not been for his neighbor's wife when Anslinger was twelve and morphine was legal), precisely because his Bureau's enforcement drove the drugs underground. Anslinger has yet to receive the benefit of a well-balanced biography. It is hoped that an objective setting of his life against his times may one day be completed, thanks in part to the thirteen boxes of his papers which he left to Pennsylvania State University (McWilliams, 1990).

(SEE ALSO: *Methadone Maintenance Programs; Treatment, History of, in the United States; U.S. Government Agencies*)

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ANTABUSE *See* Disulfiram

ANTAGONIST An antagonist is a drug that binds to a RECEPTOR (i.e., it has affinity for the receptor binding site) but does not activate the

receptor to produce a biological response (i.e., it possesses no intrinsic activity). Antagonists are also called receptor "blockers" because they block the effect of AGONISTS. The pharmacological effects of an antagonist therefore result in preventing agonists (e.g., drugs, hormones, neurotransmitters) from binding to and activating the receptor. A competitive antagonist competes with an agonist for binding to the receptor. As the concentration of antagonist is increased, the binding of the agonist is progressively inhibited, resulting in a decrease in the physiological response. High antagonist concentrations can completely inhibit the response. This inhibition can be reversed, however, by increasing the concentration of the agonist, since the agonist and antagonist compete for binding to the receptor. A competitive antagonist, therefore, shifts the dose-response relationship for the agonist to the right, so that an increased concentration of the agonist in the presence of a competitive antagonist is required to produce the same biological response observed in the absence of the antagonist.

A second type of receptor antagonist is an irreversible antagonist. In this case, the binding of the antagonist to the receptor (its affinity) may be so strong that the receptor is unavailable for binding by the agonist. Other irreversible antagonists actually form chemical bonds (e.g., covalent bonds) with the receptor. In either case, if the concentration of the irreversible antagonist is high enough, the number of receptors remaining that are available for agonist binding may be so low that a maximum biological response cannot be achieved even in the presence of high concentrations of the agonist.

(SEE ALSO: *Naloxone; Naltrexone*)

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ANTIDEPRESSANT Antidepressants are a diverse group of drugs that demonstrate a capacity to produce improvement in the symptoms of clinical depression, and they are used to treat the abnormal mood states that characterize depressive illnesses. The word *depression* is used commonly to describe a state of sadness; but health professionals use the term in a more restricted or defined manner to describe several psychiatric disorders characterized by abnormal moods. One of these is bipolar disorder, in which periods of depression (marked by dejection, lack of energy, inactivity, and sadness) alternate with periods of manic behavior (marked by abnormally high energy levels and increased activity). Another is major depression, which is often a recurring problem characterized by severe and prolonged periods of depression without the manic swing. A third is dysthymia, a chronic mood state characterized by depression and irritability, which was once referred to as depressive neurosis. The signs and symptoms of depressive mood disorders may occur as part of other medical and psychiatric disorders (i.e., following stroke); as a result of endocrine disorders; or as a consequence of excessive drug use. Often these abnormal mood states may not meet established criteria for one of the major psychiatric mood disorders, but they may nevertheless respond to one of the antidepressant drugs.

Antidepressants can also be useful in a number of medical and psychiatric disorders where depression is not the major feature. For example, some categories of antidepressants can be used to treat anxiety and panic disorders, and they are often useful as adjunctive medications for chronic pain. Antidepressant drugs are not generally helpful for short-term depressed moods that are part of everyday life or for the normal period of grief that follows the loss of a loved one.

New categories of antidepressants are being continuously developed and tested. There are now at least five categories in use. These include tricyclic antidepressants, monoamine oxidase (MAO) inhibitors, lithium, nontricyclic antidepressants, and serotonin-reuptake inhibitors (SSRIs). The chemical structures of some of these are shown below.

The tricyclic antidepressants, which have been used for many years in the treatment of depression, include such compounds as imipramine (Tofranil), nortriptyline (Aventyl), and desipramine (Norpramin). In addition to being used to treat

depression, imipramine is sometimes used to treat alcoholism and cocaine withdrawal. Desipramine is also sometimes used to treat depression associated with cocaine withdrawal. In terms of dosage, most of the tricyclics can be given in a single dose at bedtime. The tricyclics as a group, however, have two major drawbacks. First, the patient must take a specific tricyclic for a period of 2 to 4 weeks before signs of clinical effectiveness occur. Second, the tricyclics have a relatively narrow margin of safety, which means that it is easier for a depressed patient to take an overdose. As a rule, physicians are cautious about prescribing tricyclic antidepressants if the patient appears to be at risk for suicide.

The monoamine oxidase (MAO) inhibitors are generally used as second-line drugs for depressed patients who do not respond to tricyclics, because they require certain dietary restrictions (patients are not allowed liver, aged meats, most cheeses, red wine, soy sauce, etc.) The MAO inhibitors are, however, first-choice drugs for treatment of panic disorder and of depression in the elderly. They include phenelzine sulfate (Nardil), isocarboxazid (Marplan), and tranylcypromine sulfate (Parnate). These antidepressants may be given in either the morning or the evening, depending on their effect on the patient's sleep.

Although lithium (Eskalith, Lithonate) is useful in treating manic states and in preventing depression in bipolar disorders, it is not generally used for other types of depression. Lithium may have serious side effects and may be toxic at high dosages. Exposure to lithium in early pregnancy is associated with an increased frequency of birth defects, and the long-term use of lithium damages kidney function. It also seems to have no significant value in treating cocaine dependence or alcoholism.

The serotonin reuptake inhibitors (SSRIs) are the newest category of antidepressant medications. They have become the most widely used drugs for depression; fluoxetine (Prozac) has been the best-selling antidepressant since the mid-1990s. Other SSRIs include paroxetine (Paxil) and sertraline (Zoloft). A fourth drug, bupropion (Wellbutrin), is not an SSRI but is often grouped with them because it is a newer antidepressant. The SSRIs have several advantages: They can often nip mild depression "in the bud" before it develops into a major depressive episode. They can also be used to treat bulimia, obesity, and obsessive-compulsive disorder as well as depression. Since insomnia is a common side effect of SSRIs, they are usually given as a single dose in the morning. The SSRIs also have several disadvantages, including a long response time (patients may need to wait 4 weeks to see any improvement); the same failure rate as the older tricyclics (20–40percent of patients); side effects that include sexual dysfunction; and high cost ($2–3 per tablet).

When a patient does not respond to a specific antidepressant after a trial of 2 to 4 weeks, the physician may prescribe another medication. If the new drug is from the same group as the first antidepressant, the physician can rapidly decrease the dosage of the first drug while increasing the dosage of the second. If, however, the new antidepressant is from a different category, a "washout time" must be allowed in order to prevent drug interactions. A washout period of 2 to 3 weeks is necessary when the patient is switched from an MAO inhibitor to a tricyclic; a period of 4 to 5 weeks is necessary when switching from an SSRI to an MAO inhibitor.

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ANTIDOTE A medication or treatment that counteracts a poison or its effects. An antidote may work by reducing or blocking the absorption of a poison from the stomach. It might counteract its effects directly, as in taking something to neutralize an acid. Or an antidote might work by blocking a poison at its receptor site. For example, a medication called naloxone will block opiates such as heroin at its receptors and prevent deaths that occur because of heroin overdose. In a sense,

drug ANTAGONISTS can all be antidotes under some circumstances, but not all antidotes are drug antagonists.

Many cities have a telephone "poison hot line," where information on antidotes is given. In case of drug overdose or poisoning, it is advisable to call for expert medical help immediately.

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ANTIPSYCHOTIC Any of a group of drugs, also termed *neuroleptics*, used medicinally in the therapy of schizophrenia, organic psychoses, the manic phase of manic-depressive illness, and other acute psychotic illnesses. The prototype antipsychotics are the phenothiazines, such as chlorpromazine (Thorazine), and the butyrophenones such as haloperidol (Haldol). The antipsychotics are tricyclic compounds, with chemical substitution at R_1 and R_2, which determine the selectivity and potency of the neuroleptic.

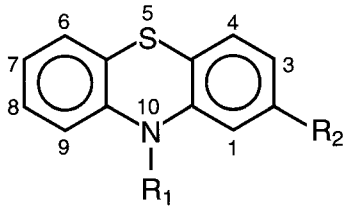

Figure 1
Antipsychotics

The "positive" symptoms of psychotic disorders, such as hallucinations, can often be effectively treated with antipsychotics; the "negative" symptoms, such as withdrawal, are less effectively managed by these drugs. Most of these drugs also have effects on movement, and a good response to the drugs' antipsychotic effects must often be balanced against motor side effects.

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ANTI-SALOON LEAGUE *See* Temperance Movement; Women's Christian Temperance Movement

ANTISOCIAL PERSONALITY Antisocial personality disorder (ASP) is particularly germane to alcohol and drug abuse because it co-occurs in a large proportion of those who abuse alcohol or drugs, and it confounds the diagnosis of, influences the course of, and is an independent risk factor for the development of alcohol or drug abuse disorder. Additionally, scientific evidence suggests that alcohol and drug abuse complicated by ASP is more heritable than is substance abuse without ASP.

In the latest diagnostic classification system DIAGNOSTIC AND STATISTICAL MANUAL-4th edition (DSM-IV), ASP is defined as a disorder that begins in childhood or early adolescence and continues into adulthood; it is characterized by a general disregard for and violation of the rights of others. At least three of the following behaviors must have occurred in any twelve-month period of time *before the age of 15*, *with two before age 13*: running away from home overnight twice, staying out late at night despite parental rules to the contrary (before age 13), truancy (beginning before age 13), initiating physical fights, using weapons in fights, cruelty to animals and to people, vandalism, forcing someone into sexual activity, arson, frequent lying to obtain favors or goods, frequently bullying, breaking into someone's house or car, and stealing from others (either passively like shoplifting, or aggressively, like mugging). In addition, three of the following behaviors must have occurred *since the age of 15*: consistent irresponsibility (e.g., inability to sustain consistent work behavior or to honor financial obligations), failure to conform to social norms by repeatedly engaging in arrestable behaviors, irritability and aggressiveness, deceitfulness (e.g.,

frequent lying or conning of others), reckless behaviors indicating disregard for safety of oneself or of others, impulsivity or failure to plan ahead, and lack of remorse for hurtful or manipulative behaviors.

A large percentage of alcohol and drug abusers meet criteria for ASP. For example, in one multisite study of 20,000 community respondents, 15 percent of the alcoholic participants compared to 2.6 percent of the population as a whole, met criteria for ASP. Comparable data from clinical samples indicate that from 16 percent to 49 percent of treated alcoholics met criteria for ASP.

A substantial proportion of those with ASP also abuse alcohol or drugs—three times as many men with a diagnosis of ASP as without abuse alcohol and five times as many abuse drugs. For females, the association is even stronger—twelve times as many women with ASP as without abuse alcohol and thirteen times as many abuse drugs. The strong association between ASP and alcohol or drug abuse may actually be caused by antisocial behaviors that occur when under the influence of alcohol or drugs judgment is impaired. It is possible, however, to distinguish *primary* abusers, those for whom the antisocial behaviors are a result of their substance use, from *secondary* abusers, for whom substance abuse is just one manifestation of a wide spectrum of antisocial behaviors. This differentiation is particularly important in understanding the genetic transmission of disease, where genetic factors responsible for the co-morbid state might be transmitted separately from those that cause substance abuse.

The course of alcohol or drug abuse is affected by ASP. Alcoholics with ASP have a more chronic and more severe course, an earlier onset of alcohol symptoms (for example, average age of onset of 20 compared to nearly 30 for those without ASP), as well as a significantly longer history of problem drinking. Furthermore, evidence is mounting that ASP alcoholics have poorer response to treatment—relapsing much earlier than alcoholics without ASP—and they may respond only to certain therapies.

Antisocial behavior problems in childhood have been identified as an independent risk factor in the development of alcoholism. One of the first studies to document this was carried out by Robins (1962) in a follow-up study of child-guidance clinic attendees; a marked excess of alcoholism was observed among those with antisocial behavior in childhood. This finding has subsequently been replicated in numerous other studies.

Data from several studies indicate that alcoholism complicated by ASP or ASP-like behaviors may be more heritable than non-ASP alcoholism. Evidence from a Swedish adoption study indicated the adopted-out sons of fathers with ASP-like alcoholism had a risk of alcoholism nine times that of adopted-out sons of other fathers.

The causes of ASP are not known, but data are accumulating from neurochemical studies that provide some clues. Neuropharmacological studies have established associations of aggressive, impulsive, hostile, and low socialization behaviors with low SEROTONIN levels. Another neurotransmitter, DOPAMINE, is linked to novelty-seeking behavior. The interactions among neurotransmitters have led researchers to postulate an association of multiple neurotransmitter dysfunctions, with a loss of impulse control and an increased appetite for novel experiences.

SUMMARY

ASP is a common concomitant of alcohol and drug abuse that affects their course and treatment; it may represent a highly heritable subtype of such abuse. Although its etiology is unknown, evidence from neuropharmacology studies has provided some leads. As with many personality disorders, there is no known treatment for ASP. It is an important disorder to consider in alcohol and drug abuse.

(SEE ALSO: *Addictive Personality; Childhood Behavior and Later Drug Use; Conduct Disorder and Drug Use*)

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ANXIETY Anxiety refers to an unpleasant emotional state, a response to anticipated threat or to specific psychiatric disorders. In anxiety, the anticipated threat is often imagined. Anxiety consists of physiological and psychological features. The physiological symptoms can include breathing difficulties (hyperventilation, shortness of breath), palpitations, sweating, light-headedness, diarrhea, trembling, frequent urination, and numbness and tingling sensations. The anxious person is usually hypervigilant and startles easily. The subjective psychological experience of anxiety is characterized by feelings of apprehension or fear of losing control, depersonalization and derealization, and difficulties in concentration. Strains around the performance of social roles (e.g., spouse, parent, wage earner) and certain life situations (e.g., separating from parents when starting school or leaving home, illness) can generate anxiety symptoms. Other factors can contribute to the etiology of anxiety, such as use of alcohol, caffeine and other stimulant drugs (e.g., amphetamine), a family history of anxiety symptoms, or a biological predisposition. In certain cases, recurrent anxiety symptoms will lead an individual to avoid certain situations, places, or things (phobias). In many cases, an anxious emotional state can motivate positive coping behaviors (e.g., anxiety that leads to studying for an exam). When the anxiety becomes excessive and impairs functioning, it can lead to the development of psychiatric illness. Individuals differ in their predisposition to anxiety.

Different constellations of anxious mood, physical symptoms, thoughts, and behaviors, when maladaptive, constitute various anxiety disorders. Panic disorder is characterized by brief, recurrent, anxiety attacks during which individuals fear death or losing their mind and experience intense physical symptoms. People with obsessive compulsive disorder experience persistent thoughts that they perceive as being senseless and distressing (obsessions) and that they attempt to neutralize by performing repetitive, stereotyped behaviors (compulsions). The essential feature of phobic disorders (e.g., agoraphobia, social phobia, simple phobia) is a persistent fear of one or more situations or objects that leads the individual to either avoid the situations or objects or endure exposure to them with great anxiety. Generalized anxiety disorder is diagnosed in individuals who persistently and excessively worry about several of their life circumstances and experience motor tension and physiologic arousal. Anxiety disorders are the psychiatric illness most frequently found in the general population.

Anxiety states can result from underlying medical conditions, and therefore these conditions should always be looked for when evaluating problematic anxiety. When anxiety develops into a psychiatric illness, various forms of treatment are available to reduce it. The choice of treatment often depends on the specific disorder. Medications may be used, including anxiolytics (e.g., BENZODIAZE-

PINES, buspirone) and ANTIDEPRESSANTS (e.g., imipramine, fluoxetine). Psychotherapies offered generally consist of cognitive-behavioral interventions (e.g., exposure therapy), but they can include psychotherapy of a supportive nature or more psychodynamically oriented approaches. Some people with severe anxiety may turn to alcohol or nonprescribed sedative-hypnotics for symptom relief, and this in turn may exacerbate the underlying condition.

(SEE ALSO: *Causes of Substance Abuse: Psychological (Psychoanalytic) Perspective: Prescription Drug Abuse*)

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APHRODISIAC An aphrodisiac is a substance that can be administered topically, internally, by injection, or by inhalation to stimulate sexual arousal or to enhance sexual performance. The term is based on Aphrodite, the ancient Greek goddess of love and beauty, and it came into the English language during the early 1800s. Although no solid scientific evidence exists for any substances that have selective effect on sexual function, many foods and food combinations have a long-standing reputation as aphrodisiacs—such as oysters, caviar, champagne, and truffles (a subterranean fungus uprooted by pigs in the oak forests of France).

Alcoholic drinks have also been considered to be an aphrodisiac, since sexual behavior often occurs after "cocktails," during or after parties, or during periods of alcohol intoxication—but only if not too much alcohol has been consumed. Objective measurements have demonstrated that ALCOHOL (a depressant) actually decreases sexual responsiveness in both men and women. This paradoxical effect was best expressed about 1605 by William Shakespeare in *Macbeth*, act 2, scene 3:

> MACDUFF:
> What three things does drink especially provoke?
> PORTER:
> Marry, sir, nose-painting, sleep, and urine. Lechery, sir, it provokes, and unprovokes; it provokes the desire, but it takes away the performance. . . .

Since the 1980s, COCAINE has gained popularity as a potential aphrodisiac, since its use purportedly enhances the sexual experience; MARIJUANA and AMYL NITRITE have had this reputation, in general, since the 1960s. Nevertheless, chronic cocaine users, like chronic heroin users, often report a loss of sexual interest and capability; therefore no rationale exists for the use of alcohol or other drugs as sexual stimulants. Quite the contrary, the use of these substances can lead to a loss of sexual desire and excitement and the development of a physical and/or psychological dependence.

A prescription drug—yohimbine, derived from the African yohimbe tree—seems to help cure some men of impotence. The data suggest that it may work as a placebo (psychologically), but urologists prescribe it nonetheless in the hope that the patient can avoid more invasive treatments. The treatment takes three to six months before there is an effect, and the natural form (available in health-food stores), is not the form used therapeutically.

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ARGOT When we talk about the argot of drug users, we mean their vocabulary or the collection of slang words and phrases used by one drug user to communicate with another—often to the exclusion of non-users. In some instances, argot extends to the intonation or pitch used to speak words and phrases.

Argot fascinates sociologists, anthropologists, and others who study human behavior because the use of argot is an example of learned behavior that helps identify members of one social group as opposed to another, Argot also marks off the boundaries of group membership: those of the in-group use argot with ease, while outsiders use argot ineptly or not at all.

In one study, we showed more than 2,000 drug users in Baltimore, Maryland, a photograph of someone injecting a drug into a vein, and we asked "What do you call this?" The vast majority, well over 50 percent, said that it was a picture of someone "firing up" and most of the others called it "shooting." Most other people in Baltimore speak of "firing up" their furnaces or "shooting" baskets on a basketball court, but they do not think about drug use when these terms are used.

The argot of drug users also varies from place to place and from time to time. A very small minority of the drug users in our study spoke of "mainlining" the drug, "spiking," or "oiling" when they saw the picture of a drug injection into a vein. These are older terms for the same injecting behavior now called "firing up" and "shooting" by younger drug users.

In some ways, argot reflects the social structure of groups: in-group members use the argot, while others do not. However, if argot was used to serve as a badge of membership in an in-group, then we might expect to hear argot in general conversations, no matter who is present. Nonetheless, sociologists studying the use of argot often have been surprised to find that argot is spoken mainly between group members but not as often when nonmembers are present.

Arguing from evidence of this type, some observers claim that argot serves more to convey and reinforce identities within groups than to distinguish one group from another. That is, the process of learning and using drug-related argot reinforces the process of joining in with others who use drugs. In some ways, this process might serve to supplement the reinforcing functions of drug use, making continued drug use more likely rather than less likely.

Many of the terms that originally were part of the argot of drug users have entered into more common usage. For example, "dope" has become a general purpose term, widely used in relation to many types of drug; many people know that "weed" or "reefer" refers to marijuana while "acid" is LSD (lysergic acid diethylamide). This encyclopedia includes a glossary of terms in the article SLANG AND JARGON, which lists many examples of argot that have become part of American slang usage.

(SEE ALSO: *Slang and Jargon*)

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ARRESTEE DRUG ABUSE MONITORING (ADAM) The Arrestee Drug Abuse Monitoring (ADAM) system evolved from the Drug Use Forecasting program (DUF). The National Institute of Justice developed this monitoring system in 1987 (Wish and Gropper, 1990). DUF was developed on the premise that a large fraction of arrestees not arrested for drug crimes (such as the possession or sale of drugs) were drug users, and that their drug use was linked to their criminality. Although many indicators were available to assess the level and nature of the nation's drug-abuse problem, little reliable data was available for arrestees, a particularly high-risk group in this regard. By 1989 the DUF program included twenty-four cities.

DUF protocol called for the collection of interview and urinalysis data from arrestees. In each city, a goal of interviewing 225 adult male and 100 adult female arrestees each quarter was set. Initially, no more than 20 percent of those included in

the sample could be charged with a drug crime. Male arrestees were eligible to participate if they had been charged with a misdemeanor or felony, but all female arrestees were eligible for participation. Urinalysis was conducted to test for the presence of a number of drugs, including cocaine, marijuana, opiates, PCP, amphetamines, methamphetamines, and designer drugs. Interviews were confidential and voluntary, and in most sites high rates of compliance from arrestees were obtained. A national laboratory analyzed all urine samples, and federal drug-testing standards for false positives and drug confirmation were applied to the testing procedure.

The DUF program had three specific goals: (1) to forecast drug epidemics, (2) to understand the nature of arrestee drug use, and (3) to provide drug treatment services for arrestees. Arrestees tested positive for drugs at very high levels: as many as 80 percent in some cities tested positive for at least one illegal drug. The drug of choice in the late 1980s through the mid-1990s was cocaine, followed by marijuana and opiates. There was a notable increase in the use of marijuana in the mid-1990s and by the late 1990s some cities found that methamphetamines were the drug of choice among arrestees. These cities were located in the western and southwestern United States. In general, women were more likely to test positive than men, and arrestees in their late twenties were the age group most likely to test positive. In addition, women charged with the crime of prostitution were the group most likely to test positive for drugs. It was no surprise that arrestees charged with drug crimes tested positive for illegal drugs at higher levels than those charged with other offenses. However, individuals arrested for property crimes such as burglary tested positive for drugs at levels comparable to those arrested for drug crimes. Individuals charged with violent crimes tested positive at the lowest levels, well below the average for the entire sample. There was considerable evidence of behaviors that placed arrestees at risk for HIV, including multiple sex partners, unprotected sex, and sharing of needles.

In 1997, the National Institute of Justice revamped and renamed DUF (NIJ, 1998) as Arrestee Drug Abuse Monitoring (ADAM). This change came in response to criticism about the sampling procedures used in DUF. In addition to a sampling plan that would yield findings which could be applied to the arrestee population in a given city, ADAM added twenty-six new cities to their programs, with a target goal of seventy-five.

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ARTS, DRUG USE AND *See* Creativity and Drugs

ASAM *See* American Society of Addiction Medicine

ASIA, DRUG USE IN Asia is the world's largest continent; India and China are its most populous countries. More than half the world's population lives in Asia. Thus we find considerable variation in drug use and drug problems there, not only among the various countries but also within them. Unfortunately, the available information about drug use in Asia is sketchy and fragmentary; few good studies have been published. Epidemiological data are almost completely absent. The rapid social, economic, cultural, and political transformations are adding to the complexity of drug-use patterns and associated drug-related problems in Asia and worldwide. This article provides a broad overview of the historical, cultural, political and economic forces that have shaped drug use in Asia. It should be kept in mind that current drug use in many parts of Asia is tied to drug-production. Myanmar and Afghanistan produce most of the world's illegal opium, while the Golden Triangle of Southeast Asia (Myanmar, Thailand, and Laos) find users contracting HIV infections from contaminated needles.

TEA

Most people know the tea plant *Camellia sinensis* in the brewed form of TEA. Tea has been part of Asian culture for thousands of years. Its use seems to have originated in southeastern China. It is mentioned in the very early Chinese medical literature. To a large extent, the medical benefits of tea can be ascribed to the chemical theophylline, which depending on its use can have either mildly calming or stimulating effects. The use of tea as a popular beverage and its production in large quantities has only been documented since the sixth century. The history of tea is also a history of international trade. Japan was one of the first countries to import tea from China, and tea became part of the Japanese culture. *Chanoyu* (the way of the tea) is a meditation ritual introduced in Japan by Zen Buddhist monks several hundred years ago, and elaborate tea ceremonies developed there. This tea ceremony is still taught and practiced in modern Japan.

Tea became the primary stimulant beverage not only in China and Japan but also in India, Malaysia, the Russian empire, and other Asian countries.

In the 1700s, tea was imported directly to Great Britain and to the British colonies by the East India Company. Even today, there are tea-preferring countries like Britain and coffee-preferring countries like Spain. The difference in preference goes back to the time of colonial trading: Those countries with tea-producing colonies drank tea, because it was cheaper than coffee; countries with coffee-producing colonies drank coffee, because for them it was cheaper than tea.

OPIUM

After tea, the drug most often associated with Asia is OPIUM. Opium is prepared from the opium poppy (*Papaver somniferum*), which grows well in the alkaline limestone soil of Turkey and Iran, east through Afghanistan and Pakistan to the northern mountainous areas of Myanmar (formerly Burma), Thailand, and Laos. The area forms a crescent, thus the name GOLDEN CRESCENT. The mountainous areas of Myanmar, Thailand, and Laos are known as the Golden Triangle.

Medical historians have been able to document that Arabian physicians of Asia Minor extracted raw opium from the seed pods of the poppy and used it to treat pain and diarrhea before A.D. 1000.

As part of a government-instituted program, students give samples of their urine to an official to test for narcotic drugs at Yothinburanat School in Bangkok, Thailand, March 4, 1998. (AP Photo/Sakchai Lalit)

Arabian traders began exporting opium to India and China about that time, and it also appeared in trade shipments to Europe. Although accurate documentation is scarce, some observers claim that opium use spread faster in precolonial and colonial India, than in China. A British royal commission investigated Indian opium use in 1895 and claimed that the people of India had not suffered detrimental effects from the taking of opium. The situation was different in China. The British traded Indian-grown opium for Chinese tea and porcelain. This led to an increasing supply of opium in China, associated with an increasing use of opium for recreational purposes. During the nineteenth century a raging epidemic of opium smoking in China led to a situation of great concern to the Chinese government. In an attempt to cut the supply of opium, the Chinese government tried to close its ports to British trade. This resulted in the Opium wars (1839–1842), but Britain won the war and the right to continue trading opium to China.

The different responses of India and China to the availability of opium might be explained, to some degree, by the way this drug was introduced to the population. In India, opium was introduced as a medicinal plant, to be taken by mouth and swallowed. In contrast, in China during the 1500s, Portuguese sailors had just introduced New World tobacco smoking as a form of a recreational drug use. Many Chinese, who had just picked up tobacco smoking, substituted opium for tobacco. Thus

opium was not only introduced as a nonmedicinal recreational drug, but it was also introduced in a different route of administration. Drugs inhaled through the lungs seem to produce faster and more severe dependence than those ingested through the gastrointestinal tract.

Effective government control of opium smoking in China did not become possible until late in the nineteenth and early twentieth centuries when Britain, the United States, and other world powers signed international agreements to help curb worldwide supply and distribution networks. They cooperated because opium abuse spread and started to affect these countries directly. In 1930, the League of Nations Commission of Inquiry into the Control of Opium Smoking in the Far East reported that opium use had not been prohibited in any Asian country except the Philippines. By 1950, this situation had changed dramatically. Many Asian countries placed high priority on narcotic-control policies. Harsh penalties, including the death penalty, had been reinstated for drug trafficking and possession of opium and derivatives, like MORPHINE and HEROIN.

Despite these government actions, opium and its derivatives are still used widely in regions where they are grown. In 1990, Myanmar, Thailand, and Laos supplied about 56 percent of the heroin consumed in the United States. By 1999, Latin America supplied most of the heroin to the United States, accounting for 82 percent of the heroin seized in the U.S. The Southeast Asian opium crop, which was on the rise in the early 1990s, suffered a sharp decline due to adverse weather in the later 1990s. China has moved to contain opium trafficking. In 1998, China began a "Drug Free Communities" program to eliminate drug trafficking and abuse as well as drug-related crime.

CANNABIS

Known in the United States mainly as the MARIJUANA plant, *Cannabis sativa* may first have been cultivated in Asia in a region just north of Afghanistan. From there it seems to have spread to China and India. It is mentioned in the early medical literature of China (e.g., in the *Shenmong bencao*) as well as in India (e.g., in the *Sushruta samhita*). Early nonmedical use has also been documented.

Cannabis use seems to have become popular especially in India and the Islamic countries. The many social rules associated with its use are evidence of its long-standing integration into Indian culture. Traditional Indian society was divided into hereditary classes or castes. The highest caste was to use white-flowered cannabis; the Kshatriya, the warriors, used the red-flowered plants; the farmers and traders, the Vaishya caste, were to use the yellow-flowered plant; and the Shudra, servant caste, used plants with dark flowers.

The earliest Indian medical text, *Sushruta samhita*, apparently dating from pre-Christian times, differentiated three major ways of preparing and administering *Cannabis*—BHANG, GANJA, and *charas. Bhang* was a sweet drink prepared from the leaves and flower shoots, which also might be brewed as a tea. *Ganja* was the dried flowers, which was smoked. *Charas* was a cake compound from the most resinous parts of the plant; this seems to have been the upper-class favorite. While *bhang*, *ganja*, and charas are still used in India today, the form of preparation may not be quite the same as the recipes in the *Sushruta samhita*.

BETEL NUT

In southern parts of Asia, mainly in India, Indonesia, Malaysia, southern China, and also in East Africa, many people chew BETEL NUT (*Areca catechu*). The nut is prepared by wrapping it in a betel pepper leaf (*Piper belle*) with a compound of lime (calcium hydroxide or calcium carbonate) and spices. Chewing this preparation produces mild stimulating effects. At the same time, the saliva becomes red and the mouth and teeth are stained red. Mouth cancer may result.

The ancient Greek traveler and historian Herodotus wrote about betel-nut chewing in 340 B.C. Although its use seems to be declining, an estimated 400 million persons are still dependent on this substance.

OTHER NATIVE DRUGS

Students interested in ETHNOPHARMACOLOGY and cultural practices associated with drug use will find many fascinating accounts in Asian history. One modern example involves the consumption of a drink called KAVA, which is prepared from the roots of *Piper methysticum*. In Polynesia, Mi-

cronesia, and Melanesia this drink is taken for recreational purposes, to calm and sedate the user.

There are ancient drug-taking practices connected to FLY AGARIC, a sometimes deadly mushroom (*Amanita mascaria*) found in several countries. One way to reduce the toxicity of this mushroom is to feed it to a reindeer and drink the reindeer urine, which contains intoxicating metabolites of the chemicals found in the mushroom.

STIMULANTS

Some Asian countries have suffered epidemics of drug use in connection with legally produced drug products. An especially widespread epidemic of AMPHETAMINE use started in Japan during World War II and continued into the 1950s. A second wave of amphetamine use was reported in the late 1970s. Recently an epidemic of METHAMPHETAMINE "(ice)" smoking spread across the Pacific into Hawaii and other American states after earlier micro-epidemics in Asia.

ALCOHOL

The account of drug use in Asia would be incomplete without mention of alcoholic beverages. At present, Asia is the continent with the lowest overall per-capita consumption of ALCOHOL. In many Asian countries, alcohol consumption is prohibited on religious grounds—because of the prohibitions of Islam: the Koran forbids its use. Nonetheless, even in the most conservative Islamic countries, there is some alcohol dependence. Saudi Arabia for example, has an ALCOHOLICS ANONYMOUS (AA) organization and a modern hospital for drug and alcohol treatment.

In addition to religious and social restrictions on alcohol consumption, there are some important biological factors known to be related to genetic variation within the Asian population. For example, many Asian people have the "flushing syndrome" in response to alcohol that is associated with their particular configuration of aldehyde dehydrogenase, an alcohol-metabolizing enzyme. One prominent sign is that their facial skin becomes flushed. Although this response might work to discourage alcohol use, and thus protect against alcohol dependence, many Asian people—especially men—are known to "drink through" the flushing response to become intoxicated. In fact, South Korean males suffer from the highest recorded prevalence rates of alcohol abuse and dependence: An estimated 44 percent of adult men have a history of currently active or former alcohol abuse and/or dependence. The reasons for this very high rate are a matter of speculation and should be a topic of intense study. As evidence of the considerable variation in alcohol problems in Asia, Taiwan has one of the lowest rates of alcohol abuse and dependence in the world for both adult men and women. This variation cannot be explained by differences in research methods, because the same methods have been used in surveys of Taiwan and South Korea. The difference must involve fundamental social and cultural differences, or fundamental biological differences in vulnerability to alcohol-related problems, or a combination.

Alcohol use is not a new phenomenon in Asia. The drinking of fermented beverages has been part of Asian cultures since antiquity, as documented in the early classical literature of China (in the *Shujing* and the *Liji*), India (in the *Susruta samhita*), and other countries. The *Susruta samhita* describes various stages of intoxication. In China, the fall of the Shang Dynasty in the eleventh century B.C. was attributed to excessive use of alcohol by the emperor and his followers. The same explanation was given for the fall of later dynasties. In China, different forms of alcohol have been fermented from various kinds of grain. In other parts of Asia, alcoholic beverages were based on a large variety of different substances, including rice in the case of Japanese sake; horse milk in the case of Kumys, an alcoholic beverage prepared by northern and central Asian nomads; and toddy-palm sap in the case of arrack prepared in southern India and Indonesia.

An early epidemic of drug use combining alcohol with a drug called *hanshi* can be traced in the ancient writings of the time of the fall and overthrow of the Chinese Han Dynasty—a time of rapid changes in society (second and third century A.D.). The use of *hanshi* was associated with an unconventional "bohemian" lifestyle, disregard of social norms, "disheveled hair," and "incorrect clothing." The *hanshi* users were reported to claim that the drug helped open their minds and clarify their thinking. Although reports of this early epidemic are sketchy, *hanshi* is mentioned in several later medieval texts, mainly in relation to remedies that

can be used to help treat its detrimental side effects. At present it is not clear which chemical compound was present in *hanshi*.

TOBACCO

Probably the most widespread twentieth-century epidemic in Asia is TOBACCO smoking. Today, in most Asian countries, local, international, and especially American tobacco manufacturers are marketing their products aggressively—in part because of declining demand in North America and in part because of the increasing economic strength of the Asian countries. One result has been an increase in the consumption of tobacco products since the 1960s, especially the smoking of cigarettes.

Tobacco became a part of Asian culture from the time it was imported by Europeans from their colonies in the Americas during the 1600s. The "hubbly-bubbly," or hookahs, of the Middle East and India were used for smoking tobacco. This was centuries before modern advertisement techniques were applied by the tobacco industry. But recently, tobacco-related diseases and deaths are becoming more prominent in the health statistics of Asia. This toll is connected directly to an increasing consumption of tobacco products. Part of the tobacco is imported from the United States and other international suppliers. Some observers noticed similarities to the situation in the nineteenth-century, when British traders aggressively fought to keep the lucrative opium trade from being interrupted. Some thus call for international agreements concerning tobacco trade, similar to those which helped curb the opium problem at the beginning of the twentieth century. International support seems to be needed to help these countries reduce tobacco-related problems.

THE FUTURE

As commerce between countries has increased, so has the traffic in drugs. For centuries Asia has had trading partners for its tea, opium, and *Cannabis*. In return it has received shipments of other goods, including pharmaceuticals. Sometimes these exchanges have been within Asia, as in the early introduction of opium into China by Arabian traders, and the later commerce in opium between colonial India and China. Now trading is done on a worldwide scale, whether it is the legal trade with tea or the illegal traffic of opium. Recently some countries in Asia have reported an increase in POLYDRUG use among their younger population.

Since the 1950s, a number of Asian countries have also experienced a growth of what might best be called "drug tourism." Travelers, mainly from the Western Hemisphere, have come to Asia to purchase and consume such drugs as opium, *Cannabis*, heroin, and magic mushrooms. For many, it has come as a surprise that Asian countries respond with harsh penalties, as did Singapore in 1994, when a man from the Netherlands was hanged for possessing a large amount of heroin. It must be kept in mind that a long history of harsh penalties and social sanctions against those who violate social conventions, including local drug regulations, are part of Asian heritage—as well as the seemingly exotic custom of drug use.

(SEE ALSO: *Source Countries for Illicit Drugs*)

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ASPIRIN *See* Pain: Drugs Used for

ASSERTIVENESS TRAINING *See* Cognitive Behavioral Therapy; Treatment; Treatment Types

ASSESSMENT OF SUBSTANCE ABUSE: DRUG ABUSE SCREENING TEST (DAST)

The assessment of drug use and related problems is important for both prevention and clinical care. Measures that are both reliable and valid provide tools for health education, for identifying problems (early if possible) in health care and community settings, and for evaluating the effectiveness of treatment. As well, this information is useful for matching individual needs and readiness for change with tailored interventions.

The Drug Abuse Screening Test (DAST) was designed to be used in a variety of settings to provide a quick index of drug-related problems. The DAST yields a *quantitative* index of the degree of consequences related to drug abuse. This instrument takes approximately 5 minutes to administer and may be given in questionnaire, interview, or computerized formats. The DAST provides a brief, self-report instrument for population screening, identifying drug problems in clinical settings and treatment evaluation.

DAST-20 and DAST-10 Versions. The DAST was modeled after the widely used Michigan Alcoholism Screening Test (Selzer, 1971). Measurement properties of the DAST were initially evaluated using a clinical sample of 256 drug-alcohol-abuse clients (Skinner, 1982). The 20-item DAST has excellent internal consistency reliability (alpha) at 0.95 for total sample and 0.86 for the drug-abuse sample. Good discrimination is evident among clients classified by their reason for seeking treatment. Most clients with alcohol-related problems scored 5 or below, whereas the majority of clients with drug problems scored 6 or above on the 20-item DAST. The DAST-10 correlates very highly ($r = 0.98$) with the longer DAST-20 and has high internal consistency reliability for a brief scale (0.92 for the total sample and 0.74 for the drug-abuse sample).

Subsequent research has evaluated the DAST with various populations and settings including psychiatric patients (Cocco & Carey, 1998; Maisto et al., 2000; Staley & El Guebaly, 1990), prison inmates (Peters et al., 2000), substance-abuse patients (Gavin et al., 1989), primary care (Maly, 1993), in the workplace (El-Bassel et al., 1997), and adapted for use with adolescents (Martino et al., 2000). Overall, these studies support the reliability and diagnostic validity of the DAST in diverse contexts.

Advantages.

1. The DAST is brief and inexpensive to administer. Versions are being developed in different languages (French and Spanish).
2. It provides a quantitative index of the extent of problems related to drug abuse. Thus, one may move beyond the identification of a drug problem and obtain a reliable estimate of the degree of problem severity.
3. The DAST has been evaluated and demonstrated excellent reliability and diagnostic validity in a variety of populations and settings.
4. Routine administration of the DAST would provide a convenient way of recording the extent of problems associated with drug abuse, ensuring that relevant questions are asked of all clients/patients.

TABLE 1
DAST Interpretation Guide

	DAST-10	*DAST-20*	*Action*	*ASAM**
None	0	0	Monitor	
Low	1–2	1–5	Brief counseling	Level I
Intermediate (likely meets DSM** criteria)	3–5	6–10	Outpatient (intensive)	Level I or II
Substantial	6–8	11–15	Intensive	Level II or III
Severe	9–10	16–20	Intensive	Level III or IV

* ASAM–American Society of Addiction Medicine Placement Criteria
** DSM-IV–American Psychiatric Association

5. The DAST can provide a reference standard for monitoring changes in the population over time, as well as for comparing individuals in different settings.

Limitations.

1. Since the content of the DAST items is obvious, individuals may fake results.
2. Since any given assessment approach provides an incomplete picture, there is a danger that DAST scores may be given too much emphasis. Because the DAST yields a numerical score, this score may be misinterpreted.

Administration, Scoring and Interpretation. The DAST may be administered in a questionnaire, interview, or computerized format. The questionnaire version allows the efficient assessment of large groups. The DAST should not be administered to individuals who are presently under the influence of drugs, or who are undergoing drug withdrawal. Under these conditions the reliability and validity of the DAST would be suspect. Respondents are instructed that "drug abuse" refers to (1) the use of prescribed or over-the-counter drugs in excess of the directions and (2) any nonmedical use of drugs. The various classes of drugs may include cannabis, (e.g., marijuana, hash), solvents or glue, tranquillizers (e.g., valium), barbiturates, cocaine, stimulants, hallucinogens (e.g., LSD), or narcotics (e.g., heroin). Remember that the questions do not refer to the use of alcoholic beverages.

The DAST total score is computed by summing all items that are endorsed in the direction of increased drug problems. Guidelines for interpreting DAST scores and recommended action are given in Table 1. A score of 3 or more on the DAST-10 and 6 or more on the Dast-20 indicates the likelihood of substance abuse or dependence (e.g., DSM IV, American Psychiatric Association). This diagnosis would need to be etablished by conducting a further diagnostic assessment.

Availability. Copies of the DAST may be obtained from H. Skinner, (E-mail: harvey.skinner @utoronto.ca), or from the Centre for Addiction and Mental Health, 33 Russell Street, Toronto, Ontario, Canada M5S 1A8, telephone: 1-800-463-6273 (http://www.camh.net). A computerized version of the DAST is included in the Computerized Lifestyle Assessment (Skinner, 1994) published by Multi-Health Systems, Toronto (http// www.mhs.com); call 1-800-268-6011 in Canada or 1-800-456-3003 in the United States.

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ASSESSMENT OF SUBSTANCE ABUSE: HIV RISK ASSESSMENT BATTERY (RAB)

The Risk Assessment Battery (RAB) is a self-administered questionnaire designed for use with substance-using populations. It was developed to provide a rapid (less than 15 minutes) and confi-

dential, non interview method of assessing both needle use practices and sexual activity associated with HIV transmission.

The forty-five questions of the RAB are simply worded and use discrete response categories. Respondents are asked to "check off" the answer that best describes their behavior. There are no open-ended questions, minimizing the need for writing skills. A brief set of instructions is included on the first page of the RAB. However, as with all self-administered questionnaires, it is particularly important to provide the respondent with a proper introduction and explanation of the form, its purpose, and how it is to be completed. A staff member should be available during administration of the test to screen for reading difficulties, answer questions as they arise, and ensure that the form is being filled out properly. Given the very sensitive nature of the information collected, it is also important that individuals administering the RAB address the issue of confidentiality. Although the more private approach of the self-administered questionnaire should reinforce the confidential nature of the assessment, it is very important that respondents understand the confidentiality of their responses will be protected.

There are two global sections within the RAB: 1) drug and alcohol use during the past 30 days, and 2) needle use and sexual behavior during the previous 6 months. Questions have been constructed to provide maximum coverage and sensitivity to potential risk behaviors within these categories. Since self-reports may be expected to provide underestimates of behaviors that are socially unaccepted, items have been assembled that assess a wider range of behaviors associated with HIV infection. Thus, questions ask not only about the behaviors directly responsible for viral transmission such as needle sharing and unprotected sexual activity, but also those associated with such activities (e.g., needle acquisition, shooting gallery attendance, exchange of money or drugs for sex). The inclusion of these items is intended to identify individuals at increased risk of HIV exposure even if transmission behaviors are not directly reported. However, endorsement of these "peripheral behaviors" does not prove that transmission behaviors have actually occurred. For example, an individual who indicates that he or she has visited a shooting gallery on numerous occasions during the assessment interval may not have shared a needle or had unprotected sex even though these behaviors are common in shooting galleries. Instead, these peripheral behaviors may be more readily reported by some respondents despite their reluctance to report primary transmission events such as sharing a syringe or unprotected sexual activity.

Scoring. Sixteen items from the RAB are used in the computation of three scores: a drug-risk score, a sex risk score, and a total score. These scores are calculated by adding responses to selected items. For individual questions, the values range from zero to a maximum of 4. Higher values for items reflect greater frequency of occurrence for the behavior. The eight-item drug-risk score has a range of 0 to 22. The range of the sex-risk score, comprised of nine items, is 0 to 18. This simple scoring system was designed to capture frequency of engaging in each of the reported risk behaviors. Scores for the various items are not differentially weighted. This scoring strategy serves to guard against underestimates of risk resulting from the tendency to under report participation in behaviors known to be most likely to transmit the AIDS virus.

As a self-administered questionnaire, the RAB offers an efficient tool for screening individuals who may be at risk for HIV infection. The RAB provides a measure of HIV-risk behaviors, which is broken down into subscales for drug risk and sex risk and combined to yield a measure of total risk. A number of studies conducted by the authors and others suggest that when properly administered, the RAB responses are equivalent to those collected via a personal interview. Test-retest reliability has also been found to be relatively high. Most importantly, the RAB has demonstrated discriminant validity in differentiating between respondents engaging in different drug-use patterns and predictive validity in identifying seroconverters on the basis of higher-risk scores.

As the AIDS epidemic enters its third decade, it has become increasingly important to have valid, reliable, and cost-effective tools to monitor behaviors associated with the transmission of HIV. It is no longer sufficient to direct prevention resources toward populations at risk in a "shotgun" approach to risk reduction. Such a strategy is costly and inefficient since many individuals within risk groups have instituted safer behaviors. Targeting risk-reduction interventions to specific segments of the population at risk and evaluating their efficacy are necessary components in a well-planned ap-

proach to HIV prevention. Measures of risk behavior, such as the RAB, are needed to target and evaluate interventions in a more precise manner.

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ASSESSMENT OF SUBSTANCE ABUSE: MAST *See* Appendix, Volume 4.

ASSESSMENT OF SUBSTANCE ABUSE: T-ACE Heavy maternal drinking is a major pregnancy risk and a significant public health problem. Fetal Alcohol Syndrome (FAS) was first reported as a recognizable clinical syndrome nearly thirty years ago. It is characterized by

1) prenatal and/or postnatal growth restriction
2) central nervous system (brain) abnormalities and
3) facial dysmorphology, i.e., an abnormal appearing face characterized by underdevelopment of the midface with small eyes, a short nose and a long simple (flat) philtrum, the area below the nose and above the upper lip.

As these children grow up, they are often mildly mentally retarded, with average IQs of about 70 and disabling behavioral abnormalities. In addition, there is a continuum of abnormalities among offspring exposed before birth to alcohol, but without the full syndrome—abnormalities that are much more common than full FAS. There are anatomic anomalies, called alcohol-related birth defects (ARBD) and alcohol-related neurobehavioral disorder (ARND), a set of behavioral abnormalities in offspring prenatally exposed to substantial levels of alcohol. Other adverse pregnancy outcomes related to maternal drinking during pregnancy include miscarriage and stillbirth.

A national goal to reduce the prevalence of FAS by one half by decreasing maternal drinking was set in Healthy People 2000. Unfortunately, the reported prevalence did not decrease through the 1990s, but, in fact, increased, possibly because of improved case finding. Regardless, it is likely that heavy drinking in pregnancy did not decrease, despite warning labels on all alcoholic beverages since 1989.

There is evidence that pregnant women are receptive to advice from their health care providers, particularly their physicians, to quit or at least cut down both drinking alcohol and smoking cigarettes. Given that generalized warnings, such as the warning label, have not proven effective, a more focused approach would seem reasonable—this would focus prevention efforts on women who drink or are likely to drink enough during pregnancy to damage their offspring. Such drinking has been labeled "risk drinking."

The precise level of drinking that might damage the embryo/fetus is unknown, but is probably variable because of differing susceptibility and differing exposures depending on exactly which adverse effect is considered and when during pregnancy the exposure occurs (critical period). Solid estimates of risk drinking have decreased over the years, as better interviewing and statistical techniques have become available. It is now reasonable to use a figure of about seven drinks per week, typically massed on one or two days, but averaging about one drink per day or 0.5 ounces of absolute alcohol per day. This is the amount of absolute alcohol in one can of beer, one glass of wine or one mixed drink of standard size. This amount of alcohol intake, while unlikely to pose any health risk to the mother, is enough to adversely affect the embryo/fetus. There is not convincing evidence of clinically important effects on the offspring from an occasional drink during pregnancy.

There are, as of yet, no laboratory tests, i.e., biological markers, which will reliably identify risk drinking women. The only way to identify them is

TABLE 1
The T-ACE Questionnaire

	Question	*+Answer*	*Score*
T	How many drinks can you hold (TOLERANCE)?	> = 6	2
A	Have people ANNOYED you by criticizing your drinking?	Yes	1
C	Have you felt you ought to CUT DOWN on your drinking?	Yes	1
E	Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (EYE-OPENER)?	Yes	1

The Tolerance Question is positive if the patient admits that she can hold, i.e., not get sick or lose consciousness, at least a sixpack of beer, a bottle of wine, or six standard drinks. As in the old song, (T) for two and two for (T) and, as in blackjack, each ACE is worth one. A total score of two or more is positive.

to obtain an appropriate history of drinking, but this is complicated by *DENIAL*—the woman doesn't want to admit drinking to herself or to her doctor. Further, time is distinctly limited during prenatal visits and there are many problems to identify and address. Thus, a brief, simple questionnaire was needed. Most brief questionnaires, such as the CAGE, were developed and tested almost entirely in male populations, and do not function well for reproductive age women.

The T-ACE questions were developed specifically as a screening test for risk drinking. They have been tested and validated over the last decade in women of multiple ethnicities, including white, African American and Native American, and in a range of socioeconomic statuses and geographic locations. The original questionnaire included the question, "How many drinks does it take to make you feel *high*?" as the (T)olerance question. An answer of greater than two standard drinks was considered positive. Several studies have now shown that substituting the *hold* question, included in Table 1, instead of the *high* question, gives better results, improving the sensitivity of the T-ACE questions.

T-ACE is a screening test, so it was designed to pick up as high a proportion of risk drinkers as possible. This version picks up about nine in ten women who drink enough in pregnancy potentially to damage their baby. If the score is less than two, i.e., the T-ACE is negative, it will correctly identify about seven in ten women who are not risk drinkers. It has a substantial false positive rate, i.e., warning the clinician, though the patient is not, in fact, a risk drinker. It has been speculated though that any woman who scores positive might, in fact, be at risk to drink too much during pregnancy and should be counseled.

A final point—*screening for risk drinking is not enough*. At the minimum a brief intervention to support the patient in becoming abstinent during pregnancy or at the minimum cutting way down is warranted, as is close follow-up. If alcohol abuse or dependence is present, consultation or referral may be warranted.

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ASSET FORFEITURE Asset forfeiture is the involuntary relinquishment of money or property without compensation as a consequence of a commission of a crime. Forfeiture laws authorize prosecutors to file civil lawsuits asking a court for permission to take property from a criminal defendant that was either used in the crime or was the fruit of a criminal act. Since the 1970s, federal asset forfeiture laws have been used against drug

dealers. By 2000, however, there were many in Congress and the legal community who urged reform of these forfeiture laws, as they have been often resulted in harsh and unfair outcomes for innocent third parties.

In 1970, Congress enacted the Comprehensive Drug Abuse Prevention and Control Act, also known as the Forfeiture Act. The Forfeiture Act authorized federal prosecutors to bring civil forfeiture actions against certain properties owned by persons convicted of federal drug crimes. The act was not used much because it limited forfeiture to the property of persons convicted of participating in continuing criminal enterprises. In 1978, Congress amended the law to allow forfeiture of anything of value used or intended to be used by a person to purchase illegal drugs. This expanded the act to allow the forfeiture of all proceeds and property traceable to the purchase of illegal drugs. The amended law authorized the federal government to proceed in rem against property. In rem forfeiture proceedings are actions taken against the property, not the owner of the property. This allows the government to remove property from persons suspected of a crime without ever charging them with a crime.

Congress amended the Forfeiture Act again in 1984 as part of the Comprehensive Crime Control Act. The amendment authorized the federal government to pursue in rem forfeitures of land and buildings. Federal authorities may seize any real property purchased, used, or intended to be used to facilitate narcotics trafficking. Although Congress appears to have intended the law to apply only to drug manufacturing or storage facilities, federal courts have interpreted the law to allow the seizure of any real property, including fraternity houses, hotels, ranches, and private residences. In addition, courts have allowed forfeitures regardless of whether the property was used to store or manufacture drugs.

The process of seizing property under the Forfeiture Act is straightforward. Forfeiture begins with the constructive or actual seizure of property after a warrant has been issued by a federal district court. This warrant must be based on the reasonable belief that the property was used in a crime subject to forfeiture, but this belief can be based on hearsay and circumstantial evidence. After the property is seized, the court holds it until the case is resolved.

In a civil forfeiture proceeding, the government must prove that the property is subject to forfeiture because there is a substantial connection between the property and the crime. If the defendant fails to rebut this proof with sufficient evidence, the government is allowed to keep the property. At the trial, the government's standard of proof is by a preponderance of the evidence, a lesser burden of proof than the criminal standard of a reasonable doubt.

The Forfeiture Act permits law enforcement agencies to receive a part of the proceeds from property forfeiture. Prior to the 1984 amendments, all revenue derived from a federal asset forfeiture was deposited into the U.S. Treasury general fund. The 1984 law allowed federal law enforcement agencies to keep all proceeds from confiscated property and to use the proceeds to support asset-seizure programs. State and local law enforcement agencies that turned over their seizure cases to federal authorities received up to 80 percent of the profit after the property had been sold. Many legal scholars criticized this feature of the law, arguing that it detracts from the traditional police function of fighting crime and created incentives for police to pursue forfeitures that lacked probable cause. Proponents of this budgetary scheme argue that drug activity is the source of much violent crime and that the proceeds benefit community programs and increase the capacity to fight violent crime.

Most states also have forfeiture laws upon conviction of certain crimes. These laws often mandate forfeiture of prohibited drugs, property used to contain, protect, or secure prohibited drugs, firearms, and vehicles. In contrast to the federal law, many states require that profits from the sale of forfeited property be deposited in the state's general treasury fund.

Defendants have employed several defenses to forfeiture and some have proved successful. If notice and a hearing before a court do not precede the initial seizure, a defendant may argue that forfeiture violates the Due Process Clause of the Fifth and Fourteenth Amendments. If a forfeiture is disproportionate to the offense that gave rise to it, it may be found to violate the Eighth Amendment's Excessive Fines Clause.

Congress has also responded to criticism by enacting an "innocent owner" defense in civil drug forfeiture cases. These are cases where forfeiture is sought without prosecution of the owner. A defen-

dant in a civil forfeiture case may invoke this defense if the property was connected with illegal drugs without the owner's knowledge or consent. For example, if the owner of an automobile innocently allows another person to borrow the car and that person commits a drug offense in the car, the owner can offer this defense and retain the car.

As state and federal prosecutors intensified their use of asset forfeiture laws, public grew. By the early 1990s, the federal government was prosecuting only 20 percent of the individuals from whom they seized property through forfeiture. According to Department of Justice statistics, over 28,000 properties were seized in 1996, with a combined value of $1.264 billion. Critics have argued that the government routinely violates the Fifth Amendment's ban against taking property without due process of law, largely because it sees forfeiture as an easy way to collect funds. Supporters have countered that forfeiture has helped in the war on drugs by stripping criminals of their resources.

Congress finally responded by passing the Civil Asset Forfeiture Reform Act of 2000. It requires federal prosecutors to show a substantial connection between the property and the crime. In addition, it allows the property to be released by the district court pending final disposition of the case when the owner can demonstrate that possession by the government causes a hardship to the owner. Moreover, the law allows owners of property to sue the government for any damage to the property if the victim of the seizure prevails in a civil forfeiture action.

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ASSOCIATION FOR MEDICAL EDUCATION AND RESEARCH IN SUBSTANCE ABUSE (AMERSA) The Association for Medical Education and Research in Substance Abuse (AMERSA) is a national organization of more than 300 medical and allied faculty, which was founded in 1976 for the promotion of education and research in the field of substance abuse. The organization was derived from an informal coalition of U.S. Federal Career Teachers in alcoholism and drug abuse; these career teachers, one on the faculty of each of fifty-five medical schools, were funded by the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA) to promote enhanced teaching at their respective medical campuses. The Career Teachers Program, established in 1972, was regarded as a highly successful vehicle for highlighting an issue of considerable importance in the medical curriculum. As the program wound down (it came to an end in 1981), the participants felt it important to secure the continuation of their mission and established AMERSA as a national membership organization open to all medical faculty and faculty in allied health programs.

In the year of its establishment, AMERSA held its first national meeting, which was followed by meetings of increasing attendance in each succeeding year. The national meetings have been the focus of federal participation in teaching programs and have focused on curriculum techniques and new research findings.

AMERSA established a quarterly publication, *Substance Abuse*, in 1979, presenting educational and research findings; it serves as a vehicle for broadening the base of teaching in the members' fields. In addition, a variety of curricula were established by members, with coordination through the AMERSA national headquarters (located in Providence, Rhode Island) and augmented by the Center for Medical Fellowships in Alcoholism and Drug Abuse, located at New York University.

Full membership is available for all persons holding faculty appointments in health-professional schools and/or to those involved in substance abuse education or research. Membership benefits include a free subscription to *Substance Abuse*; reduced rates at the annual conference; and a national voice that supports academic programs in universities, professional schools, and organizations that promote substance abuse education and research.

The organization's members work in a variety of ways to effect their educational ends. Much effort is invested in developing curriculum and curriculum outlines for courses directed at a variety of disciplines and various educational levels. In addition,

most members work actively within their respective departments to develop subspecialty expertise—as in psychiatry and internal medicine. Efforts are also directed at schoolwide initiatives—as with programs organized through the deans of medical schools.

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ATHLETES AND DRUGS *See* Anabolic Steroids

ATROPINE *See* Jimsonweed; Scopolamine and Atropine

ATTENTION DEFICIT DISORDER Children and adolescents who "act out" their feelings, frustrations, and emotional conflicts are said to exhibit externalizing behavior. Within the framework of the American Psychiatric Association's DIAGNOSTIC AND STATISTICAL MANUAL, 3rd edition, revised (DSM-III-R), once a certain level of severity is demonstrated, these youth qualify for the umbrella diagnosis of *disruptive behavior disorder*. Three disorders are encompassed within this general diagnostic category: (1) attention deficit/hyperactivity disorder (ADHD); (2) CONDUCT DISORDER (CD); and (3) oppositional defiant disorder.

It should be noted that in recent years some controversy has developed concerning diagnostic techniques. It has been suggested that with the introduction of the American Psychiatric Association's DIAGNOSTIC AND STATISTICAL MANUAL, fourth edition (DSM-IV) in 1994, accurately diagnosing ADHD in adults has become problematic "because of the vague nature of the criteria" in DSM-IV (Higgins, 1999). Comparatively, in more recent studies, a different approach for administrating the Diagnostic Interview Schedule for Children (DISC), based on communication principles, has marked a shift from structured interviewing to a more open communication model. This communication model is defined by three factors: (1) a schematic representation of the areas to be covered; (2) a common language for the categories, diagnosis, and criteria; and (3) the respondent was permitted to chose the order of the diagnostic areas to be covered. This new method is a notable departure from the structured diagnostic interviews that have been implemented in the past. Significant developments have been made in these structured diagnostic interviews over more than two decades, but along with these improvements have come problems. This alternative diagnostic method may have potential for strengthening the authority of child psychiatric diagnosis (Edelbrock, Crnic & Bohnert, 1999).

Children who qualify for a diagnosis of ADHD often, but not invariably, meet criteria also for a diagnosis of conduct disorder or oppositional defiant disorder. In addition, attention deficit disorder does not always occur in conjunction with behavioral hyperactivity. These latter individuals are assigned the diagnosis of *undifferential attention deficit disorder*.

Characteristically, youth with ADHD demonstrate an excessively high level of behavioral activity across a wide variety of situations. The behavioral disturbance is most apparent, however, where the appropriate level of behavioral activity should be low, such as in a classroom where focused task-oriented cognitive demands are placed on the youngster. In naturalistic settings, ADHD children, although not overtly hyperactive, typically are impulsive, emotionally labile, restless, and distractable. They often act in a socially intrusive and inappropriate manner such that they are considered to be immature. Consequent to poor foresight and poor impulse control, combined with a high behavior level, ADHD children frequently get into social difficulties with adults and peers, which subsequently lead to rejection. Physical injury is common in ADHD because of poor self-control (e.g., dashing across the street without looking).

Because ADHD symptomatology is often present in children with poor coordination, reading and learning problems, and other neurodevelopmental disturbances, in the past labels such as "minimal brain damage" and "minimal brain dysfunction" were assigned, although no neurologic pathology or injury was detectable. The terms "hyperactivity" and "hyperkinesis" were subsequently introduced; however, these labels emphasize the motor aspects of the disorder. The current diagnostic label—attention deficit disorder—circumvents these problems by focusing on the core etiological determinant for the multifaceted expression of cognitive, behavioral, and emotional disturbances. For this diagnosis to be assigned, the disorder must first be

expressed before age 7, have at least a 6-month duration, and not be the consequence of a pervasive developmental disorder (American Psychiatric Association, 1987).

EPIDEMIOLOGY

Approximately 10 percent of boys and 3 percent of girls in the general population qualify for a diagnosis of ADHD. The symptom presentation is different between the genders, with girls being somewhat older than boys at the time of first diagnosis. Girls manifest more mood changes, fears, and social withdrawal than boys but less aggressivity and impulsivity. Among children receiving psychiatric treatment, ADHD is estimated to be present in 40 to 70 percent of inpatient cases and 30 to 50 percent of outpatient cases.

GENETIC ETIOLOGY

Behavior-activity level is a heritable trait of temperament characterizing the human species. Individuals who are at the high end of this trait, compared to the average, are behaviorally highly active or hyperactive. Thus, although not necessarily implying pathology or a disorder, but rather normal variation in behavioral disposition, high-end active individuals demonstrate a rapid behavioral tempo and greater vigor and forcefulness than the average. For clinicians, it is important to distinguish between individuals with behavior-activity level as a temperament trait and extreme cases that comprise psychological and psychiatric disorder.

For many individuals, extremely high manifestations of behavior-activity level has a genetic basis. ADHD aggregates in families and twin studies show high concordance rates for this disorder. The neurobiological mechanisms underlying ADHD is an active topic of research. As of the mid-1990s, we assume that the neurochemistry in such people is likely to be disturbed; however, a candidate neurotransmitter system has yet to be identified. Most likely, multiple neurochemical systems are involved. Neuroimaging studies have revealed lowered brain metabolism, particularly in the frontal regions, in ADHD children (Zametkin et al., 1990).

NONGENETIC ETIOLOGY

Injury to the brain, particularly in the anterior region, can produce the symptoms of ADHD. For the developing fetus, malnutrition, exposure to toxins (especially alcohol), and medical illness during pregnancy augment the risk for ADHD symptoms to appear. Circumstances around birth, especially the occurrence of toxemia (toxins in the blood) or hypoxemia (not enough oxygen in the blood), increase the risk for neurological injury in the newborn, which ultimately could result in symptoms of ADHD. During childhood development, many factors, particularly head trauma (by accident or maltreatment), infection, toxic poisoning, and malnutrition can produce ADHD symptoms. Neurologic conditions (e.g., epilepsy) and neurodevelopmental disorders (e.g., dyslexia, autism) are also commonly associated with some symptoms of ADHD. Although all these latter conditions produce ADHD-type symptoms, according to DSM-III-R they must be *excluded* as etiologic factors to make the diagnosis of the ADHD syndrome. In other words, the diagnosis of ADHD is assigned only where there is *no* neurodevelopmental disability or injury.

NATURAL HISTORY

Recognizing that there is substantial variability in the etiology of ADHD, it is obvious that the lifetime course and outcome is also highly variable. Symptoms persist into adulthood in about 50 percent of cases. Under these circumstances, the person is assigned a diagnosis of *attention deficit disorder-residual type*.

RELATION OF ADHD TO DRUG ABUSE

Serious psychiatric disorder is common among adults with a history of ADHD. ANTISOCIAL PERSONALITY disorder, alcohol and substance abuse, depression, and anxiety are the most common associated disorders. These associated disorders should not be viewed as invariant outcomes of ADHD but rather as disturbances for which ADHD youth are at increased risk. Whether any of these psychiatric outcomes are manifested depends on a variety of factors besides ADHD, including the child's self-esteem, opportunity for normal socialization with peers, success in school, and level of social and family support (Tarter, 1988).

With respect to alcohol and other drug abuse, augmented risk appears to be circumscribed to youth who have both ADHD and a conduct disor-

der. The association, however, between ADHD and substance abuse is complex. Alcohol and other drugs may be more subjectively rewarding for ADHD youth and adults than in the general population. Drug use is commonly tied to a general pattern of social deviancy and nonnormative peer affiliation. Where the ADHD person has been ostracized by the normative peer group, the use of alcohol/drugs may be just another manifestation of generalized maladjustment. Furthermore, alcohol/drug use may be mediated by a coexisting psychiatric disorder, such as anxiety and depression, and thus reflect an attempt at self-medication. Therefore, although there is substantial evidence demonstrating an increased risk for alcohol/drug abuse in ADHD youth, this association is complex and is contingent on many factors.

TREATMENT

Because the psychological manifestations of ADHD are multifaceted, it is necessary that comprehensive treatment interventions that encompass multiple components be implemented (Danforth, Barkley, & Stokes, 1991).

Pharmacotherapy. PSYCHOSTIMULANTS are therapeutically beneficial for approximately 75 percent of ADHD children and adults. The most commonly used medications are METHYLPHENIDATE (Ritalin), *d*-AMPHETAMINE, and PEMOLINE (Cylert). Tricyclic antidepressants are also effective in many cases. These medications have been shown to be useful for reducing problem behavior of ADHD in more than 100 research studies; however, they do not improve school performance or eliminate a conduct disorder where this disturbance is present.

Additionally, it is interesting to note that recent studies of the effects of stimulant medication treatment have produced disparate results. A 1998 study by N. M. Lambert and C. S. Hortsough puts forward that stimulant medication treatment may be a "gateway to abuse", suggesting a connection between stimulant treatment and adult dependence on cocaine as well as tobacco. Alternately, a 1999 study by Joseph Biederman, Eric Mick, Stephen V. Faraone, T. Wilens, and T. Spencer found pharmacotherapy to be responsible for a significant reduction in substance abuse for young people with ADHD (Beiderman, Mick & Faraone, 2000).

Lifestyle. Coordinated effort should be made to promote a healthy lifestyle, including scheduled regulation of bedtime, meals, homework, and recreation. Nutrition is important; however, contrary to popular belief and anecdotal reports, there is no substantive evidence linking diet or food allergies to the cause of ADHD. There is no scientific evidence indicating that a special diet or nutritional supplements can ameliorate ADHD.

Education. Informing parents and school personnel about the causes of ADHD and the nature of the behavior disorder can constructively assist the child by evoking empathy rather than anger. Thus, family counseling and teacher education are integral components of treatment to help maximize the child's adjustment in the home and at school.

Environmental Engineering. Structuring the environment so that the child is not easily distracted is an important intervention. In the home, this entails minimizing distracting stimulation from radio or television, especially while the youngster is doing homework. In the classroom, consideration should be given to the child's seat location to enable the teacher to ensure that the child persists at tasks, is not distracted by other students, or has no opportunity to be disruptive.

Behavior Modification. Behavior-modification strategies are effective for training the youngster to control impulses and to monitor behavior cognitively. Behavior-modification methods, which help the child, are also useful in teaching effective parenting skills.

(SEE ALSO: *Psychomotor Stimulant; Vulnerability As Cause of Substance Abuse*)

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REVISED BY CHRIS LOPEZ

AVERSION THERAPY *See* Treatment

AYAHUASCA In 1851, the botanist Richard Spruce observed natives along the Rio Negro in Brazil preparing a beverage from the roots of a vine, which he called *Banisteria caapi*, of the family Malpighiaceae (it was recently designated *Banisteriopsis caapi*.) He later observed the use of a similar drink in the Ecuadorian Amazon basin, where it was called *ayahuasca* (from the Quechua language, spoken in the Andes). He noted that the brew was often a mixture of *Banisteria caapi* with the roots of another indigenous plant. There were apparently several variations in the recipe for *caapi* and most of those who have studied it believe that each recipe produces somewhat different psychic effects. In 1929, the great pioneer of psychopharmacology, Louis Lewin, published a monograph describing the pharmacological actions and possible therapeutic uses of *Banisteria caapi*, whose actions he believed to be due to an active alkaloid, harmine. In early studies in patients with Parkinsonism, harmine produced improvements in chewing, swallowing, and movement that lasted from two to six hours. Curiously, it was reported to have little or no psychic effects. It was later shown that harmine acts to inhibit the enzyme monoamine oxidase, thereby raising levels of the neurotransmitters DOPAMINE and NOREPINEPHRINE.

Mixtures containing *Banisteriopsis caapi* are still in use among the indigenous peoples of the Amazon. A tea brewed from it and the leaves of *Psychotria viridis* has been used in shamanistic rituals for hundreds of years in Colombia, Brazil, and Peru. In recent years, a number of people seeking alternatives to Western medicine, and for other reasons, have participated in Santo Daime rituals, in which drinking ayahuasca is a central feature. The tea is said to induce ecstatic states, during which the participants claim to experience great insight. In southern Brazil, some psychotherapists and homeopaths have been known to bring clients or patients to participate in such rituals.

(SEE ALSO: *Hallucinogenic Plants; Hallucinogens*)

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JEROME H. JAFFE

B

BAC *See* Blood Alcohol Concentration, Measures of

BARBITURATES Barbiturates refer to a class of general central nervous system depressants that are derived from barbituric acid, a chemical discovered in 1863 by the Nobel Prize winner in chemistry (1905) Adolf von Baeyer (1835–1917). Barbituric acid itself is devoid of central depressant activity; however, German scientists Emil Hermann Fischer and Joseph von Mering made some modifications to its structure and synthesized barbital, which was found to possess depressant properties. Scientists had been looking for a drug to treat anxiety and nervousness but without the dependence-producing effects of OPIATE drugs such as OPIUM, CODEINE, and MORPHINE. Other drugs such as bromide salts, CHLORAL HYDRATE, and paraldehyde were useful sedatives, but they all had problems such as toxicity or they left such a bad taste in patients' mouths that they preferred not to take them. Fischer and von Mering noted that barbital produced sleep in both humans and animals. It was introduced into chemical medicine in 1903 and was soon in widespread use.

By 1913, the second barbiturate, PHENOBARBITAL, was introduced into medical practice. Since that time, more than 2,000 similar chemicals have been synthesized but only about 50 of these have been marketed. Although the barbiturates were quickly used to treat a number of disorders effectively, their side effects were becoming apparent. The chief problem, an overdose, can result in respiratory depression, which can be fatal. By the mid-1950s, more than 70 percent of admissions to a poison-control center in Copenhagen, Denmark, involved barbiturates. Additionally, it became apparent that the barbiturates were subject to abuse, which could lead to dependence, and that a serious withdrawal syndrome could ensue when the drugs were abruptly discontinued. In the 1960s, the introduction of a safer class of hypnotic drugs, the BENZODIAZEPINES reduced the need for barbiturates.

Barbiturates are dispensed in distinctly colored capsules making them very easy to identify by the lay public. In fact, users within the drug culture often refer to the various barbiturates by names associated with their physical appearance. Examples of these names include blue birds, blue clouds, yellow jackets, red devils, sleepers, pink ladies, and Christmas trees. The term *goofball* is often used to describe barbiturates in general. All barbiturates are chemically similar to barbital, the structure of which is shown in Figure 1.

All barbiturates are general central nervous system depressants. This means that sedation, sleep, and even anesthesia will develop as the dose is increased. Some barbiturates also are useful in reducing seizure activity and so have been used to treat some forms of epilepsy. The various barbiturates differ primarily in their onset and duration of

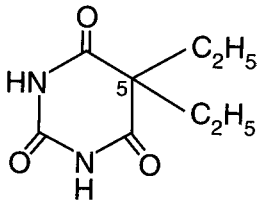

Figure 1
Barbital

action, ability to enter the brain, and the rate at which they are metabolized. These differences are achieved principally by adding or subtracting atoms to the two branches on position #5 in Figure 1. The barbiturates are classified on the basis of their duration of action, which ranges from ultrashort-acting to long-acting. The onset of action of the ultrashort-acting barbiturates occurs in seconds and lasts a few minutes. The short-acting compounds take effect within a few minutes and can last four to eight hours, while the intermediate- and long-acting barbiturates can take almost an hour to take effect but last six to twelve hours. Table 1 lists the common barbiturates, their trade names, typical route of administration, and plasma half-life. The plasma half-life is a measure of how long the drug remains in the blood, but not how long the effects last, although it does provide a general indication of when to expect the effects to wane (a half-life of five hours means that one-half of the drug will be removed from the system in five hours; one-half of the remaining drug will be removed during the next five hours, etc.).

EFFECTS ON THE BODY AND THERAPEUTIC USES

Barbiturates affect all excitable tissues in the body. However, NEURONS are more sensitive to their effects than other tissues. The depth of central nervous system depression ranges from mild sedation to coma and depends on many factors including which drug is used, its dose, the route of administration, and the level of excitability present just before the barbiturate was taken. The most common uses for the barbiturates are still to promote sleep and to induce anesthesia. Barbiturate-induced sleep resembles normal sleep in many ways, but there are a few important differences. Barbiturates reduce the amount of time spent in rapid eye movement or REM sleep—a very important phase of sleep. Prolonged use of barbiturates causes restlessness during the late stages of sleep. Since the barbiturates remain in our bodies for some time after we awaken, there can be residual drowsiness that can impair judgment and distort moods for some time after the obvious sedative effects have disappeared. Curiously, some people are actually excited by barbiturates, and the individual may even appear inebriated. This paradoxical reaction often occurs in the elderly and is more common after taking phenobarbital.

The general use of barbiturates as hypnotics (SLEEPING PILLS) has decreased significantly, since they have been replaced by the safer benzodiazepines. Phenobarbital and butabarbital are still available, however, as sedatives in a number of combination medications used to treat a variety of inflammatory disorders. These two drugs also are used occasionally to antagonize the unwanted overstimulation produced by ephedrine, AMPHETAMINE, and theophylline.

Since epilepsy is a condition of abnormally increased neuronal excitation, any of the barbiturates can be used to treat convulsions when given in anesthetic doses; however, phenobarbital has a selective anticonvulsant effect that makes it particularly useful in treating grand mal seizures. This selective effect is shared with mephobarbital and metharbital. Thus, phenobarbital is often used in hospital emergency rooms to treat convulsions such as those that develop during tetanus, eclampsia, status epilepticus, cerebral hemorrhage, and poisoning by convulsant drugs. The benzodiazepines are, however, gradually replacing the barbiturates in this setting as well.

It is not completely understood how barbiturates work but, in general, they act to enhance the activity of GABA on GABA-sensitive neurons by acting at the same receptor on which GABA exerts its effects (see Figure 2). GABA is a NEUROTRANSMITTER that normally acts to reduce the electrical activity of the brain; its action is like a brake. Thus, barbiturates enhance the braking effects of GABA to promote sedation. There is an area in the brain called the reticular activating system, which is responsible for maintaining wakefulness. Since this area has many interconnecting or polysynaptic neurons, it is the first to succumb to the barbiturates, and that is why an individual becomes tired and falls asleep after taking a barbiturate.

TABLE 1
Classification of Barbiturates on the Basis of Duration of Action

Drug Class and Generic Names	*Trade Names*	*Routes of Administration**	*Half-Life (in hours)*
Ultrashort-Acting:			
methohexital sodium	Brevital	IV	3.5–6**
thiamylal sodium	Surital	IV	†
thiopental sodium	Pentothal	IV	3–8**
Short-Acting:			
butalbital	‡	PO	35
hexobarbital	Sombulex	PO; IV	3–7
pentobarbital	Nembutal	PO; IM	15–48
secobarbital	Seconal	PO; IM	15–40
Intermediate-Acting:			
amobarbital	Amytal	PO; IM	8–42
aprobarbital	Alurate	PO	14–34
butabarbital	Butisol	PO	34–42
talbutal	Lotusate	PO	†
Long-Acting:			
phenobarbital	Luminal	PO; IV	24–96
mephobarbital	Mebaral	PO	11–67

*IV = intravenous; IM = intramuscular; PO = oral.
**Values are for whole body, half-life in the brain is less than 30 minutes.
†Half-life data not available for human subjects.
‡Various preparations in combination with acetaminophen.
SOURCE: Rall, 1990; Csáky, 1979.

PHARMACOKINETICS AND DISTRIBUTION

The ultrashort-acting barbiturates differ from the other members of this class mainly by the means by which they are inactivated. Methohexital and its relatives are very soluble in lipids (i.e., fatty tissue). The brain is composed of a great deal of lipid; when the ultrashort-acting barbiturates are given intravenously, they proceed directly to the brain to produce anesthesia and unconsciousness. After only a few minutes, however, these drugs are redistributed to the fats in the rest of the body so their concentration is reduced in the brain. Thus, recovery from IV barbiturate anesthesia can be very fast. For this reason, drugs such as methohexital and thiopental are used primarily as intravenous anesthetic agents and not as sedatives.

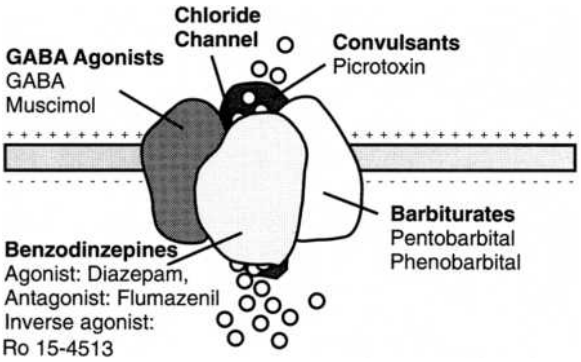

Figure 2
Barbiturates

The other longer-acting barbiturates must be metabolized by the liver into inactive compounds before the effects wane. Since these metabolites are more soluble in water, they are excreted through the kidneys and into the urine. As is the case with most drugs, metabolism and excretion is much quicker in young adults than in the elderly and infants. Plasma half-lives are also increased in pregnant women because the blood volume is expanded due to the development of the placenta and fetus.

TOLERANCE, DEPENDENCE, AND ABUSE

Repeated administration of any number of drugs results in eventual compensatory changes in the body. These changes are usually in the opposite direction of those initially produced by the drug such that more and more drug is needed to achieve the initial desired effect. This process is called TOLERANCE. There are two basic mechanisms for tolerance development: tissue tolerance and metabolic or pharmacokinetic tolerance. Tissue tolerance refers to the changes that occur on the tissue or cell that is affected by the drug. Metabolic tolerance refers to the increase in the processes that metabolize or break down the drug. This process generally occurs in the liver. Barbiturates are subject to both types of tolerance development.

Tolerance does not develop equally in all effects produced by barbiturates. Barbiturate-induced respiratory depression is one example. Barbiturates reduce the drive to breathe and the processes necessary for maintaining a normal breathing rhythm. Thus, while tolerance is quickly developing to the desired sedative effects, the toxic doses change to a lesser extent. As a result, when the dose is increased to achieve the desired effects (e.g., sleep), the margin of safety actually decreases as the dose comes closer to producing toxicity. A complete cessation of breathing is often the cause of death in barbiturate poisoning (Rall, 1990).

If tolerance develops and the amount of drug taken continues to increase, then PHYSICAL DEPENDENCE can develop. This means that if the drug is suddenly stopped, the tissues' compensatory effects become unbalanced and withdrawal signs appear. In the case of barbiturates, mild signs of withdrawal include apprehension, insomnia, excitability, mild tremors, and loss of appetite. If the dose was very high, more severe signs of withdrawal can occur, such as weakness, vomiting, decrease in blood pressure regulatory mechanisms (so that pressure drops when a person rises from a lying position, called orthostatic hypotension), increased pulse and respiratory rates, and grand mal (epileptic) seizures or convulsions. DELIRIUM with fever, disorientation, and HALLUCINATIONS may also occur. Unlike withdrawal from the opioids, withdrawal from central nervous system depressants such as barbiturates can be life threatening. The proper treatment of a barbiturate-dependent individual always includes a slow reduction in the dose to avoid the dangers of rapid detoxification.

Few, if any, illegal laboratories manufacture barbiturates. Diversion of licit production from pharmaceutical companies is the primary source for the illicit market. Almost all barbiturate users take it by mouth. Some try to dissolve the capsules and inject the liquid under their skin (called skin-popping) but the toxic effects of the alcohols used to dissolve the drug and the strong alkaline nature of the solutions can cause lesions of the skin. Intravenous administration is a rare practice among barbiturate abusers.

Many barbiturate users become dependent to some degree during the course of treatment for insomnia. This type of problem is called iatrogenic, because it is initiated by a physician. In some instances the problem will be limited to continued use at gradually increasing doses at night, to prevent insomnia that is in turn due to withdrawal. However, some individuals who are susceptible to the euphoric effects of barbiturates may develop a pattern of taking increasingly larger doses to become intoxicated, rather than for the intended therapeutic effects (for example, to promote sleepiness). To achieve these aims, the person may obtain prescriptions from a number of physicians and take them to a number of pharmacists—or secure their needs from illicit distributors (dealers). If the supply is sufficient, the barbiturate abuser can rapidly increase the dose within a matter of weeks. The upper daily limit is about 1,500 to 3,000 milligrams; however, many can titrate their daily dose to the 800 to 1,000 milligram range such that the degree of impairment is not obvious to others. The pattern of abuse resembles that of ethyl (drinking) ALCOHOL, in that it can be daily or during binges that last from a day to many weeks at a time. This pattern of using barbiturates for intoxification is more typically seen in those who, from the beginning, obtain barbiturates from illicit sources rather than those who began by seeking help for insomnia.

Barbiturates are sometimes used along with other drugs. Often, the barbiturate is used to potentiate, or boost, the effects of another drug upon which a person is physically dependent. Alcohol and HEROIN are commonly taken together in this way. Since barbiturates are "downers," they also are used to counteract the unwanted overstimulation associated with stimulant-induced intoxication. It is not uncommon for stimulant abusers (on

COCAINE or amphetamines) to use barbiturates to combat the continued "high" and the associated motor disturbances associated with heavy and continued cocaine use. Also, barbiturates are used to ward off the early signs of withdrawal from alcohol.

Treatment for barbiturate dependence is often conducted under carefully controlled conditions, because of the potential for severe developments, such as seizures. Under all conditions, a program of supervised withdrawal is needed. Many years ago, pentobarbital was used for this purpose and the dose was gradually decreased until no drug was given. More recently, phenobarbital or the benzodiazepines—CHLORDIAZEPOXIDE and diazepam—have been used for their greater margin of safety. The reason that the benzodiazepines sometimes work is because the general central nervous system depressants—barbiturates, alcohol, and benzodiazepines—develop cross-dependence to one another. Thus a patient's barbiturate or alcohol withdrawal signs are reduced or even eliminated by diazepam.

(SEE ALSO: *Addiction: Concepts and Definitions; Withdrawal*)

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SCOTT E. LUKAS

BARBITURATES: COMPLICATIONS

Barbiturates are central nervous system (CNS) DEPRESSANTS ("downers"). These drugs produce sedative, hypnotic, and anesthetic effects. Depending on the dose used, any single drug in this class may produce sedation (decreased responsiveness), hypnosis (sleep), and anesthesia (loss of sensation). A small dose will produce sedation and relieve ANXIETY and tension; a somewhat larger dose taken in a quiet setting will usually produce sleep; an even larger dose will produce unconsciousness. The sleep produced by barbiturates, however, is not identical with normal sleep. Normal sleep consists of alternating phases of slow-wave sleep (SWS)—when the electroencephalogram (EEG) shows a high-voltage and low-frequency pattern—and rapid-eye-movement (REM) sleep. In the REM sleep phase, the EEG shows an arousal pattern and skeletal muscles relax, eyes move rapidly and frequently, and dreaming is thought to take place. Barbiturates decrease REM (or dreaming) sleep and thereby disturb the balance between SWS and REM sleep.

As is true for most drugs that act on the CNS, the effects of these drugs are also influenced markedly by the user's previous drug experience, the circumstances in which the drug is taken, and the route of administration of the drug. For example, a dose taken at bedtime may produce sleep, whereas the same dose taken during the daytime may produce a feeling of euphoria, incoordination, and emotional response. This, in many ways, is what happens with alcohol intoxication. In fact, the behavioral effects of this class of drugs is very similar to those observed after drinking ALCOHOL, and the user may experience impairment of skills and judgment not unlike that experienced with alcohol. It is therefore not surprising that the effects of barbiturates are enhanced when taken in combination with alcohol, antianxiety drugs (BENZODIAZEPINES), and other CNS depressants such as opioids, antihistamines, and OVER-THE-COUNTER cough and cold medications containing these drugs. Barbiturates, however, differ from some other SEDATIVE-HYPNOTIC drugs in that they do not elevate the PAIN threshold. In fact, patients experiencing severe pain may become agitated and delirious if they are given barbiturates without also receiving ANALGESICS.

Barbiturates are generally classified as being long, intermediate, short, and ultra-short acting on the basis of their duration of effect. The long-acting barbiturates, such as phenobarbital, which were at one time mainly employed as daytime sedatives for

the treatment of anxiety, produce sedation that lasts from twelve to twenty-four hours. Phenobarbital is still one of the drugs used for the treatment of grand mal epilepsy. The short- and intermediate-acting drugs such as pentobarbital and secobarbital, which were once mainly employed as hypnotics, produce CNS depression that ranges from three to twelve hours, depending on the compound used. The ultrashort-acting barbiturates (e.g., thiopental) are used for the induction of anesthesia, because of the ease and rapidity with which they induce sleep when given intravenously. The effects of barbiturates on judgment and other mental as well as motor skills, however, may persist much longer than the duration of the hypnotic effect. For this and other reasons, for the treatment of anxiety or insomnia, barbiturates have largely been replaced by the generally safer group of drugs called benzodiazepines.

The respiratory system is significantly depressed by the administration of barbiturate doses that are larger than those usually prescribed. Furthermore, there is a synergistic effect when barbiturates are combined with alcohol and other central nervous system depressants—often with a fatal outcome. Barbiturates are frequently used for suicides. For this reason, too, the barbiturates have been displaced by the less toxic benzodiazepines. The symptoms of acute barbiturate toxicity resemble the effects observed after excessive alcohol ingestion. Although repeated administration of barbiturates results in CNS tolerance, thus producing less intoxication, tolerance does not appear to develop to the same extent in regard to the respiratory depressant and lethal effects of the barbiturates; the person addicted to barbiturates may therefore be at a greater risk of respiratory toxicity because of less pronounced CNS euphoric effects with higher doses. Tolerance to barbiturates also affects metabolism; the administration of these drugs speeds up not only their own metabolism (i.e., shortens their effectiveness) but also the metabolism of a large number of other drugs. This property has been of use in some special cases (as in jaundice of the newborn), but it can be hazardous in others when it decreases the effectiveness of another drug (e.g., an anticoagulant used to treat thrombosis).

Long-term users experience withdrawal symptoms when the barbiturate is stopped abruptly. Abrupt cessation also leads to an increase in the amount and intensity of REM sleep (REM rebound). The intensity of the withdrawal symptoms varies with the degree of abuse and may range from sleeplessness and tremor in mild cases to delirium and convulsions in severe cases. Fatalities have occurred as a result of barbiturate WITHDRAWAL, usually from withdrawal of short-acting barbiturates.

In some individuals, barbiturates may produce CNS excitement rather than CNS depression. This type of idiosyncratic reaction occurs most frequently in elderly people. Among the side effects sometimes seen, there may be rashes and muscle and body aches.

(SEE ALSO: *Expectancies; Sleep, Dreaming, and Drugs*)

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JAT M. KHANNA

BEERS AND BREWS Beers and brews are beverages produced by yeast-induced fermentation of malted cereal grains, usually barley malt, to which hops and water have been added. They generally contain 2 to 9 percent ethyl ALCOHOL, although some may contain as much as 15 percent. Various types and flavors are created by adding different combinations of malts and cereals and allowing the process to continue for varying lengths of time.

BREWING HISTORY

The origin of beer is unknown, but it was an important food to the people of the Near East, probably from Neolithic times, some 10,000 years ago. The making of beer and of bread developed at the same time. In Mesopotamia (the ancient *land between the rivers* as the Greeks called it), an early record from about 5,000 years ago describing the recipe of the "wine of the grain" was found written in Sumerian cuneiform on a clay tablet. In ancient Egypt, at about the same time, barley beer was

brewed and consumed as a regular part of the diet. It was known as hek and tasted like a sweet ale, since there were no hops in Egypt. Egyptians continued to drink it for centuries, although the name was changed to hemki. More than 3,200 years ago, the Chinese made a beer called kiu that was most likely made from two parts millet and one part rice. With water added, the concoction was heated in clay pots; flour and various plants were added to provide the yeast and flavors, respectively.

The ancient Greeks, however, preferred wine and considered beer to be a drink of barbarians. Beer was drunk on special occasions in ancient Rome; Plutarch wrote of a feast in which Julius Caesar served his officers beer as a special reward after they had crossed the Rubicon river. Once the art of brewing reached England, beers and ales became the preferred drink of the rich and poor alike. King Henry VIII of England was said to consume large quantities during breakfast. It was soon discovered that sailors who drank beer avoided scurvy, a disease caused by a lack of adequate amounts of vitamin C. Thus, beer was added to each ship's provisions and was even carried on the Mayflower during the crossing of the Atlantic Ocean in 1620. American colonists quickly learned to make their beer with Indian corn (maize), and much U.S. beer is still made with corn, although rice and wheat are also used in the mix with barley malt.

MAKING BEER

The first step in making beer is to allow barley to sprout (germinate) in water, a process that releases an important enzyme, amylase. Germinated barley seeds are called malt. Once the malt is crushed and suspended in water, the amylase breaks down the complex starch into more basic sugars. The reaction is stopped by boiling, and the concoction is filtered. This clear solution is mixed with hops (to provide the bitter flavor) and a starter culture of yeast (to begin the alcoholic fermentation process). Carbon dioxide gas (the fizz or bubbles) is produced, along with ethyl alcohol (ethanol or drinking alcohol). The malt and hops are then removed (and generally sold for cattle feed) while the yeast is skimmed off as fermentation proceeds. After the desired effect is achieved, the beer is filtered and bottled, or it is stored in kegs for aging. During the aging process (2 to 24 weeks) proteins settle to the bottom or are digested by enzymes. The carbonation (fizz) that occurred during fermentation is then drawn off and forced back in during the bottling process.

BEER TYPES

There are two major types of beers: top-fermenting and bottom-fermenting. Top fermentation occurs at room temperature 59° to 68°F (15°–20°C) and is so named because the yeast rises to the top of the vessel during fermentation. This older process produces beers that have a natural fruitiness and include the wheat beers, true ales, stouts, and porters. Their flavor is most completely expressed when served at moderate (i.e., room) temperatures. The development of yeasts that sank during this process resulted in brews that were more stable between different batches. Most of the major brewers have switched to the bottom yeasts and cold storage (lagering). The significance of using yeast that sinks during fermentation is that airborne yeasts cannot mix with the special yeast and contaminate the process.

The most popular type of beer in the United States is lager, a pale, medium-hop-flavored beer. It is mellowed several months at 33°F (0.5°C) to produce its distinctive flavor. Lager beers average 3.3 to 3.4 percent ethyl alcohol by weight and are usually heavily carbonated. Pilsner is a European lager (that originated in medieval Pilsen, now the Czech city of Plzen) that is stored longer than other lagers and has a higher alcohol content and a rich taste of hops. Dark beers are popular in Europe but are not generally produced in the United States. The dark color is achieved by roasting the malt; dark beer has a heavier and richer taste than lager beer. British beers are many and varied, both pale and dark; some have a number of unique additives, including powdered eggshell, crab claws or oyster shells, tartar salts, wormwood seeds, and horehound juice. Porter, popular in England, is another dark beer—originally called porter's beer, it was a mixture of ale and beer. The porters of today are a sweet malty brew and contain 6 to 7 percent alcohol. Malt liquors are beers that are made using a higher percentage of fermentable sugars, resulting in a beverage with 5 to 9 percent alcohol content; the mild fruity flavor has a spicy taste and lacks the bitterness of hops. Low-calorie (sometimes called "light" or "lite") beers are pro-

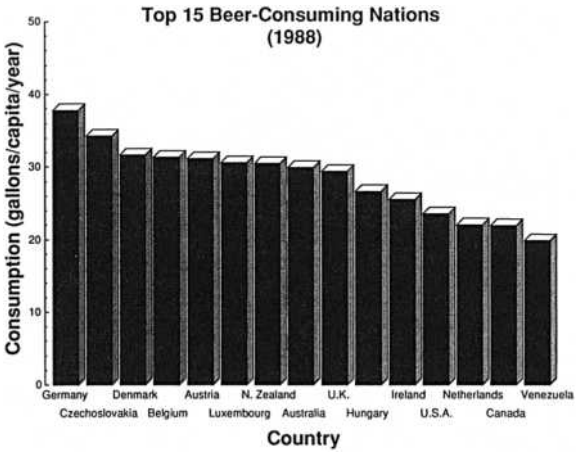

Figure 1

duced by decreasing the amount of grain used in the initial brew (using more water per unit of volume) or by adding an enzyme that reduces the amount of starch in the beer. These light beers contain only about 2.5 to 2.7 percent alcohol.

Brewing is subject to national laws concerning allowable ingredients in commercial products. Although chemical additives are allowed by some countries (e.g., the United States), German and Czech purity laws consider beers and brews a natural historical resource and disallow anything that was not part of the original (medieval) brewing tradition. Individuals sensitive to U.S. or Canadian beers are often able to drink pure beers.

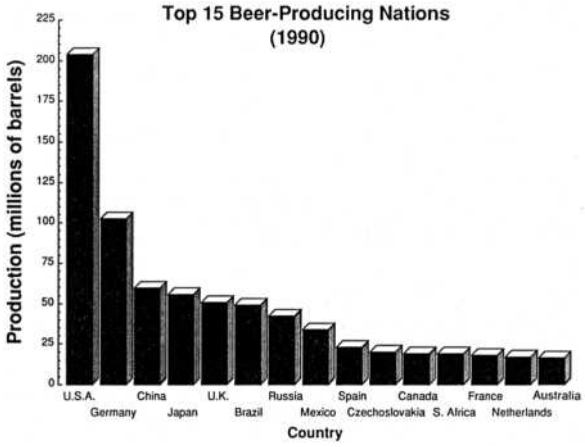

Figure 2

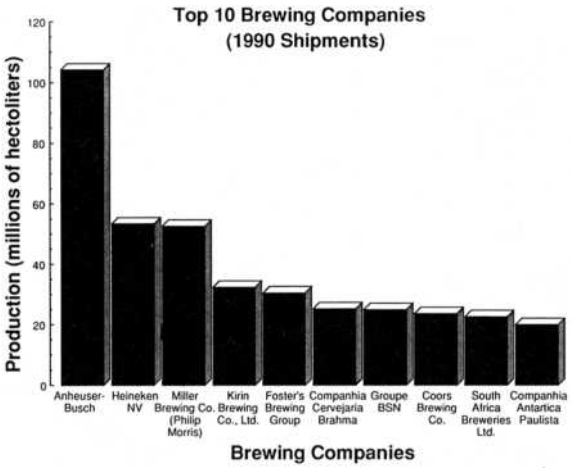

Figure 3

SOURCE: Impact Databank, M. Shanken Communications, Inc., New York, N.Y.

WORLDWIDE CONSUMPTION OF BEERS AND BREWS

About 128.6 billion liters of beer were commercially produced in the world in 1997 according to the *World Drink Trends, 1999 Edition* (cited in Alcoweb). The United States led the world in beer production, producing 23.6 billion liters of beer in 1997. China followed with 17.0 billion liters, and Germany rounded out the top three in beer production volume with 11.5 billion liters. For the same year, *World Drink Trends* reports that the Czech Republic drank the most beer, consuming 161.4 liters per person. Other countries leading in beer consumption were the Republic of Ireland (152.0 liters per person) and Germany (131.2 liters per person). A 1999 study conducted by Euromonitor cited in *Prepared Foods* reported that the United States led the world in beer consumption, consuming over 17 billion liters. *Beverage World International* cites another Euromonitor study that lists the top three brewers for 1997 as Anheuser-Busch, Heineken, and Miller Brewing. Figures 1–3 show the relative distribution of the top drinking and brewing nations, as well as the top brewing companies.

(SEE ALSO: *Alcohol: History of Drinking*)

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REVISED BY NANCY FAERBER

BEHAVIORAL ECONOMICS Contemporary conceptualizations view drug dependence as a "syndrome in which the use of a drug is given a much higher priority than other behaviors that once had higher value" (Jaffe, 1990). Thus, in order to understand drug dependence, the complex interactions among reinforcers must be understood. Consider, for example, the frequently observed case of polydrug abuse. On some occasions, the drugs may be used simultaneously (e.g., cocaine and heroin), whereas on other occasions one drug may be used in lieu of another (e.g., benzodiazepines and opioids). Understanding reinforcer interactions is also vital to developing effective treatment because treatments, be they pharmacological (e.g., methadone, nicotine gum) or nonpharmacological (e.g., AA meetings, alternative behaviors), often try to supplant drug reinforcers with other more acceptable reinforcers. Although understanding reinforcer interactions is important for understanding drug abuse, no method currently exists to quantify or even categorize these different types of reinforcer interactions.

Behavioral economics provides a means to understand the interactions among qualitatively different reinforcers. The value of behavioral economics derives from its unique ability to quantify the effects of qualitatively different reinforcers and their interactions (Bickel et al., 1992; Bickel, DeGrandpre, & Higgins, 1995; Bickel & Vuchinich, 2000; Hursh, 1980, 1991).

Several concepts from behavioral economics are relevant to this important issue. A central concept is the *demand law* which stipulates that total consumption decreases as price increases, all else being equal (Allison, 1979). Price can be considered anything (e.g., response requirement, monetary price, delay, changes in the amount of the commodity while holding the monetary or work price constant) that decreases availability of a commodity. Indeed, drug self-administration studies that vary price (response requirement) report results consistent with the demand law; that is, drug consumption decreases as the response requirement decreases (Griffiths, Bigelow, & Henningfield, 1980; Young & Herling, 1986). However, behavioral economics does more than simply restate this observation with a different terminology; it adds by quantitatively characterizing the relation between price and consumption via the economic measure of *own-price elasticity* (Bickel & DeGrandpre, 1996; Hursh & Bauman, 1987; Samuelson & Nordhaus, 1985). Own-price elasticity measures the proportional change in consumption across price conditions. If consumption of a particular reinforcer decreases proportionally to a large extent as price increases, then the consumption is referred to as *elastic*. If consumption decreases proportionally to a limited extent as price increases, then the consumption is referred to as *inelastic*. Elastic and inelastic consumption are quantified by elasticity coefficients greater than 1.0 and less than 1.0, respectively (Hursh, 1980). When examined across a broad range of prices, elasticity of demand for many reinforcers is often mixed: inelastic at low prices and elastic at higher prices.

With this method, then, qualitatively different reinforcers can be compared and distinguished in drug-dependent populations. For example, in a recent study money and cigarettes were compared among cigarette-deprived subjects on progressive ratio schedules (Bickel & Madden, 1999). Response requirements were increased across sessions and the same response requirement was imposed separately for both commodities. At the lowest response requirements, money was self-administered to a greater extent than cigarettes (a greater intensity of demand). As response requirement increased, money was shown to be more sensitive to price than cigarettes. The own-price elasticities of money and cigarettes were 2.1 and 0.9, respectively, with money being 2.3-fold more sensitive to price. Such

efforts can be meaningfully extended to clinical settings via simulation technology (Petry & Bickel, 1998; Jacobs & Bickel, 1999). Jacobs and Bickel used questionnaires to assess the reported consumption of cigarettes and heroin singly and concurrently across a range of prices ($0.01 to $1,120) in opioid-dependent smokers undergoing treatment for heroin addiction. Across conditions in which cigarettes and heroin were available alone, or concurrently, demand for heroin was less elastic than for cigarettes. For example, heroin consumption was defended to a greater extent across increases in price than cigarettes. Taken together, these studies indicate that individuals with drug dependence value the primary drug of dependence more than other commodities.

Own-price elasticity, like other behavioral effects, is *not* an inherent "property" of a drug, but is determined by several variables including the context in which it is measured (Hughes, Higgins, & Bickel, 1988). Indeed, the presence of other reinforcers can alter own-price elasticity (Hursh, 1984; Bickel et al., 1995). Within an economic framework, reinforcer interactions lie on a continuum that can be quantified with a measure termed cross-price elasticity (i.e., proportional change in consumption of reinforcer A as a function of the price of reinforcer B) (Hursh & Bauman, 1987). At one end of the continuum, reinforcers are *substitutable* reinforcers; that is, as the price of one reinforcer increases (e.g., price of attending a movie theater), consumption of a second reinforcer (i.e., the substitute) will increase (e.g., video rentals). At the other end of the continuum, reinforcers can be *complementary;* that is, as the cost of one reinforcer increases (e.g., soup), the consumption of a second reinforcer (i.e., the complement) also decreases (e.g., soup crackers). Between these two extremes are *independent* reinforcers; as the cost of one reinforcer increases (e.g., movie attendance), the consumption of a second reinforcer (e.g., soup crackers) will remain unchanged. The quantification of substitutes, compliments, and independent reinforcers is measured by cross-price elasticity values greater than zero, less than zero, and equal to zero, respectively (Hursh & Bauman, 1987).

A review and reanalysis of self-administration studies support this continuum (Bickel et al., 1995). Specifically, the results of sixteen drug self-administration studies that employed concurrently available reinforcers were reanalyzed using the economic measure of cross-price elasticity. In that review, price was defined as responses required per milligram of drug per ingestion (i.e., unit price) (Hursh et al., 1988; Bickel et al., 1990). A wide variety of reinforcers (e.g., cocaine, food, heroin, and cigarettes) across several species (e.g., rats, humans, baboons, and rhesus monkeys) were examined. Overall, the results of reanalysis demonstrated that each of the studies demonstrated one of the three types of interactions specified by economics. Extensions can also be made to clinical settings again by simulations. For example, the Petry and Bickel (1998) study described earlier found that heroin prices affected other drug purchases. The cross-price elasticity measure showed that as the price of heroin rose and heroin purchases decreased, valium and cocaine purchases increased. However, increases in the price of valium did not affect heroin purchases. This suggests an asymmetrical relationship between heroin and valium purchases when the price of one drug increases while the price of the other remains constant.

Collectively, these studies suggest that the availability of a concurrent reinforcer can significantly modulate drug intake. Sometimes, reinforcers interact as substitutes, whereas at other times they function as complements. Introducing a *substitute* tends to decrease drug consumption, while introducing a *complement* may increase drug consumption. Unfortunately, few of these studies has prospectively examined these interactions. Additional research that systematically and parametrically examines reinforcer interactions may facilitate a greater understanding of the ways in which the availability of alternative reinforcers increases or decreases drug consumption.

This approach to studying interactions of any reinforcers may have several important implications. First, this research may have implications for understanding behavioral vulnerability. For example, one issue in drug dependence is why only a few people exposed to a drug of abuse go on to become dependent. Perhaps, vulnerable individuals have limited availability to alternative nondrug substitutes and easy access to complements for drug taking (e.g., Carroll, Lac, & Nygaard, 1989). Second, this research may provide a useful way to characterize the various types of polydrug abuse (e.g., Petry & Bickel, 1998). Third, such research may provide an empirical basis for developing treatment strategies; that is, in treatment it may be

worthwhile to decrease the response cost of obtaining other substitutable nondrug reinforcers. With regard to complements, this research suggests that drug-abuse treatment may be more successful if consumption of complements is also decreased. For example, Higgins and colleagues (1993) examined the effects of disulfiram (antabuse) therapy among patients abusing cocaine and alcohol. Disulfiram is a medication that is used to deter alcohol use by inducing nausea and vomiting when alcohol is consumed. This study found that supervised disulfiram therapy was associated with significant decreases in alcohol and cocaine use as measured by breath and urine samples. More recently, Carroll and colleagues (1998) examined the effects of three standardized psychotherapy treatments alone and with disulfiram treatment among a large, diverse sample of individuals who abuse cocaine and alcohol. Their results indicated that disulfiram treatment was associated with significantly better retention in treatment and longer duration of abstinence from alcohol and cocaine use. Fourth, behavioral economic analyses of research may predict conditions in which relapse is likely. Relapse may occur when substitutable reinforcer interactions become unavailable and complements become available (Vuchinch & Tucker, 1988, 1996). Thus, the study of reinforcer interactions may increase our understanding of the etiology, maintenance, and treatment of drug dependence as well as relapse.

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BEHAVIORAL MODIFICATION AS A TREATMENT *See* Treatment Types: Approaches Based on Behavioral Principles

BEHAVIORAL TOLERANCE In everyday language, TOLERANCE implies the ability to withstand something. In pharmacology, the term *tolerance* is close to this meaning. To understand the technical meaning of the word, however, requires an understanding of the concept of the *potency* of a drug. A drug's potency is expressed in terms of the amount (the dose) of the drug needed to produce a certain effect. To illustrate, drugs may be compared with respect to potency. For example, relief from headache may be achieved with 650 milligrams of aspirin or with 325 milligrams of ibuprofen; in this case ibuprofen is said to be more potent, because less drug is needed to produce a particular effect (relief of headache). Tolerance is said to occur when a drug becomes *less potent* as a result of prior exposure to that drug. That is, following exposure (usually repeated or continuous administrations) to a drug, it may take more of the drug to get the same effect as originally produced.

The expression *behavioral tolerance* often is used simply to refer to a drug's decreased potency in affecting a specified behavior after repeated or continuous exposure to the drug. In other contexts, however, the expression has taken on a more restricted and special meaning; it is employed only when behavioral factors have been shown experimentally to have contributed to the development of tolerance.

This special meaning is applied when either of two sets of circumstances are encountered. In the first, drug tolerance is shown to be specific to the context in which the drug is administered; in the second, drug tolerance is shown to occur only if drug administration precedes particular behavioral circumstances. Examples of each may help clarify the distinctions between them and between "simple" tolerance and behavioral tolerance.

Context-specific tolerance has been researched extensively by Siegel and his colleagues (see Siegel, 1989, for an overview). In a typical experiment two groups of subjects are compared; subjects in both groups receive the same number of repeated exposures to the drug (e.g., morphine) and then are tested for their response to the drug (e.g., alleviation of pain). For one group, the test occurs in the environment where drugging took place; for the other the test occurs in a novel environment. Typically, only those from the group tested in the familiar environment show tolerance. Siegel's theory is that subjects develop, via the principles of Pavlovian conditioning, a conditioned compensatory response that is elicited by the drug-administration context—and that this response counteracts the effect of the drug (see Baker & Tiffany, 1985, for a different view). The phenomenon of context-specific tolerance helps explain why many overdoses of abused drugs occur when the drug is taken in a novel situation—the new context does not elicit compensatory responses that counteract the effects of the drug.

The importance of the temporal relationship between drug administration and behavior is illustrated by the phenomenon of "contingent" tolerance. The basic technique for identifying contingent tolerance was pioneered by Chen (1968), and a clear example is provided by Carlton and Wolgin (1971). Three groups of rats had the opportunity to drink milk for 30 minutes each day.

For each group, injections of a drug or just a saline vehicle were made twice each day: For Group 1, each session was preceded by an injection of 2 milligrams per kilogram of AMPHETAMINE, followed by an injection of just the saline vehicle; for Group 2, the order of injections was reversed: saline before drinking, amphetamine after; Group 3 (the control group) received saline both before and after each session.

For Group 1 the drug initially decreased drinking, but during the course of several administrations, drinking recovered to control levels (i.e., tolerance developed). For Group 2, no effect on drinking was observed as a function of receiving the drug after sessions, so after several days (by which time subjects in Group 1 were tolerant) these subjects were given amphetamine before (rather than after) and saline after sessions (i.e., the conditions for Group 1 were implemented). Even though these subjects had received amphetamine just as frequently as the subjects in Group 1, when it was given before the session, drinking was suppressed just as much as it had been for Group 1 initially. Following repeated precession exposure to amphetamine the subjects in Group 2 became tolerant. These findings and many others like them show that, in many cases, for tolerance to develop to a drug's behavioral effects, mere repeated exposure to the drug is not enough. In addition, the drug must be active while the behavior of interest is occurring. (See Goudie & Demellweek, 1986, and Wolgin, 1989, for reviews.)

Contingent tolerance is sometimes called *learned* tolerance because it appears that it is a manifestation of learning to behave accurately while under the influence of a drug. An influential theory about the origin of contingent tolerance is the "reinforcement loss" theory of Schuster, Dockens and Woods (1966; for a review see Corfield-Sumner & Stolerman, 1978). Loosely stated, the theory is that contingent tolerance will emerge in situations where the initial effect of the drug is to produce a loss of reinforcement (e.g., result in a failure to meet the demands of the task). Although there are limits to the generality of the theory (Genovese, Elsmore, & Witkin, 1988), it has an excellent predictive record.

(SEE ALSO: *Addiction: Concepts and Definitions; Reinforcement; Tolerance and Physical Dependence; Wikler's Pharmacologic Theory of Drug Addiction*)

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BENZEDRINE/BENZEDRINE INHALERS *See* Amphetamine Epidemics

BENZENE *See* Inhalants

BENZODIAZEPINES The benzodiazepines were introduced into clinical practice in the 1960s

for the treatment of anxiety and sleep disorders. Members of this class of drug were classified initially as *minor tranquilizers* although this term has fallen into disfavor. These agents have proven to be safe and effective alternatives to older SEDATIVE-HYPNOTIC agents such as BARBITURATES, CHLORAL HYDRATE, glutethimide, and carbamates. Benzodiazepines are widely prescribed drugs, with 8.3 percent of the U.S. population reporting medical use of these agents in 1990.

BASIC PHARMACOLOGY

All benzodiazepines produce similar pharmacologic effects, although the potency for each effect may vary with individual agents. They decrease or abolish ANXIETY, produce sedation, induce and maintain sleep, control certain types of seizures, and relax skeletal muscles. The basic chemical structure is shown in Figure 1.

Dissimilarity in the effects of different benzodiazepines tend to be more quantitative than qualitative in nature. Many of these differences are attributable to how benzodiazepines are absorbed, distributed, and metabolized in the body. A few benzodiazepines—clorazepate for example—are pro-drugs; that is, they become active only after undergoing chemical transformation in the body. The extent to which a benzodiazepine is soluble in fatlike substances—that is, the degree to which it is lipophilic—determines the rate at which it crosses the tissue barriers that protect the brain. Drugs that are highly lipophilic such as DIAZEPAM (Valium) rapidly enter and then leave the brain. Benzodiazepines are metabolized in the body in a number of ways (see Table 1). Many benzodiazepines are transformed in the liver into compounds that possess pharmacologic activity similar to that of the originally administered drug. Diazepam, prazepam, and halazepam are all converted to the active metabolite desmethyldiazepam, which is eliminated from the plasma at a very slow rate. Oxazepam (Serax) and lorazepam (Ativan), in contrast, are conjugated with glucuronide, a substance formed in the liver, to form inactive metabolites that are readily excreted into the urine.

Most of the effects that result from the administration of benzodiazepines are a consequence of the direct action of these agents on the central nervous system. Benzodiazepines interact directly with proteins that form the benzodiazepine receptor. Benzodiazepine receptors exist as part of a larger receptor complex (Figure 2). The interaction of the NEUROTRANSMITTER gamma-amino butyric acid (GABA) with this complex leads to the enhanced flow of chloride ions into neurons (Kardos, 1993). This complex is referred to as the $GABA_A$ receptor-chloride ion channel complex. Much of the available evidence indicates that the action of benzodiazepines involves a facilitation of the effects of GABA and similarly acting substances on the $GABA_A$ receptor complex, thus leading to an increased movement of chloride ions into nerve cells. Entry of chloride ions into neurons tends to diminish their responsiveness to stimulation by other nerve cells, and consequently substances that produce an increase in chloride flow into cells depress the activity of the central nervous system. This depressant effect becomes manifested as either sedation or sleep. Agents that increase chloride ion inflow include not only the benzodiazepines but also other central nervous system depressant agents such as ETHANOL (alcohol) and the barbiturates. Benzodiazepines differ from barbiturates in that they require the

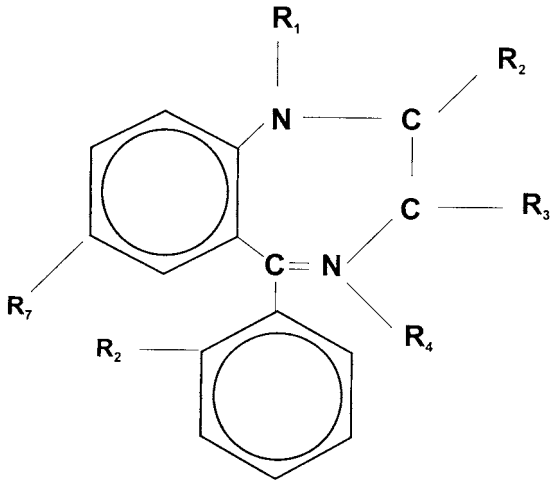

Figure 1
Outline of the basic structure of the benzodiazepines. R denotes substituent groups such –H, –O, –OH, –NO_2, and –Cl that are attached to the core benzodiazepine structure. These groups determine the precise physiochemical and pharmacologic properties of each benzodiazepine.
SOURCE: Adapted from Rall, T.W. (1990). *Hypnotics and sedatives: Ethanol.* Figure created by Rebecca Bulotsky.

TABLE 1
Benzodiazepines Available in the United States

Anxiolytics

Generic Name	*Trade Name*	*Usual Dose (mg/day)*	*Half-life (hours)*	*Transformation Pathway*	*Metabolites (half-life, hours)*
Alprazolam	Xanax	0.75–4	8–15	Oxidation	Alphahydroxyalprazolam Benzophenone
Bromazepam			20–30	Oxidation	
Chlordiazepoxide	Librium	15–100	5–30		Desmethylchlordiazepoxide Demoxepam Desmethyldiazepam (36–96)
Clonazepam	Klonopin	1.5–20	18–50	Nitroreduction	Inactive 7-amino or 7-acetyl amino derivatives
Clobazam	Frisium	20–30	18	Oxidation	Desmethylclobazam (up to 77)
Clorazepate	Tranxene	15–60	30–100	Oxidation	Desmethyldiazepam (36–96)
Diazepam	Valium	4–40	20–70	Oxidation	Desmethyldiazepam (36–96)
Halazepam	Paxipam	60–160	14	Oxidation	Desmethyldiazepam (36–96) 3-hydroxyhalazepam
Lorazepam	Ativan	2–4	10–120	Conjugation	Inactive glucuronide conjugate
Oxazolam				Oxidation	Desmethyldiazepam (36–96)
Oxazepam[a]	Serax	30–120	5–15	Conjugation	Inactive glucuronide conjugate
Prazepam	Centrax	20–60	30–100	Oxidation	Desmethyldiazepam (36–96)

Hypnotics

Generic Name	*Trade Name*	*Usual Dose (mg/day)*	*Half-life (hours)*	*Transformation Pathway*	*Metabolites (half-life, hours)*
Brotizolam			4–7	Oxidation	
Estazolam	ProSom	1–2	8–24	Oxidation	
Flunitrazepam			10–40	Oxidation nitro-reduction	Desmethylflunitrazepam
Flurazepam	Dalmane	15–30	.5–3.0	Oxidation	Desalkylflurazepam (36–120) Hydroxyethyl flurazepam (1–4) Flurazepam aldehyde (2–8)
Lormetazepam			8–20	Conjugation	
Nitrazepam	Mogadon	2.5–10	20–30	Nitroreduction	
Quazepam	Doral	7.5–15	20–40	Oxidation	Oxoquazepam (25–35) Desalkylflurazepam (36–120)
Temazepam	Restoril	15–30	8–20	Conjugation	
Triazolam	Halcion	.125–.5	2–6	Oxidation	

Perioperative Hypnotic

Generic Name	*Trade Name*	*Usual Dose (mg/day)*	*Half-life (hours)*	*Transformation Pathway*	*Metabolites (half-life, hours)*
Midazolam	Versed	1–2.5 mg/ml	1–4	Oxidation	Hydroxymethylmidazolam

NOTE: The half-life of a compound is the amount of time that must pass for the level of that agent in the plasma to be reduced by half.

[a]Oxazepam is also a metabolite of diazepam, clorazepate, prazepam, halazepam, and temazepam.

SOURCES: *Drug Facts and Comparisons.* (1994). St. Louis, MO: Facts and Comparisons. Greenblatt, D. J. (1991). Benzodiazepine hypnotics: Sorting the pharmacokinetic facts. *Journal of Clinical Psychiatry, 52* (Suppl. 9), 4–10. Greenblatt, D. J., & Shader, R. I. (1987). Pharmacokinetics of antianxiety drugs. In H. Y. Meltzer (Ed.), *Psychopharmacology: The Third Generation of Progress.* NY: Raven Press.

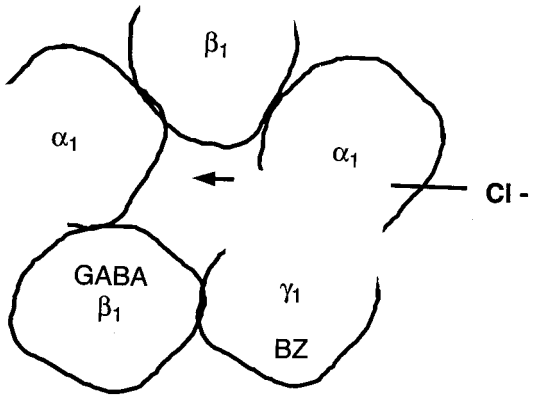

Figure 2
Schematic of one possible form of the $GABA_A$ receptor-chloride ion channel complex. Chloride ions enter through the center channel formed by alpha (α), beta (β), and gamma (γ) subunits. GABA receptors on β subunits regulate the flow of chloride ions through the channel. The activity of GABA receptors can be modulated by benzodiazepine receptors located on the γ subunit.
SOURCE: Figure created by Rebecca Bulotsky.

release of GABA to affect the movement of chloride, whereas at higher doses barbiturates, through their own direct effects, can act to increase chloride inflow into cells.

The $GABA_A$ receptor complex is composed of alpha, beta, and gamma subunits (Zorumski & Isenberg, 1991). Each subunit consists of a chain of twenty to thirty amino acids. Multiple subtypes of the alpha, beta, and gamma subunits have been shown to exist, and the types of subunits that form a single receptor complex appear to vary in different areas of the central nervous system. Some researchers have proposed that different drugs selectively interact with benzodiazepine receptors composed of a particular kind of α subunit, thereby leading to differences in drug effects. Although there is little evidence to support this hypothesis, future research should clarify the issue.

New compounds, such as the imidazopyridines, have been developed that act at the benzodiazepine receptor but are chemically distinct from the benzodiazepines. Zolpidem is an imidazopyridine used in clinical practice as a hypnotic agent. Other new drugs have been synthesized that can stimulate the benzodiazepine receptor but do not produce the maximal effects that result from the administration of higher doses of benzodiazepines. These drugs are classified as partial AGONISTS. The drug abecarnil, which belongs to the beta-carboline class of compounds, is an example of such an agent that has been used experimentally to treat anxiety.

Flumazenil is a benzodiazepine derivative that has no activity of its own but acts to antagonize the actions of benzodiazepines at the benzodiazepine receptor. It is used to reverse the effects of these drugs during anesthesia or in benzodiazepine overdoses. Other compounds, including some of the beta-carbolines such as methyl-beta-carboline-3-carboxylate, act on the benzodiazepine receptor to produce effects that are opposite to those of benzodiazepines (Kardos, 1993; Zorumski & Isenberg, 1991). Administration of these inverse agonists can lead to the appearance of anxiety and convulsions.

THERAPEUTIC USE

Benzodiazepines are used for a variety of therapeutic purposes. Anxiety is the experience of fear that occurs in a situation where no clear threat exists. Numerous studies have demonstrated that anxiety disorders, including generalized anxiety disorder and many phobias, can be treated effectively with benzodiazepines. Panic disorder is a psychiatric illness in which patients experience intense sporadic attacks of anxiety often accompanied by the avoidance of open spaces and other places or objects that are associated with panic. High-potency benzodiazepines such as alprazolam (Xanax) or clonazepam (Klonopin) can prevent the occurrence of panic attacks in patients suffering from panic disorder. Flurazepam (Dalmane), triazolam (Halcion), and the other benzodiazepines listed in Table 1 are used in the treatment of insomnia and other sleep disorders. All rapidly acting benzodiazepines marketed in the United States have hypnotic effects. Classification of a benzodiazepine as a hypnotic is often more a marketing strategy than it is a decision based on pharmacologic differences among the class of drugs.

Status epilepticus is a seizure or a series of seizures that occurs over an extended period of time. This condition can lead to irreversible brain damage and is often successfully managed by the intravenous infusion of diazepam. Clonazepam is used either alone or in combination with other anticonvulsant medications to treat absence seizure and other types of seizure disorders. Clorazepate is used

to control some types of partial seizures—that is, seizures that occur in a limited area of the brain. The increase in central nervous system excitability, seizures, and anxiety that may appear during alcohol withdrawal can be treated with any benzodiazepine. Midazolam (Versed) is a benzodiazepine that is rapidly metabolized in the body and is used to help induce anesthesia during surgical procedures. The skeletal-relaxant properties of benzodiazepines make them useful for the treatment of back pain due to muscle spasms.

ADVERSE EFFECTS

Benzodiazepines have proven to be exceptionally safe agents. The dose at which these agents are lethal tends to be exceedingly high. Fatalities are more apt to occur when these drugs are taken in combination with other central nervous system depressant agents such as ethanol. Sedation is a common adverse effect associated with benzodiazepine use. Light-headedness, confusion, and loss of motor coordination may all result following the administration of benzodiazepines. MEMORY impairment may be detected in individuals treated with benzodiazepines, and this effect may prove to be particularly troublesome to ELDERLY patients who are experiencing memory-related problems. PSYCHOMOTOR impairment can be hazardous to individuals when they are driving. This problem can be exacerbated in individuals who consume ethanol while they are being treated with benzodiazepines. Hypnotic agents that are converted into active metabolites that are slowly eliminated from the body, such as flurazepam, may produce residual daytime effects that can impair tasks such as driving. The adverse effects of benzodiazepines on performance tend to be more of a problem in elderly people than in younger individuals. Patients with cirrhosis, a liver degenerative disease, are also more likely to experience benzodiazepine toxicity than are those with normal liver function. The appearance of the adverse effects associated with benzodiazepine administration in both elderly people and in cirrhotic patients can be minimized by treating them with agents such as oxazepam and lorazepam, which tend not to accumulate in the blood because they are excreted rapidly into the urine as glucuronide conjugates.

A small number of patients may exhibit paradoxical reactions when they are treated with benzodiazepines (Rall, 1990). These may include low-level anxiety, restlessness, depression, paranoia, hostility, and rage. Sleep patterns may be disrupted by benzodiazepine administration, and nightmares may increase in frequency. Benzodiazepines suppress two stages of the sleep cycle—the stage of deepest sleep, stage IV, and the rapid eye movement (REM) stage in which dreaming occurs.

TOLERANCE AND PHYSICAL DEPENDENCE

TOLERANCE to a drug involves either a decrease in the effect of a given dose of a drug during the course of repeated administration of the agent or the need to increase the dose of a drug to produce a given effect when it is administered repeatedly. Chronic treatment of animals with benzodiazepines leads to a reduction in potency of these agents as enhancers of chloride ion uptake. These effects at the cellular level are paralleled by the appearance of tolerance to the sedative effects of benzodiazepines. Tolerance also develops to the impairment of motor coordination that is produced by these drugs. Limited evidence suggests that the antianxiety effects of benzodiazepines may not diminish with time, or at the very least that benzodiazepines retain their effectiveness as antianxiety agents for several months.

PHYSICAL DEPENDENCE results from adaptive changes in the nervous system that may be related to the development of tolerance. Dependence of this sort can be detected by the appearance of a characteristic abstinence or WITHDRAWAL syndrome when chronic administration of a drug is either abruptly discontinued or after the administration of an antagonist to the drug that has been taken for a prolonged period of time (Ciraulo & Greenblatt, in press). Individuals who are treated chronically with benzodiazepines may exhibit signs and symptoms of withdrawal when the administration of these drugs is discontinued. Minor symptoms of withdrawal include ANXIETY, insomnia, and nightmares. Less common and more serious symptoms include psychosis, death, and generalized seizures. Signs of withdrawal may become evident twenty-four hours after the discontinuation of a benzodiazepine that is rapidly eliminated from the blood. Peak abstinence symptoms may not appear until two weeks after discontinuation of a benzodiazepine that is removed from the body slowly. Some of

the symptoms that appear after benzodiazepine treatment is discontinued may be due to the recurrence of the anxiety disorder for which the drug had been originally prescribed.

In animals, the severity of withdrawal can be directly related to the dose and length of time of administration of a benzodiazepine. This kind of relationship has been harder to demonstrate in clinical studies. Many patients who are treated with benzodiazepines for prolonged periods of time may experience at least some symptoms of withdrawal, but most of these individuals should not be viewed as benzodiazepine "addicts" because they have relied on their medications for medical reasons, have taken the medications as directed by their physicians, and will not continue to compulsively seek out benzodiazepines once their prescribed course of treatment with these medications has been discontinued. The intensity of abstinence symptoms that may be seen in patients who are physically dependent on benzodiazepines can be markedly reduced if patients are allowed to gradually taper off their medications. There may be a risk of physical withdrawal from benzodiazepines in some patients who abruptly stop the medication following as few as four weeks after treatment. Patients who discontinue taking rapidly metabolized hypnotic drugs such as triazolam may be at risk for experiencing rebound insomnia, even if they have been under treatment for a few days to one week. Serious problems associated with benzodiazepine withdrawal are more likely to be a problem for patients who have been treated with high doses of these medications for four or more months.

ABUSE AND DEPENDENCE

Although no consensus exists as to the definition of drug addiction, diagnostic criteria for drug abuse and dependence have been developed by both the American Psychiatric Association and the World Health Organization. Drug abuse can be viewed as the use of a pharmacological substance in a manner that is not consistent with existing medical, social, or legal standards and practice. Alternatively, drug abuse has been defined in the DIAGNOSTIC AND STATISTICAL MANUAL of Mental Disorders of the APA as involving a "maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to repeated use" (American Psychiatric Association, 1994). Abuse of drugs may involve the use of drugs for recreational purposes—that is, drugs are administered to experience their mood-elevating (euphoric) effects. For some individuals, self-administration of drugs for these purposes may lead to compulsive drug-seeking behavior and other extreme forms of drug-controlled behavior. These behavior patterns may become further reinforced by the effects of withdrawal symptoms that dependent individuals attempt to reduce by the administration of the abused agent. The APA specifies that individuals can be classified as being drug dependent if they exhibit signs of drug tolerance, symptoms of withdrawal, cannot control their drug use, feel compelled to use a drug, and/or continue to use a substance even if the consequences of this use may prove harmful to them (American Psychiatric Association, 1994).

Abuse of drugs may sometimes represent self-medication. COCAINE and AMPHETAMINE users sometimes rely on benzodiazepines to relieve the jitteriness that may result from the administration of PSYCHOMOTOR STIMULANTS. Some abusers of benzodiazepines may be medicating themselves with these agents to treat preexisting conditions of anxiety and DEPRESSION.

The ABUSE LIABILITY of benzodiazepines—that is, the likelihood that they will be misused—has been assessed in studies of the tendency of either human beings or animals to administer these agents to themselves and studies of the subjective effects that result from the administration of different benzodiazepines. When provided access to cocaine and other psychomotor stimulants, animals will consistently self-administer these agents at high rates over time. Primates will intravenously self-administer benzodiazepines at moderate rates that are below those observed for the administration of BARBITURATES or COCAINE. This finding and the results of a number of additional animal studies indicate that the benzodiazepines have a lower abuse liability than do the barbiturates or the psychomotor stimulants (Ciraulo & Greenblatt, in press).

Individuals with a history of sedative-hypnotic abuse will self-administer triazolam and diazepam (Roache & Griffiths, 1989). In contrast, normal volunteers do not prefer diazepam to placebo. Subjective responses to drugs can be assessed through the use of instruments such as the Addiction Research Center Inventory-Morphine Benzedrine Group Scale and the Profile of Moods States that

help to standardize the reports of subjects concerning their drug-induced experiences. Investigations in which subjective responses of normal subjects to benzodiazepine administration have been assessed indicate that these agents tend not to produce mood elevations in normal populations. On the other hand, individuals with a history of either alcoholism or sedative-hypnotic abuse are more likely to experience euphoria after the administration of a single dose of either diazepam or other benzodiazepines. Adult children of alcoholics experience mood elevation after the ingestion of either alprazolam or diazepam, thus suggesting that these individuals may have a predisposition to benzodiazepine abuse.

Studies suggest that benzodiazepines are less likely to be abused than the barbiturates, opiates, or psychomotor stimulants, but that they carry more risk for abuse than do medications such as the antianxiety agent buspirone or drugs that have sedating effects such as the antihistamine diphenhydramine (Preston et al., 1992). There also may be differences among the benzodiazepines themselves. Some authorities believe that diazepam has greater abuse liability than halazepam, oxazepam, chlordiazepoxide, or clorazepate, although others believe that there is little difference among them. Diazepam, lorazepam, alprazolam, and triazolam all produce mood effects that are similar to those of known drugs of abuse. The rate at which these drugs reach the brain after administration may be a major determining factor in the onset of euphoria or pleasant effects associated with abuse. Inferences about abuse potential are made on the basis of subjective effects and self-administration in drug abusers and alcoholics. Many experts question the applicability of these findings to the general population.

Studies that accurately reflect the extent of benzodiazepine abuse in the United States are not available. A survey of American households produced by the National Institute on Drug Abuse suggested that the nonmedical use of tranquilizers was not a major health problem (Ciraulo & Greenblatt, in press). Only 2.4 percent of individuals between the ages of 18 and 24 and 1.3 percent of survey respondents who were older than 26 reported using tranquilizers for nonmedical purposes. This type of survey does not take into account benzodiazepine usage among groups such as homeless people, prisoners, and migrant workers, and so it cannot convey a complete picture of how benzodiazepines are misused at the nationwide level (Cole & Chiarello, 1990).

Benzodiazepines are frequently used by individuals who abuse other drugs, but they are rarely used as either initial or primary drugs of abuse. Benzodiazepine abusers often take these drugs in combination with other agents. In Scotland, drug abusers have often injected temazepam in combination with the OPIOID drug BUPRENORPHINE (Ruben & Morrison, 1992). Large percentages of methadone-clinic patients have urine tests that are positive for benzodiazepines. METHADONE-MAINTENANCE patients have indicated that diazepam, lorazepam, and alprazolam can produce desirable pleasurable effects (Sellers et al., 1993). Whether methadone patients use benzodiazepines to increase the effects of methadone or as self-medication for anxiety is not clear.

The percentage of alcoholics admitted for treatment who also concurrently use benzodiazepines ranges between 12 to 23 percent. High rates of benzodiazepine abuse have been found in alcoholics who have experienced failure in treatment programs for alcohol abuse. Clinical experience suggests that benzodiazepine abuse occurs with the greatest frequency in alcoholics with severe dependence and in alcoholics who abuse multiple types of drugs.

Individuals with a history of either alcohol abuse or alcohol dependence often have anxiety disorders. The issue of treating alcoholics with benzodiazepines is complex because some of these patients can take the medications without abusing them or relapsing to alcohol use whereas others take them in higher than prescribed doses and find that their desire to drink alcohol is increased.

SUMMARY

A large number of benzodiazepines are available for clinical use. These agents all share a set of pharmacologic properties that result from enhanced chloride flux at the $GABA_A$-receptor complex, which in turn results in the inhibition of neuronal activity in many regions of the central nervous system. Differences in activity among the benzodiazepines appear to be related primarily to differences in rates of absorption and metabolism, although recent research has suggested that intrinsic activity at benzodiazepine receptor subtypes

also may influence drug effects. These drugs have been used extensively to treat anxiety, insomnia, seizures, and other disorders. They are safe and effective and their use has rarely been associated with irreversible adverse effects. Both physical and psychological dependence may be problematic for some individuals who are treated on a long-term basis with these agents or who have abused alcohol or other drugs.

(SEE ALSO: *Addiction: Concepts and Definitions; Benzodiazepines: Complications; Sleep, Dreaming, and Drugs*)

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BENZODIAZEPINES: COMPLICATIONS As medicines, BENZODIAZEPINES have been widely used—as tranquilizers, to allay anxiety. Until the 1990s, they were believed to be both effective and extremely safe; however, beginning in the early 1980s, problems with these drugs started to become evident. Currently, the medical profession in many countries is trying to inculcate a cautious attitude toward their prescription and use. Lay people and the media have also become increasingly critical of the widespread use of these medicines for apparently trivial indications. To understand these problems, some aspects of the different types and effects of these medicines will be outlined.

WHAT ARE BENZODIAZEPINES?

These medicines are used to lessen a patient's anxiety; they include such drugs as CHLORDIAZEPOXIDE (Librium), diazepam (Valium), lorazepam (Ativan) and oxazepam (Serenid; Serox). The term *benzodiazepine* describes a basic chemical structure. Some, like diazepam, are long acting and can be taken once daily; others, like lorazepam and alprazolam (Xanax), need to be taken more often. Most sleeping tablets (hypnotics) are benzodiazepines, and these include short-acting drugs such as triazolam (Halcion), medium-acting drugs such as temazepam (Restoril), and long-acting drugs such as flurazepam (Dalmane) and nitrazepam (Mogadon).

Other medicines are used in psychiatry, such as ANTIDEPRESSANTS, ANTIPSYCHOTICS, and lithium. These have effects that differ from the benzodiazepines, effects both therapeutic and unwanted.

WHAT DO BENZODIAZEPINES DO?

Tranquilizers promote calming, soothing, and pacifying—without sedating or depressant effects. They are effective in lessening ANXIETY whatever

its context. Thus, they are useful in treating *generalized anxiety*, which is often quite severe and comes on without apparent cause. Tranquilizers can also be used to deaden the upset of *normal anxiety*, the anxiety felt by people under stress, feeling threatened by life's problems. In these instances, the reasons for feeling anxious are clear, the degree of anxiety seems in line with the stress experienced—but despite this, help is sought for the symptoms. Unfortunately, the borderline between the medical disorder of clinical generalized anxiety and the normal response to stress is not always clear. The professional consulted will usually try to make the distinction and avoid using tranquilizers to treat people upset by adverse circumstances—thereby "medicalizing" everyday social and personal problems.

Similar considerations apply to the use of benzodiazepines as sleeping tablets. Short-term use of these drugs—for example, for disturbed sleep with jet travel across time zones, severe stress, or shift work—is generally accepted. Long-term use in the chronically poor sleeper is not usually encouraged, however.

Benzodiazepines can also be used as sedatives before surgical operations, as light anesthetics during operations, and to lessen muscle spasms, such as occur with sports injuries. Some benzodiazepines can be used to treat some forms of epilepsy. Benzodiazepines are prescribed mainly by general family practitioners, although they vary greatly in how often they use these medicines. Some still prescribe them widely, some hardly at all. Other doctors who use benzodiazepines include psychiatrists, orthopedic specialists, and gynecologists.

HOW MUCH ARE THEY USED?

An international survey at the beginning of the 1980s showed that tranquilizers and sedatives of any type had been used at some time during the previous year by 12.9 percent of U.S. adults, 11.2 percent in the United Kingdom (U.K.), 7.4 percent in the Netherlands, and 15.9 percent in France. Persistent long-term users comprised 1.8 percent of all U.S. adults, 3.1 percent in the U.K., 1.7 percent in the Netherlands and 5.0 percent in France. The proportion of repeat prescriptions for tranquilizers has increased steadily since about 1970 in many countries, the U.K. in particular. This suggests that fewer people are being newly started on tranquilizers but that a large group of long-term users is accumulating. People starting tranquilizers have at least a 10 percent chance of going on to long-term use, that is for more than 6 months. Some of these chronic users have chronic medical or social problems, and the tranquilizer blunts the unpleasant feelings of tension, anxiety, insomnia and, to a lesser extent, depression.

UNWANTED (SIDE) EFFECTS

Side effects are reactions to drugs that are not therapeutic or helpful, and they are therefore unwanted. The most common side effects from taking benzodiazepine are drowsiness and tiredness, and they are most marked within the first few hours after large doses. Other complaints of this type include dizziness, headache, blurred vision, and feelings of unsteadiness. The elderly are particularly sensitive to tranquilizers and may become unsteady on their feet or even mentally confused.

The feelings of drowsiness are, of course, what is wanted with a sleeping tablet. With the longer-acting benzodiazepines and with higher doses of medium-duration or with short-acting drugs, drowsiness can still be present the morning after taking a sleeping tablet; the drowsiness may even persist into the afternoon. The elderly are more likely to experience such residual, or "hang-over," effects.

As well as these feelings of sedation, special testing in a psychology laboratory indicates that alertness, coordination, performance at skilled work, mental activities, and memory can all be impaired. Patients should be warned about this, and advised not to drive or operate machinery, at least initially until the effects of the benzodiazepine can be assessed and the dosage adjusted if necessary. If driving is essential—that is, to the patient's livelihood—small doses of the benzodiazepines should be taken at first and the amount built up gradually under medical supervision. Judgment and memory are often impaired early in treatment, so important decisions should be deferred.

As with many drugs affecting the brain, benzodiazepines can interact with other drugs, especially ALCOHOL. People taking tranquilizers or hypnotics should not also drink alcoholic beverages. Other drugs whose effects may be enhanced include antihistamines (such as for hay fever), painkillers, and

antidepressants. Cigarette smoking may lessen the effect of some benzodiazepines.

Patients taking benzodiazepines may show so-called paradoxical responses—that is to say, the effects produced are the opposite of those intended. Feelings of anxiety may heighten rather than lessen, insomnia may intensify, or, more disturbing, patients may feel hostile and aggressive. They may engage in uncharacteristic criminal activities, sexual improprieties or offenses such as importuning or self-exposure, or show excessive emotional responses such as uncontrollable bouts of weeping or giggling. All these are signs of the release of inhibitions, and they are also characteristic of alcohol effects in some people. Although these paradoxical effects may not last long, it is better to stop the benzodiazepine.

Benzodiazepines can affect breathing in individuals who already have breathing problems, such as with bronchitis. Other side effects that may be occasionally encountered include excessive weight gain, rash, impairment of sexual functioning, and irregularities of menstruation. Benzodiazepines should be avoided during pregnancy whenever possible, as there may be a risk to the fetus. Given during childbirth, benzodiazepines pass into the unborn infant and may depress the baby's breathing after birth. They also pass into the mother's milk and may sedate the suckling baby too much. Many people have taken an overdose of a tranquilizer as a suicidal attempt or gesture. Fortunately, these drugs are usually quite safe and the person wakes up unharmed after a few hours' sleep.

SIDE EFFECTS VERSUS MAIN EFFECTS

There are more subtle side effects of benzodiazepines, effects that interfere in various ways with the treatment of the anxiety or sleep disorder. The benzodiazepine lessens the symptom but does not alter the underlying problem—say, an unhappy marriage or a precarious job. Indeed, by lessening the symptoms, the individual may lose his or her motivation to identify, confront, and tackle the basic problems. Giving benzodiazepine medicalizes the problem by making the nervous or sad person into a patient, implying that there is something physically wrong. Finally, some events like bereavement need "working through"—typically by grieving—but benzodiazepines can stop this normal process and actually prevent the bereaved individual from coming to terms with loss.

LONG-TERM EFFECTS OF BENZODIAZEPINES

It is not clear whether benzodiazepines and hypnotics continue to be effective after months or years of daily use. Undoubtedly, many patients believe that they continue to benefit in being less anxious, or in sleeping better. The effect of the drug may be more to stop the anxiety or insomnia that follows withdrawal, however, than to combat any continuing, original anxiety. Most of the side effects lessen over time, a process known as *tolerance*. Some impairments, however, such as memory disturbances, may persist indefinitely, but patients usually come to terms with this—for example, by resorting to written reminders.

REBOUND

Rebound occurs when stopping the drug makes the underlying condition worse. Most is known about rebound in insomnia. Sleeping tablets may improve sleep by inducing it more rapidly, making it sounder, and prolonging it. When the sleeping tablet is stopped, rebound may occur on the following night or two, with the insomnia being worse than ever. Eventually, the rebound insomnia subsides, but the patient may have been so distressed as to resume medication, thereby running the risk of indefinite use. The risk of rebound is greatest with short-acting benzodiazepines, especially in higher dose.

A similar problem follows stopping a daytime tranquilizer, particularly lorazepam. Anxiety and tension rebound to levels higher than those experienced on treatment and often higher than the initial complaints. Tapering off the tranquilizer over a week or two lessens or avoids this complication. Rebound may even be seen in the daytime between doses of tranquilizer. The patient, increasingly anxious as the effect of the earlier dose wears off, watches the clock until his or her next dose is due. Rebound may also occur later in the day after taking a short-acting sleeping tablet the night before.

WITHDRAWAL

In withdrawal, symptoms occur which the patient has not previously experienced. They come on

a day or two after stopping alprazolam or lorazepam, after a week or so on stopping diazepam or chlordiazepoxide. The symptoms rise to a crescendo and then usually subside over two to four weeks. In an unfortunate few, the symptoms seem to persist for months on end—sometimes called the *post-withdrawal syndrome*. The existence of this condition is disputed by some doctors, who ascribe the symptoms to return of the original anxiety for which the drug was given.

Patients commonly experience bodily symptoms of anxiety such as tremor, palpitations, dry mouth, or hot and cold feelings. Insomnia is usually marked. Some complain of unpleasant feelings of being out of touch with reality or with their own bodies. Severe headaches and muscle aches and pains can occur, sleep is greatly disturbed, appetite is lost as is several pounds of weight. Disturbances of perception are characteristic of benzodiazepine withdrawal and include intolerance to loud noises or bright lights, numbness or pins and needles, unsteadiness, a feeling of being in motion (as on a ship at sea), and a sensing of strange smells and tastes. Some people become quite depressed; rarely, some experience epileptic fits or a paranoid psychosis (with feelings of persecution and loss of contact with reality).

HOW BIG IS THE PROBLEM?

The withdrawal symptoms are evidence of physical dependence—that is, the body has become so used to the effects of the benzodiazepine that it cannot manage without. About a third of long-term (over a year) steady users show withdrawal, even when the tranquilizer or hypnotic is tapered off. Some users have tried to stop and have encountered problems. Many others have never tried to stop and so are unaware whether they are dependent. Because these people continued to take the doses prescribed by their doctors, the medical profession was reluctant for a long time to admit the scale of the problem—perhaps 500,000 people dependent on tranquilizers in the U.K. alone. In addition, the similarity between some withdrawal symptoms and features of the original anxiety has led to confusion in the mind of both the patient and the doctor. True withdrawal symptoms, however, arise at a predictable time after stopping the benzodiazepine and are new experiences for the patient; the old anxiety and insomnia symptoms are familiar to the patient and may return at any time, depending on external stresses.

HOW TO WITHDRAW

Essentially, the patient must be prepared for withdrawal by being told what to expect; he or she should be taught other ways of combatting anxiety; and withdrawal should be by graded tapering off the dose over six to twelve weeks, occasionally longer. Many people experience little or no upset, a few undergo much distress. Sometimes substituting diazepam in the lorazepam or alprazolam user helps. Antidepressants may be needed if the patient becomes very depressed, but by and large, other drugs are unhelpful.

Family and social support is essential. Usually the family doctor can supervise the withdrawal quite safely, but occasionally specialist advice is sought. A self-help group may provide useful continued advice and support.

It is important that tranquilizers are never stopped abruptly. There is a greatly increased risk of severe complications such as seizures or convulsions.

ABUSE OF TRANQUILIZERS

Only a few patients prescribed benzodiazepines push the dose up above recommended levels. If this happens, the user may become intoxicated, with slurred speech and incoordination. Some people with alcohol problems also abuse benzodiazepines. Intravenous (IV) injection of benzodiazepines and hypnotics has become an increasing problem and has led to controls on these drugs concerning manufacture and prescription in various countries, including the United States and the U.K. Some addicts abuse benzodiazepines alone; others combine it with heroin-type drugs. Injection of benzodiazepines can result in clotting of the veins. It also carries the risk of getting infectious diseases from sharing dirty syringes, such as hepatitis and the human immunodeficiency virus (HIV or the AIDS virus).

ALTERNATIVES TO THE TRANQUILIZERS

Dissatisfaction with the benzodiazepine tranquilizers and hypnotics has led to numerous initiatives to find better alternatives. Some drugs have

been developed that are better benzodiazepines, in that they are less sedative and perhaps less likely to induce dependence. Others are chemically not benzodiazepines but share many of their properties, both therapeutic and unwanted. Other compounds seem to act in a totally different way in the brain and are less sedative and probably much less likely to induce dependence. One such compound—buspirone (Buspar)—has been available for a few years, but many others are in the process of development. Finally, interest has been rekindled in the use of other types of older drugs to treat anxiety; examples include the antihistamines and the beta blockers.

Problems with the benzodiazepines has led to a reevaluation of the whole role of prescribed medicines in the management of anxiety, insomnia, and stress-related disorders. Numerous nondrug methods have been developed and improved, among them relaxation training; cognitive therapy, in which patients learn to think less anxious thoughts; behavior therapy, in which the patient learns to confront stressful situations; and sleep counseling. Alternative medicine, like ACUPUNCTURE, is enjoying a vogue and helps some anxious people.

CONCLUSIONS

Hailed as wonder drugs, prescribed widely and for long periods of time, the benzodiazepines have now been shown to be problematic medicines with undoubted benefits but definite risks. For short-term treatment in the severely anxious and sleepless, they are still useful—although other drugs are beginning to supplement and even supplant them. For the bulk of anxious people, though, nondrug treatments are increasingly popular.

(SEE ALSO: *Addiction: Concepts and Definitions; Complications; Iatrogenic Addiction; Sleep, Dreaming, and Drugs; Tolerance and Physical Dependence; Withdrawal*)

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BENZOYLECOGNINE Cocaine is metabolized by plasma and liver enzymes (cholinesterases) to water-soluble metabolites that are excreted in the urine. The two major metabolites are benzoylecognine and ecognine methyl ester, with only benzoylecognine reported to have behavioral activity. Since COCAINE has a relatively short half-life and may only be present in the urine for twenty-four to thirty-six hours, benzoylecognine levels in urine are useful markers of cocaine use, because it

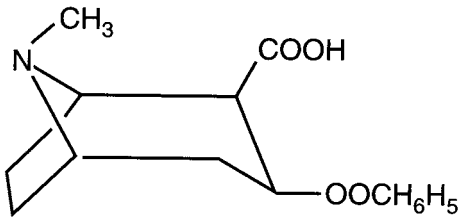

Figure 1
Benzoylecognine

is present for a longer time in urine, two to four days, depending on the quantity of cocaine ingested. Assays for this metabolite are frequently employed in treatment programs, to evaluate compliance with the program, and in workplace drug testing to indicate cocaine use. Under these conditions, it is important to keep in mind that benzoylecognine in the urine is an indication of prior cocaine use, but reflects neither current use nor impairment.

(SEE ALSO: *Cocaethylene; Drug Testing and Analysis*)

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BETEL NUT Betel nut, the seed of the betel palm (*Areca catechu*), is one of the most widely used substances in areas of the western Pacific and parts of Africa and Asia. It is prepared with other substances as a mixture for chewing and is used as a mild stimulant by more than 200 million people.

References to betel nut appear in ancient Greek, Sanskrit, and Chinese texts from more than a century B.C. Ancient historic documents of Ceylon refer to its use, and its prevalence in Persia by 600 A.D. is documented by Persian historians. Its use in different parts of the Arab world by the eighth and ninth centuries is also well documented, and it had become an important aspect of the economy and social life in India, Malaysia, the Philippines, and New Guinea. Betel was probably brought to Europe by Marco Polo, around 1300; it soon proved to be an important commodity in the western Pacific and a source of tax revenue for the Dutch in the mid-1600s.

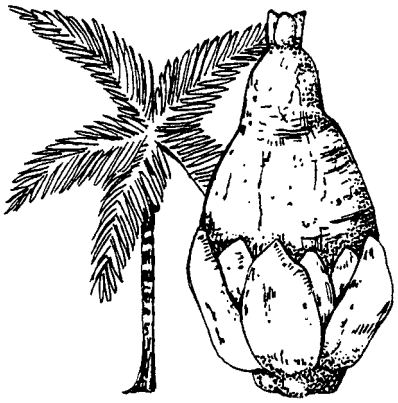

Figure 1
Betel Palm and Betel Nut

PATTERNS OF USE

The mottled brown-and-gray betel seeds are gathered before they ripen during the period between August and November. They are boiled in water, cut into slices and dried in the sun, becoming dark brown or reddish in color. This betel seed, or nut, becomes the primary ingredient in the betel nut chewing mixture ("quid"), which is made up of several ingredients. While the component substances differ in different parts of the world, all the preparations contain fresh chunks or dried powdered forms of the betel nut. Mixtures prepared for children frequently contain only the husk of the nut, while the full strength form for adults always has the nut itself.

The second ingredient of the quid is usually a form of peppermint or mustard, or the leaf, bean and/or bark of the shrub-like or climbing pepper plant vine (*Piper betel*). The third component is slaked lime, which is usually produced from limestone or by burning sea shells or coral stones in the presence of water. This process produces calcium hydroxide, usually used as a white powder. While all betel-nut quids contain some of the three main components, other ingredients, such as spices, dyes, and aromatics are frequently added. In India, tobacco is mixed with the quid. The combination of nut, mustard or vine, lime, and other ingredients create an alkaline, bitter-tasting mixture that is chewed, forming a red paste which stains the teeth, mouth, gums, and lips, and generating large amounts of saliva. Like tobacco chewers, betel-nut chewers spit out the excess juices.

Some habitués chew betel nut all day long. Others use it as part of social custom, not unlike the use of KAVA, or of the consumption of ALCOHOL in Western countries. In some areas of the world, such as Papua New Guinea, betel-nut mixtures are often offered as a before-dinner "appetizer" or as an after-dinner treat. Like kava, in many places, sharing betel-nut mixture is important in courtship and marriage customs and in establishing friendships.

ACTIVE INGREDIENTS

The major active ingredient of betel nut is arecoline, present in a concentration estimated to be 0.25 percent. The mixture also contains small amounts of pilocarpine and muscarine. These three ingredients are all natural plant products that act in the body in a manner similar to the normal brain NEUROTRANSMITTER acetylcholine. In the presence of calcium hydroxide, arecoline is also converted to another psychoactive substance, aredaidine.

Chewing betel nut produces immediate effects that in some ways resemble those of NICOTINE, but which are likely to continue for hours. These include euphoria and feelings of general arousal and activation, perceived by the user as a decrease in tiredness and a blunting of feelings of irritability.

Other prominent effects are also related to the acetylcholine-like actions. These include sweating, increased production of saliva, and an increase in breathing rate and lacrimation (tearing of the eyes). Effects on the digestive tract include a decrease in appetite and, especially if the drug is taken on an empty stomach, diarrhea. All these effects can be blocked by atropine, a type of anti-acetylcholine drug.

Some of the active ingredients in betel nut are used in modern pharmacological treatment in the Western world. Betel-nut preparations have been used in Western society as a purgative and in veterinary medicine as an agent for treating worm infestation in animals. Probably the most interesting use appears around 1842, when betel nut was included in toothpastes in England. It was touted as an important way to prevent decay, a claim that may or may not be accurate, although much larger doses than those found in toothpastes would be required to really be clinically effective. It was also said that this ingredient would help strengthen the tooth enamel and remove tartar, claims of questionable value. In view of the fact that betel, as it is commonly used, stains the teeth dark red to black, is thought to cause tooth decay, and can cause serious lesions of the mouth and throat, it is curious that it should have appeared in Western society in preparations for dental care.

SOME DANGERS

The acetylcholine-type drugs, especially muscarine, can be deadly when taken in high doses. In fact, muscarine is the active ingredient causing some forms of lethal mushroom poisoning, but it is unlikely that the mixture of any of the plant products in betel-nut preparations are potent enough to cause lethal overdose. Regular "recreational" use of betel nut is, however, responsible for a number of adverse health consequences that can contribute to the risk for early death.

The most prominent dangers associated with betel-nut chewing are probably the result of a combined effect of the active ingredients and the lime on the gums. The first and most frequently observed physical changes are white plaques appearing on the mucosal lining of the mouth or on the tongue. These are precancerous lesions (leukoplakia) that often lead to the development of very aggressive and serious tumors (squamous cell carcinoma), which can subsequently invade muscles and bone tissue. The prevalence of this cancer among regular betel-nut users is estimated to be as high as 7 percent. Potentially lethal cancers may also develop in the esophagus. Chronic use may also cause oral submucous fibrosis—a form of fiber formation (fibrosis) that usually starts just beneath the gums and may involve the back of the throat and the pharynx. The problem is estimated to be seen in at least mild form in up to 50 percent of chronic betel-nut chewers. This condition usually has a very slow onset, and if use continues it is irreversible, untreatable, and likely to become progressively more severe. The major finding involves a loss of elasticity of the tissue lining the mouth, which causes stiffness that can become so severe as to interfere with eating. Associated problems are a burning sensation in the mouth, ulcers or blisters on the lining of the mouth, decreased sense of taste, and dryness of the mouth lining.

There is little doubt that betel-nut substance can produce fairly intense psychological dependence. Individuals can develop a pattern of constant use, feeling unhappy and incomplete if they cannot get their betel nut. They are also likely to feel they cannot work properly without it, and may spend a great deal of money and time obtaining and using betel-nut mixtures. It is not clear, however, that there is a prominent and identifiable form of physical withdrawal associated with cessation of use.

Betel-nut consumption can be viewed as a public-health hazard in parts of the world where its use is prevalent, because, at least theoretically, the habit of spitting the juice on the street can increase the spread of diseases such as tuberculosis.

(SEE ALSO: *Plants, Drugs from*)

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BETTY FORD CENTER *See* Treatment Programs/Centers/Organizations: An Historical Perspective

BHANG This is one of the many names given to the HEMP plant, *Cannabis sativa*, and its products. Bhang is of Hindi origin (from *bhãg*, which came into English about 1563) and refers to the leaves and flowering tops of uncultivated hemp plants. In 1895, the Indian Hemp Commission took the position that bhang was not a major health hazard. Bhang is taken in a beverage in India called *thandaii*, may be served in sweetmeats, or is used in making ice cream. It is often served at weddings or religious festivals and is freely available from sidewalk stands in the major cities. Generally, in India, the use of bhang and other cannabis products has been considered lower class. Probably as a result of continuing British-based influence, the upper-class drugs are alcohol and opium.

(SEE ALSO: *Cannabis Sativa; Marijuana; Plants, Drugs from*)

LEO E. HOLLISTER

BLOOD ALCOHOL CONCENTRATION, MEASURES OF The first analytical methods for measuring ALCOHOL (ethanol) in blood and other body fluids were developed in the nineteenth century. Although by modern standards these pioneer efforts were fairly crude, they were sufficiently reliable to establish a quantitative relationship between blood-alcohol concentration (BAC) and the various signs and symptoms of inebriation. A significant advance in methodology came in 1922 when Erik M. P. Widmark published his micromethod for analyzing ethanol in specimens of capillary blood.

Blood was drawn by pricking a fingertip or earlobe. The specimen for analysis, 100–150 milligrams, was collected with specially prepared S-shaped glass capillaries that contained a thin film of potassium oxalate and sodium fluoride on the walls of the tube. In Widmark's day, the small amounts (aliquots) of blood needed for each analysis could be measured more accurately by weight than by volume, since constriction pipettes were not yet available. Widmark therefore weighed the amount of blood required to the nearest milligram (0.001 g) with the aid of a torsion balance. The results of ethanol determinations were then reported in terms of mass per mass units, actually milligram of ethanol per gram of whole blood (mg/g), sometimes referred to as per mille (meaning, parts per thousand). This way of reporting BAC survives today in Scandinavian countries where Widmark's method became widely used for legal purposes.

Widmark's micromethod of blood-ethanol analysis involved the following four steps: (1) separation of ethanol from blood by diffusion in specially blown glassware; (2) oxidation of ethanol with a mixture of potassium dichromate and sulfuric acid; (3) addition of potassium iodide to the reaction mixture after oxidation of ethanol; and (4) back titration of liberated iodine with standard sodium thiosulfate and a starch indicator to detect an endpoint.

Many later modifications to this basic procedure appeared, such as using a different endpoint indicator for the titration (e.g., methyl orange), or another kind of oxidizing agent (e.g., ferrous salts), or separation of ethanol from the biological matrix in a different way. It became common practice to refer to these modified methods with the name of the scientist who first published the report—including Harger, Kozelka-Hine, Smith, Southgate-Carter, and Cavette, to name just a few.

Later developments in methods of ethanol analysis (such as gas chromatography) plus the availability of modern clinical laboratory equipment made it more convenient to dispense the aliquots of blood needed for analysis by volume rather than by weight. Micropipettes and more recently diluter-dispenser devices are now widely used for dilution of blood prior to the analysis. The term "concentration" has little meaning when used alone, because it can be expressed in many different ways. The choice of units for reporting BAC differs among countries: for example, milligrams per hundred milliliters (mg/100 ml) in Great Britain (unfortunately often appearing as the ambiguous mg%);

TABLE 1
Concentrations of Alcohol (Ethanol) in Whole Blood for Legal Purposes

Concentration Unit	*Country*	*Legal Limit*
Percent weight/volume (% w/v)	United States*	0.10 g/100 ml
Milligrams per 100 milliliter (mg/dl)	Britain	80 mg/100 ml
Milligrams per milliliter (mg/ml)	Netherlands	0.50 mg/ml
Milligrams per gram (mg/g)	Sweden**	0.20 mg/g
Milligrams per gram (mg/g)	Norway**	0.50 mg/g

*The Uniform Vehicle Code of the National Committee on Uniform Traffic Laws and Ordinances recommends 0.08 grams per 100 milliliters of blood or per 210 liters of breath; at least six U.S. states have adopted this recommendation.
**1 milliliter whole blood weighs 1.055 grams.

gram percent weight per volume (g% w/v) in the United States; and milligrams per milliliter (mg/ml) in many European countries. Other ways of reporting BAC in clinical medicine are milligrams per deciliter (mg/dl), grams per liter (g/liter), or micrograms per liter (μg/liter). When countries outside Scandinavia enacted legal limits of ethanol in the blood of motorists, the concentrations were defined in units of mass of ethanol per unit volume; whether it was grams, milligrams, or micrograms of ethanol in a volume of milliliters, deciliters, or liters seems chosen arbitrarily.

Because the specific gravity of whole blood is greater than water (on average, 1 ml of whole blood weighs 1.055 g), BAC expressed in terms of mass per mass (w/w) is not the same as mass per volume (w/v). In fact, a concentration of 0.10% w/v equals 0.095% w/v. This difference of about 5.5 percent could mean punishment or acquittal in borderline cases of driving while under the influence of alcohol. With the current trend toward "per se" ethanol limits in many U.S. states, great care is needed to ascertain whether w/v or w/w units were intended by the legislature when the statute was drafted. Table 1 gives examples of concentration units commonly used to report BAC for legal purposes. Note that if ethanol were determined in plasma or serum, the concentration would be about 10 to 15 percent higher than for the same volume of whole blood, because there is more water in the sample after the erythrocytes (red blood cells) are removed.

In clinical chemistry laboratories, the Système International d'Unités (SI) has gained worldwide acceptance. According to the SI system, the amount of substance implies "mole" rather than mass. The mole or a submultiple thereof replaces mass units such as grams or milligrams. Accordingly, the concentration of a substance of known molecular weight might appear as mole/liter or millimole per liter (mmol/l) or micromole per liter (μmol/liter). Note that liter is the preferred unit of volume when reporting concentrations of a substance in solution in the SI system. The molecular weight of ethanol is 46.06, and therefore a concentration of 1.0 mol/l corresponds to 46.06 g of ethanol in 1 liter of

TABLE 2
Concentrations of Alcohol (Ethanol) in Breath for Legal Purposes

Concentration Unit	*Country*	*Legal Limit*
Grams per 210 liters (g/210 l)	United States*	0.10 g/210 l
Micrograms per 100 milliliter (μg %)	Britain**	35 μg/100 ml
Micrograms per liter (μg/l)	Netherlands**	220 μg/l
Milligrams per liter (mg/l)	Sweden*	0.10 mg/l
Milligrams per liter (mg/l)	Norway*	0.25 mg/l

*Blood/breath ratio of ethanol is assumed as 2,100:1.
**Blood/breath ratio of ethanol is assumed as 2,300:1.

TABLE 3
Effects of Blood Alcohol Levels

BAL	*(BAC)*	*Effects*
50	(0.05%)	There may be no observable effects on behavior, but thought, judgment, and restraint may be more lax and vision is affected. Significantly more errors in tasks that require divided attention; more steering errors; and increased likelihood of causing an accident.
80	(0.08%)	Reaction time for deciding and acting increases. Motor skills are impaired. The likelihood of a crash increases to three to four times the likelihood when sober.
100	(0.10%)	Six times as likely to be involved in a crash. Reaction time to sights and sounds increases. Physical and mental coordination are impaired; movement becomes noticeably clumsy.
150	(0.15%)	Twenty-five times as likely to be involved in a crash. Reaction time increases significantly, especially in tasks that require divided attention. Difficulty performing simple motor skills. Physical difficulty in driving.
200	(0.20%)	One hundred times as likely to be involved in a crash. Motor area of brain significantly depressed, and all perception and judgment distorted. Difficulty standing, walking, and talking. Driving erratic.
300	(0.30%)	Confusion and stupor; inability to track a moving object with the eyes. Passing out is likely.
400	(0.40%)	Coma is likely.
450–500	(0.45–0.50%)	Death is likely.

SOURCE: Mothers Against Drunk Driving (MADD) and the National Safety Council.

solution. Likewise 1.0 mmol/l contains 46.06 mg; 1.0 μmol/l contains 46.06 μg, and so on. Publications in the field of biomedical alcohol research often report BAC in this way. It follows that 0.1 g% w/v or 100 mg/dl is the same as 21.7 mmol/l.

Statutory limits of BAC existed in several countries before methods of analyzing the breath were developed. It therefore became a standard practice to convert the concentration of ethanol measured in the breath (BrAC) into the presumed concentration in the blood. For this purpose, a conversion factor, usually 2,100:1 was used. Presumably, it was less troublesome to make this conversion than to rewrite the statute to include both BAC and BrAC as evidence of impairment. Accordingly, breath-ethanol analyzers were calibrated in such a way that the readout was obtained directly in terms of the presumed BAC. This conversion of breath to blood ethanol created the dilemma of a constant blood breath ratio existing for all subjects under all conditions of testing. In the United States and elsewhere, a blood/breath factor of 2,100:1 was approved for legal purposes with the understanding that this gives a margin of safety (about 10%) to the accused. Indeed, more recent research suggests that the blood/breath factor should be 2,300:1 for closer agreement between direct BAC and the result derived from BrAC. In the Netherlands and Great Britain, 2,300:1 was chosen to set the legal limit of BrAC when evidential breath-ethanol analyzers were introduced in these countries. Similarly, in some U.S. states, a legal limit of 0.1 g/210 liters in breath is considered equivalent to 0.1 g% w/v in blood for law-enforcement purposes. Table 2 gives the statutory limits of breath-ethanol concentrations in several countries.

Both the prescribed BAC or BrAC limits for motorists and the units of concentration used to differ among countries and even within regions of the same country. The notion of reaching an international agreement about one common BAC or BrAC limit for motorists is an attractive one but hardly attainable.

(SEE ALSO: *Blood Alcohol Content; Breathalyzer; Driving Under the Influence; Drug Testing and Analysis*)

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BLOOD ALCOHOL CONTENT The consumption of alcoholic beverages results in the absorption into the bloodstream of ALCOHOL (ethanol, also called ethyl alcohol) from the stomach and small intestine. The amount of alcohol distributed in the blood is termed blood alcohol concentration (BAC) and is proportional to the quantity of ethanol consumed. It is expressed as the weight of alcohol in a fixed volume of blood, for example, grams per liter (g/l) or milligrams per deciliter (mg/dl). The measurement of blood alcohol concentrations has both clinical and legal applications.

Consuming food with alcohol generally decreases the amount of alcohol that can be quickly absorbed into the bloodstream. Consuming more than one drink per hour causes the BAC to increase rapidly, because it is exceeding the rate at which the body can metabolize alcohol. The percentage of body fat that contributes to a person's total weight also affects BAC. A larger proportion of fat provides less body water into which the alcohol can distribute, thus increasing BAC. For this reason, women generally have a higher BAC for a given number of drinks when compared to men.

(SEE ALSO: *Blood Alcohol Concentration, Measures of*)

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BNDD *See* U.S. Government Agencies: Bureau of Narcotics and Dangerous Drugs

BOLIVIA, DRUG USE IN Bolivia is a land of gaunt mountains, cold desolate plains, and semitropical lowlands situated in the central part of South America. Straddling the Andes mountains, Bolivia's 424,165 square miles occupy an area about the size of Texas and California combined. It is a big country, but with a population of only 7.9 million. About 15 percent are of European heritage; 25 percent are Aymara Indians, 30 percent are Quechua Indians, and 30 percent are mestizos (of mixed Indian and European ancestry). Although Bolivia is rich in mineral resources—petroleum, natural gas, tin, lead, zinc, copper, and gold—it is an economically depressed country, with 66 percent of the population living below the poverty line. Most of the population works in agriculture, which is generally low paying, while a small number work in the mines. In 1998, Bolivia had a national debt of some 4.1 billion in U.S. dollars and an annual gross domestic product (GDP) of 23.4 billion dollars.

Much of the Bolivian population lives on the bleak, treeless, windswept Altiplano (high plain), a plateau more than 13,000 feet above sea level. The Altiplano is an arid expanse of red earth, of about 40,000 square miles, with widely scattered llamas, sheep, cattle, and homesteads. However, the Altiplano is considered to be the most livable part of the country, with 70 percent of the population residing along its western quarter. Much of the rest of the people live in the Yungas, the Chapare, and the Beni—the tropical jungles of northeastern and central Bolivia, where *Erythroxylum coca* thrives. *Erythroxylum coca*, or simple "coca," is the shrub from which COCAINE is derived (Inciardi, 1992).

COCA PRODUCTION

Historically, the chewing of coca leaves was a cultural practice among the Indian peasant laborers of the Andes. The mild stimulation received from the low cocaine-content leaves enabled workers to endure the burdens of their 12- to 14-hour days in the mines and in the fields, so both Bolivian and Peruvian laws have permitted controlled production of coca for domestic consumption—about 12,000 kilograms (kg) in Bolivia (which also includes production for international pharmaceutical use). A part of the Bolivian economy has therefore always depended on the cultivation, transport, and sale of coca leaves.

The growers of illegal coca in Bolivia are the thousands of farm families who have shifted away from the cultivation and harvest of more traditional crops. In the early 1990s, coca accounted for as much as 40 percent of Bolivia's agricultural production, about 50 percent of its gross domestic product, and about 67 percent of its export earnings. However, the Bolivian government, with the

In the Bolivian jungle, a soldier helps to destroy an illegal chemical lab that was used for processing coca into cocaine paste for shipment to Colombia. (© Bill Gentile/CORBIS)

assistance of the United States, began to take steps in the 1990s to eradicate illegal coca cultivation. Bolivia is now the third-largest cultivator of coca, after Peru and Columbia. Voluntary and forced eradication programs have dramatically reduced coca production, with a 55 percent reduction since 1995. The Bolivian government has encouraged farmers to grow legal crops and has set a goal of total elimination of illegal coca production by 2002.

COCA PASTE USE

Not surprisingly, drug use in Bolivia is related to the production of coca and cocaine. Many people in Bolivia have tried coca products in one form or another. However, in Bolivia abuse of cocaine generally involves neither the chewing of coca leaves nor the ingestion of either powder-cocaine or CRACK-cocaine, but rather, the smoking of COCA PASTE—an intermediate product in the transformation of the coca leaf into pure cocaine. In jungle refineries, coca leaves are treated with a wide variety of chemicals, including alcohol, benzol (a petroleum derivative used in the manufacture of motor fuels and insecticides), sulfuric acid, leaded gasoline, sodium carbonate, and kerosene. The process yields crude cocaine (coca paste). Whereas the cocaine content of leaves is relatively low, 0.5 percent to 1 percent by weight, paste has a cocaine concentration ranging up to 90% (Inciardi, 1992).

Known to most South Americans as *basuco*, *susuko*, *pasta basica de cocaina*, or just simply *pasta*, coca paste is typically smoked straight or in cigarettes mixed with either TOBACCO or MARIJUANA. The smoking of coca paste became popular in Bolivia and other parts of South America beginning in the early 1970s (Jeri, 1984). Readily available and inexpensive, it had a high cocaine content and was absorbed quickly. As the phenomenon was studied, however, it was quickly realized that paste smoking was far more serious than any other form of cocaine use. In addition to cocaine, paste contains traces of all the chemicals used to process the coca leaves initially, the oxidized products of these solvents, plus any number of other alkaloids present in the coca leaf.

When the smoking of paste was first noted in South America, the practice seemed to be restricted to the coca-processing regions of Bolivia, Colombia, Ecuador, and Peru, appealing primarily to low-income groups—it was a cheaper price than refined cocaine. By the early 1980s, however, it had spread to other South American nations and to the various segments of the social strata; throughout that decade, paste smoking further expanded to become a major drug problem for much of South America. Although there have been no systematic studies of coca paste use in Bolivia, most observers report that it is concentrated among the impoverished youths of the country's many rural and urban shantytowns (Farah, 1989; Germani, 1988; Noya, 1989), where it contributes to other health-compromising conditions such as poor nutrition, sniffing of gasoline or other INHALANT drugs, and excessive use of alcoholic beverages. New population surveys being completed in Bolivia should help people in that country understand the nature and magnitude of

their coca-related problems and help them devise preventive strategies.

(SEE ALSO: *Coca Plant; Colombia As Drug Source*)

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BOOZE *See* Distilled spirits; slang and jargon

BORDER MANAGEMENT The effective management of U.S. borders has become a priority for the U.S. government, as it attempts both to control illegal immigration and to prevent the importation of illegal narcotics. Though most of the focus has been placed on the U.S.-Mexico border, increasing drug traffic and illegal immigration from Canada has led to more surveillance of the northern border as well. The effectiveness of border management has historically been very difficult, as many federal agencies had some jurisdiction in this area. The failure to coordinate and consolidate border operations limited the ability of the government to meet its objectives. However, by the late 1990s, Congress had moved toward a border policy that was supported by increased funding and a stronger management system.

In 1977, a U.S. government interagency team led by the Office of Drug Abuse Policy (ODAP) conducted a comprehensive review of border control and recommended consolidation of the principal border-control functions into a single border-management agency. Executive departments failed to agree on distribution of resources and organizational placement of the new agency. The border-management agency never materialized.

Border control in the United States was described in the review as an extremely complex problem involving vast distances, many modes of transportation, millions of arrivals and departures, and millions of tons of cargo. Laws to be enforced involved illegal drugs and other contraband, terrorists, public-health threats, agricultural pests and diseases, endangered species, entry visas, duties, and so forth. Nine federal agencies shared border-control responsibilities, contributing to overlap, duplication of effort, and duplicated management systems.

The ODAP report recommended consolidating the inspection and patrolling functions, including operational and administrative support. The potential for improved effectiveness in a consolidated border-management agency was widely recognized. A similar report by the U.S. Congress's General Accounting Office (GAO) also recommended single-agency management and responsibility for border control. Controversy over which activities to include and which executive department should control the new agency was, however, effective in blocking further action.

Major change came when Congress passed the Illegal Immigration Reform and Immigrant Responsibility Act of 1996 (IIRIRA). The IIRIRA is a tough, enforcement-oriented law that seeks to restrict the passage of undocumented aliens across the U.S. borders. The IIRIRA mandated increasing the number of Border Patrol agents by 5000. The law also mandated that the additional Border Patrol agents be deployed in sectors along the border in proportion to the numbers of illegal crossings at such sectors. The legislation, however, requires that the Attorney General coordinate with and act in conjunction with state and local law enforcement agencies to ensure that deployment of resources to

the border does not degrade or compromise the capabilities of interior Border Patrol stations.

Even before the passage of the IIRIRA, the Border Patrol had begun to implement new enforcement strategies. A 1998 Government Accounting Officer report noted that the Immigration and Naturalization Service (INS) had has made progress in implementing some, but not all, aspects of the necessary strategy to curtail illegal entry in the southwest. The strategy, begun in 1994, called for the Border Patrol to (1) allocate additional Border Patrol resources in a four-phased approach starting with the areas of highest-known illegal activity; (2) make maximum use of physical barriers; (3) increase the proportion of time Border Patrol agents spend on border enforcement activities; and (4) identify the appropriate mix of technology, equipment, and personnel needed for the Border Patrol. At ports of entry along the southwest border, the strategy called for the inspections program to increase inspector staff and use additional technology to increase the deterrence and detection of illegal entry and to improve management of legal traffic and commerce.

In addition to the increases in personnel, the IIRIRA required the construction of new barriers along the border and authorizes the purchase of new equipment. The law directed the Attorney General to install additional barriers to deter illegal crossings, especially in areas of high numbers of illegal entries. The legislation mandated the construction of fencing and road improvements in the 14-mile border area near San Diego, starting at the Pacific Ocean and extending eastward. In particular, the law mandated the construction of second and third fences, in addition to the existing reinforced fence, as well as roads between the fences.

As for policing illegal narcotic shipments, the U.S. Customs Service has employed technology to assist its agents. For example, giant x-ray machines have been installed at ports of entry. Trucks and their cargo are examined in this non-intrusive way to detect cocaine vapors. Other high-tech equipment, such as night-vision goggles, motion sensors and low-light TV cameras are now being used on the border.

(SEE ALSO: *Drug Interdiction; Operation Intercept*)

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REVISED BY FREDERICK K. GRITTNER

BRAIN STRUCTURES AND DRUGS

Psychoactive or behaviorally active drugs are substances that alter internal and external behavioral processes including activity levels, moods and feelings. As a result of these changes, while some of these substances can lead to compulsive drug use and drug addiction, others are used to manage neuropsychological disorders. In both cases these drugs do not produce unique behavioral or neurological effects. Their behavioral activity results from modifying existing neuronal systems. To understand the actions of abused drugs on the brain, one must have an understanding of the functions that brain cells serve in the expression of behavior in general. This article focuses on information that will assist readers in understanding the biological basis of drug actions on the brain, and particularly the actions of commonly abused drugs. First, the general classification of brain cells will be discussed, followed by a discussion of brain structure as it relates to function and drug action. The classification of brain cells based on the chemical nature of communication between cells will then be discussed as it relates to the actions of abused drugs.

CLASSIFICATION OF BRAIN CELLS

The brain is a complex structure that has many different types of cells. Brain cells are subdivided into groups based on a number of criteria that include whether they serve as (1) structural support cells (glia) or (2) cells that receive and transmit information (called neurons or nerve cells). If cells are the latter, then additional criteria are: (a) shape or size; (b) their connections; (c) the distance over which they transmit information; and (d) which

chemicals are released to transmit information to other cells. Most of the effects that drugs produce, which are related to abuse potential, are situated on brain cells that process or transmit information. For that reason, the discussions to follow will consider only actions on interneurons (nerve cells that connect to other nerve cells). The actions of drugs on the brain are complex and seldom involve only one type of brain cell. Nerve cells have a high level of connectivity between one another. Cells in one brain region send inputs to and receive outputs from other regions. These factors make the identification of cells in the brain responsible for a given drug effect difficult to distinguish. This is true of even the most simple behaviors, which involve complex interactions between millions of cells. For these reasons, the understanding of the processes underlying addiction is incomplete; however, significant progress has been made during the past 20 years. For example, it is generally believed that there are brain systems that are dedicated to the processes underlying euphoria and feelings of well-being that are stimulated by abused drugs.

ORGANIZATION OF THE BRAIN: BRAIN REGIONS

The Cerebral Cortex. A number of experimental approaches have developed to study the basis of behavior in the brain. One of these has been to study the role of brain regions in behavior. The brain is composed of distinct substructures. The most general categorization scheme separates the brain into five segments called *lobes* (Figure 1). From front to back these include the frontal, parietal and occipital lobes and the cerebellum. The temporal lobe is on the lateral surface of the brain. The outermost surface of the brain is called the *cortex*; this part of the brain has expanded in size the most in higher animals and is thought to be responsible for the high level of intelligence in nonhuman and human primates. Areas of the cortex are specialized so that specific physiological functions are mediated by cells in defined cortical regions. For example, visual processes occur in cells located on the surface of the back of the brain in the occipital lobe. In front of this region, on the border between the parietal and frontal lobes, is the area that controls movement, which is called the motor cortex. The area of the brain that controls sensation, the sensory cortex, is just in front of the motor cortex. The area in front of

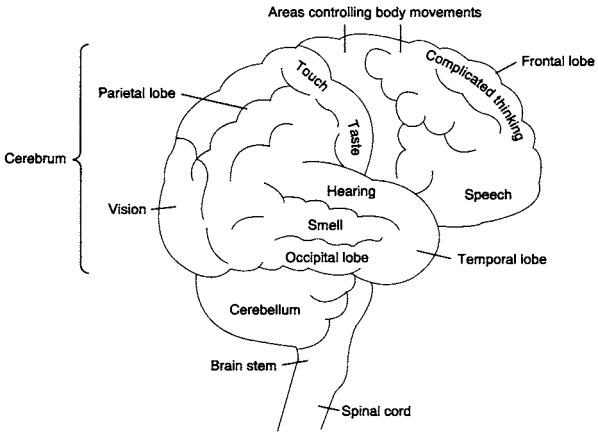

Figure 1
The Lobes of the Brain.
The brain consists of several major sections or lobes. These include the frontal, parietal, occipital, temporal, and cerebellum.

the sensory cortex in the most frontal portion of the brain, the frontal cortex, is involved in cognitive functions and thinking. It is the evolution of this region that is believed to be responsible for superior cognitive functioning in humans.

The Thalamus. Information processing includes sensory information that comes in from sense organs (for example, eyes, ears, tongue) to the brain through the spinal cord or directly through cranial nerves (nerve cells connected directly to the brain). This in-coming information from sense organs goes to a central relay station called the thalamus. The thalamus is specialized, much like the cerebral cortex, in that defined areas receive input that is specific to a sensory modality. For example, input from the eyes through the optic nerve goes to a region of the thalamus called the dorsal lateral geniculate nucleus. This area of the thalamus, in turn, sends the information transmitted from sense organs to the appropriate area of the cortex. For example, the lateral geniculate sends visual information to the area of the cortex specialized for vision, which is located in the occipital lobe. Similarly, the cerebral cortex sends commands to the effector systems (usually muscles) that act on the environment through the same relay system. As one can easily see, the thalamus is a very important structure for the coordination of inputs and outputs from the brain. Thus, degenerative diseases of this structure are very debilitating, as

would be drugs that specifically altered the function of this structure.

The Brain Stem. Other areas of the brain are responsible for life processes of which we are not usually aware. These processes are generally controlled by the part of the brain called the brain stem, which is located between the spinal cord and the cerebral hemispheres of the brain. The brain stem contains the cell bodies for centers that maintain heart rate, blood pressure, breathing, and other involuntary or unconscious life-sustaining processes. A number of psychoactive agents have actions on NEURONS located in the brain stem. For example, OPIATES such as morphine or heroin have a direct inhibitory effect on the brain stem respiration (breathing) centers. This is why heroin OVERDOSES are often fatal—since the breathing centers stop working. Drugs that do not affect neurons in this area, such as marijuana, are seldom life-threatening. A significant part of the reticular formation is also located in the brain stem. This system sends outputs into the brain and down the spinal cord. It regulates arousal by increasing or decreasing the brain's responses to environmental events. Thus, morphine decreases pain by altering the sensitivity of brain cells involved in pain perception. The brain stem is important in the control of pain and also contains the cell bodies for some important nerve cells involved in the euphoric and depressant actions of drugs.

BRAIN SYSTEMS

The Limbic System. Another important anatomical brain system through which abused drugs act is the LIMBIC SYSTEM. This system is a collection of structures that lie between the brain stem and the cerebral cortex. It includes the olfactory bulb, frontal and cingulate cortices, NUCLEUS ACCUMBENS, amygdala, hypothalamus, hippocampus and septum, all of which have direct connections with one another. The limbic system is involved in the control of motivated behaviors such as eating, drinking and sexual behaviors and in the expression of emotional behaviors including anxiety and aggression. Tumors or lesions of these structures often lead to abnormal emotional expression. Drugs that directly affect this system can produce changes in goal-directed behaviors, mood (euphoria-dysphoria) and emotions.

The Motor System. Motor function (movement) involves a number of brain structures that include the caudate nucleus-putamen, which sits above and in front of the thalamus, the premotor cortex, and the motor cortex as previously described. Drugs that increase (stimulants) or decrease (depressants such as alcohol) activity levels may do so by affecting the activity of these structures. Although the basic mechanisms may differ for drugs of different classes, the overall effect may be the same.

NEUROTRANSMITTER SUBSTANCES

Besides categorizing the parts of the brain by structure, the brain can also be separated into systems based on the distribution of the chemicals that nerve cells use to communicate with one another. Thus, cell bodies for some important nerve cells are localized in specific brain nuclei (collection of nerve cell bodies). Some drugs of abuse have specific actions on subsets of cells that use or release a specific chemical to communicate with other cells. For example, ALCOHOL (ethanol) is believed to act on at least three systems in the brain–the ones containing the nerve cells that release SEROTONIN, GLUTAMATE, and GAMMAAMINOBUTYRIC ACID (GABA). The cell bodies of serotonin-releasing nerve cells are localized in the brain-stem region called the raphe' nuclei, while glutamate-releasing and GABA-releasing cells are distributed widely throughout the brain (see figure 2).

What about the different actions of drugs of abuse, where do they act in the brain? As stated previously, it is still not fully understood how the actions of drugs on the brain eventually affect behavior. Our knowledge at this time (2000), however, indicates some specific actions on some defined sites and cell systems. The so-called stimulant drugs (e.g., AMPHETAMINE, COCAINE, METHAMPHETAMINE) produce overall effects on the brain resulting in increased activity, faster speech and thought patterns, and euphoria. This overall effect results from changes in a number of specific behavioral patterns and represents a complex action of these drugs on several important neuronal systems in the brain. Neurochemical studies of the brain have shown that these stimulant drugs enhance and/or prolong the action of the neurotransmitters DOPAMINE, NOREPINEPHRINE, and SEROTONIN that are released by cells that produce these chemicals to

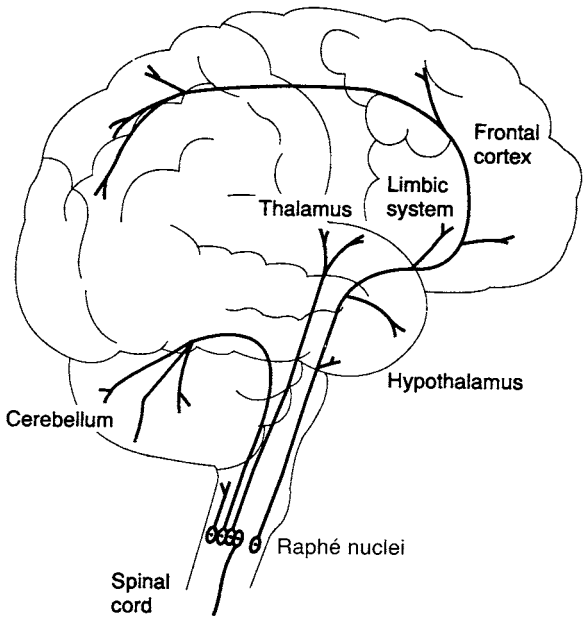

Figure 2
Serotonin Pathways.
The serotonergic neurons originate mainly from the raphé nuclei in the brain stem and project to the forebrain, the cerebellum, and the spinal cord. Very high concentrations of serotonin are also found in the pineal gland. Serotonin-containing neurons are involved in such functions as pain, temperature regulation, sensory perception, and sleep.

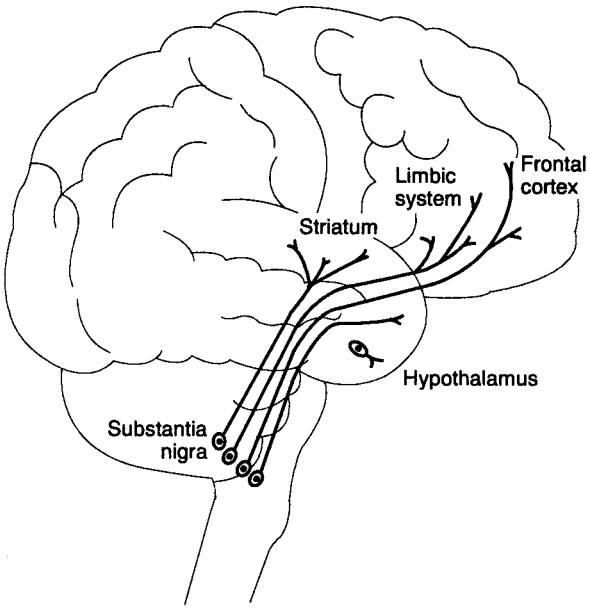

Figure 3
Dopamine Pathways.
Dopamine is not only a precursor of noradrenaline but also a transmitter of its own. Dopamine represents more than 50 percent of the total catecholamine content of the central nervous system. The highest levels of dopamine are found in the neostriatum, nucleus accumbens, and tuberculum olfactorium. There are four main dopaminergic systems in the brain: the nigrostriatal, the mesolimbic nad mesocortical, and the tuberoinfundibular systems. The nigrostriatal system appears to be involved in motor function, while the the tuberoinfundibular dopamine neurons are involved in hypothalamic-pituitary control. The functions of the mesolimbic and mesocortical systems are less well known, although it is conceivable that they play a role in psychotic disease and in reinforcing effects of drugs.

communicate with other brain cells. The dopamine cells send inputs to only a few structures in the forebrain. These include the caudate nucleus that is involved in motor functions and areas of the limbic system that are involved in emotional behaviors and euphoria. The areas of this system to which dopamine cells send inputs include the amygdala, the nucleus accumbens, the olfactory tubercle, and the frontal and cingulate cortices (see Figure 3). Norepinephrine and serotonin cells send inputs more widely to most all forebrain regions, even though their cell bodies are localized in specific brain-stem nuclei. These drugs stimulate motor activity by increasing the function of the dopamine system, which sends inputs to the caudate nucleus. Stimulants produce feelings of well-being and euphoria by enhancing dopaminergic activity in limbic areas. Serotonin is also involved in the effects of stimulant use and withdrawal, but just how is not yet clear.

BIOLOGICAL BASIS OF ADDICTION

Activation of Brain Reinforcement or Hedonic Systems. One common effect of abused drugs is that they produce feelings of euphoria and pleasantness or they decrease unpleasantness. Abused drugs produce these reinforcing effects by activating brain-cell systems that are naturally involved in the reinforcing effects of non-drug rein-

forcers such as eating desirable foods, listening to pleasing music, sex, leaving unpleasant circumstances, and so on. Chemical stimulation by drugs can, however, produce activation of these systems far beyond that produced by these other natural reinforcers. The activation of the mesocorticolimbic dopaminergic system is believed to be critically involved in the neuronal processes that regulate the reinforcing effects of all environmental events. The major components of this system (see Figure 4) include the ventral tegmental area, nucleus accumbens, frontal cortex and ventral caudate-putamen. In addition, the nucleus accumbens is regulated by cells originating in the limbic system including the amygdala, frontal cortex, hippocampus, and thalamus. The nucleus accumbens alters activity in motor systems by the activation of cells in the ventral pallidum and ventral tegmental area (Koob1992; Feldman et al., 1997). Many researchers believe that the ability to modulate these systems by chemical agents is the factor that leads to abuse. Such euphoric effects appear to pose a particular problem for those adolescents who have underdeveloped inhibitory systems, have limited experience with socially accepted forms of personal gratification, and have higher than average levels of aggression. Even without such characteristics, adolescence is one of the most confusing and stressful periods of human development. Ready access to simple chemical means of activating reward systems under these conditions can easily lead to abuse.

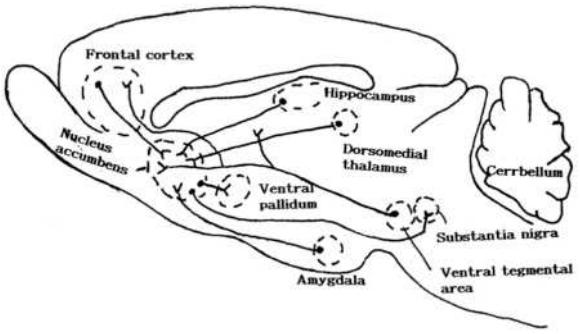

Figure 4
Neuronal circuits involved in the reinforcing effects of stimulant drugs.
The major components of this system include the dopamine pathways from the ventral tegmental area to the nucleus accumbens caudate-putamen and frontal cortex. The limbic system inputs to the accumbens from the amygdala, frontal cortex, hippocampus and thalamus and the outputs from the nucleus accumbens to the ventral pallidum and substantia nigra. (Adapted from Koob, 1972, and Feldman et al., 1977)

Drugs Do Not Have Intrinsic Hedonic Properties. The euphoria that occurs after chemical activation of these systems is not only the result of the direct actions of the drug on neurons but is also influenced by the expectations of the individual and the environment in which the drug is taken. Studies in laboratory animals have shown large differences in the effects of a drug on the brain depending on whether the drug is self-administered or administered to the animal passively (not under its control). It has become clear that the act of drug taking and control over when the drug is taken are perhaps the two most important factors in the pleasant feelings that follow drug intake. The drug itself has no consistent intrinsic hedonic properties. Why is it important for an individual to control the onset of drug action through self-administration? It suggests that the activation of these brain systems is also under behavioral influences. Drugs have behavioral effects that are not the same in everybody and can even change in the same individual. For example, alcohol can produce feelings of euphoria in a social situation or depression when one is alone. Another example is cocaine, a very potent stimulant of brain systems involved in euphoria and feelings of well-being. However, when animals are given simultaneous infusions of cocaine without control of delivery, cocaine becomes a stressor that will lead to the animal's death much faster than animals controlling and self-administering the drug.

Dopamine Hypothesis of Drug Abuse. It is widely accepted since the mid-1990s that the abuse potential of a wide variety of drugs, at least in part, is directly related to the direct actions of these chemical agents on brain mesocorticolimbic dopamine cells (see Figure 3 and 4). The dopamine cells in this system send inputs to the limbic system, including the limbic cortical regions. The dopamine hypothesis states that drugs that are abused directly activate these dopamine-releasing nerve cells, resulting in the production of reinforcement and/or feelings of euphoria and well-being. This may be correct for PSYCHOMOTOR

STIMULANTS like amphetamine and cocaine, which have direct actions upon dopamine releasing nerve cells, but convincing evidence for dopamine being primarily responsible for the abuse of alcohol (ethanol) opiates, and particularly for BENZODIAZEPINES is lacking. Dopamine-releasing nerve cells clearly have an important function in the behavioral process and in the euphoria produced by psychomotor stimulants. To ascribe a universal role for these cells in all euphorogenic processes is, however, likely to be an oversimplification. Scientists initially focused on dopamine nerve cells often at the expense of exploring the involvement of other brain neurochemicals. However, more recent studies have demonstrated the involvement of additional neurochemicals and neuronal inputs. This research has increased knowledge that complex brain neuronal networks regulate behavioral effects that are called euphoria. It is likely that outputs of the cerebral cortex have a significant role in these processes. The roles of these neuronal systems have not been explored.

A drug may be subject to abuse if it directly activates the neuronal networks that are responsible for feelings of well-being and euphoria (positive reinforcement) or if it decreases the unpleasant or aversive nature of the environment in which the individual exists (negative REINFORCEMENT). Most scientific studies of the biological basis of addiction have focused upon the positive reinforcing effects of drugs, a focus which has led some to emphasize the role of dopamine in addiction. However, drug self-administration in humans and in laboratory animal models likely involves both positive and negative reinforcement. The act of taking the drug itself may result in circumstances that produce strain and pressures upon one's normal patterns of living. In addition, the drug itself may activate body and brain systems that are involved in stress, thus directly producing unpleasant circumstances. For these reasons, the research with animals that has implicated dopamine in the positive reinforcing effects of stimulant drugs is likely more the result of both positive and negative reinforcement. It could be that decreases in the unpleasant nature of one's existence may involve increases in the activity of dopamine-releasing nerve cells. It should also be noted that stressful situations can activate some of these same regions.

Neuronal Network Hypothesis of Drug Abuse. In addition to dopamine, the nerve cells that appear to be involved in the euphoric properties of drugs include acetylcholine, glutamate, opioid, and serotonin-releasing cells. As of the very beginning of the 21st century, there is significantly less research data supporting the involvement of these cells; however, it is clear that the brain's opioid receptors are necessary for the reinforcing effects of opiates and that dopamine may not be the exclusive mediator of the euphoric effects of opiates. Serotonin and glutamate may have important roles in the euphoric properties of alcohol, while acetylcholine-releasing neurons may have a role in the general processes underlying euphoria.

Studies in laboratory animals have suggested that specific brain circuits are involved in the processes related to drug reinforcement. These include areas of the cortex, the midbrain, and the brain stem and involve acetylcholine, dopamine, glutamate, gammaaminobutyric acid, norepinephrine, opioid, and serotonin-releasing neurons. The frontal and cingulate cortices (nucleus accumbens, lateral hypothalamus, and amygdala) that are included in the limbic system are part of these circuits, as are the ventral pallidum and thalamus. Brain-stem dopamine, norepinephrine, and serotonin nerve-cell nuclei send projections to these forebrain regions involved in such processes. In turn, these regions send output nerve cells to structures that utilize acetylcholine and glutamate primarily. Some of these forebrain structures are in turn connected to the brain-stem cell nuclei for dopamine, norepinephrine, and serotonin releasing nerve cells by GABA-releasing nerve cells.

CONCLUSION

This is a simplified description of complex neuronal networks that are believed to play a major role in the production of euphoric effects or reinforcement in the brain. It is likely that this complexity will increase as research continues to define and elucidate the basic biology of brain-behavior relationships. Investigations of drug self-administration continue to add significantly to this field of study in the early 2000s. This ongoing research will help us to understand the basic biology of drug abuse so that more efficient and effective forms of treatment and prevention can be developed.

ACKNOWLEDGMENTS

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STEVEN I. DWORKIN

High school students Mary Herz and Mitch Sutherland take a mandatory Breathalyzer test from principal George Yerger before entering the prom in Grant, Nebraska, May 3, 1997.
(AP Photo/George Hipple)

BREATHALYZER Breath-analysis machines detect and measure the ALCOHOL present in deep lung air and convert this to an estimate of BLOOD ALCOHOL CONCENTRATION (BAC). The basis for this calculation is the relatively constant though small proportion of alcohol that the body excretes through the lungs. BAC is approximately 2,300 times breath alcohol concentration, although there is some variation among individuals. Breath analysis machines use methods such as thermal conductivity and infrared absorption to detect alcohol in lung air. Because breath alcohol analysis is quick and non-invasive, it is a useful tool in a variety of situations. The breathalyzer has traditionally been associated with law enforcement agencies for monitoring drinking and driving. However, it is increasingly being used in clinical sellings. A number of models—both portable and fixed ones—are available.

The Breathalyzer breath test machine is the tradename of the model manufactured by Smith and Wesson, but the name has become synonymous with breath test machines. It has emerged as a powerful tool for law enforcement officers in policing motorists who are operating motor vehicles while under the influence of illegal levels of alcohol.

Officers routinely conduct field sobriety tests on motorists they suspect of driving while intoxicated. An officer first requests that the motorist suspected of intoxication perform certain physical tests, such as walking a straight line, putting a finger to the nose, or balancing on one foot, in order to corroborate the officer's conclusion of intoxication of the motorist based on objective findings. If the officer concludes that the motorist has failed one or more of these tests, the officer requests that the motorist submit to a Breathalyzer test. The results of the test either bolster and corroborate police opinion testimony of intoxication or, in those states that set presumptive blood alcohol intoxication levels, to demonstrate that the motorist's blood alcohol level exceeded the permissible level.

If a motorist refuses to take a breathalyzer test, the police cannot compel the person to take the test. However, states have enacted implied consent laws that are civil, rather than criminal, in nature. Under these laws, if a motorist refuses to take the breathalyzer test, the motorist's driver's license is automatically suspended for a set period of time. Thus, motorists who are confronted with the alternatives must balance the criminal sanctions that follow a high alcohol reading from the breathalyzer against the immediate suspension of their driving privileges. However, in the late 1990s, some states, including New York and California, enacted laws that made refusing a breathalyzer test a crime. In these and several other states, legislators concluded that a license suspension was not a severe enough penalty for drunk drivers.

Because breathalyzer test results serve as powerful incriminating evidence, defendants and their

lawyers often seek to challenge the reliability of the tests. This has produced a group of experts that routinely testify as to the way the test was administered and the reliability of the breathalyzer machine itself. The breathalyzer must be calibrated periodically. Calibration is a procedure performed by laboratory personnel to ensure the accuracy and reliability of the instrument. Routine maintenance is also performed to ensure the continued accuracy and proper function of the breathalyzer. Once calibrated, a certificate of calibration is completed by the laboratory and a certified copy provided to the law enforcement agency using that breathalyzer. Failure to follow maintenance schedules can raise a reasonable doubt about the machine's results and lead to an acquittal. Apart from the alleged technical defects of a breathalyzer, experts often testify that the officer failed to follow the proper protocol for operating the machine or that the defendant's blood alcohol level was incorrectly inflated due to biological factors.

Breathalyzers are also being used as preventive devices. Courts are now ordering persons convicted of repeat driving while intoxicated violations to install a breathalyzer interlock on their cars. The driver must breathe into the machine before starting the car. If the alcohol level is too high, the car will not start. After the car has started, the driver must periodically breathe into the device for a retest. If the driver fails the test, the car honks its horn and flashes its lights.

(SEE ALSO: *Driving Under the Influence; Drunk Driving*)

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BRITAIN, DRUG USE IN The legal use of what we now term *illicit drugs* was widespread in nineteenth-century Britain. Opiates in various forms were used by all levels of society, both for self-medication and for what we now call recreational use. The differentiation between medical and nonmedical usage was not clearly drawn then. Concepts such as addiction were not then widely accepted. The story of drug use in Britain since the late nineteenth century is the story of how and why drugs became defined as a social problem and which factors brought the establishment of certain forms of drug-control policy. These were, in fact, issues that often bore little relationship to the objective dangers of the drugs concerned.

In the early twentieth century, there was limited involvement either by doctors or by the state in the control of drug use and addiction. The supply of opiates and other drugs was controlled by the pharmaceutical chemist. As dispensers and sellers of drugs over the counter, they were the de facto agents of control. A rudimentary medical system of treatment operated via the Inebriates Acts (codified in 1890), whereby some inebriates could be committed to a form of compulsory institutional treatment. Legislation covered only liquids that were drunk (e.g., LAUDANUM) not injectables. Users of hypodermic morphine or cocaine were therefore not included under this system.

Drug addiction was not perceived as a pressing social problem in early twentieth-century Britain, nor, indeed, was it one. Numbers of addicts decreased as overall consumption declined. No specific figures are available for that period, but various indicators, such as poisoning mortality statistics, indicate this conclusion. The twentieth century nevertheless brought increased controls and the classification of opiates and other drugs as *dangerous*. Dangerous drugs were regulated through a penal system of control rather than through the mechanisms of health policy.

Two factors brought regulation. The first was Britain's involvement in an international system of drug control; the second was the impact of World War I (1914–1918) and its aftermath. U.S. pressure on the international scene pushed an initially unwilling Britain into a system of control that rapidly extended from the 1909 Asian regulation discussed at Shanghai to the worldwide system envisaged in the 1912 Hague Convention.

Prior to World War I, however, only the United States, by way of the HARRISON NARCOTICS ACT of 1914, had put this system of drug control into operation. Britain favored a simple extension of the existing Pharmacy Acts. The influence of emergency wartime conditions, however, brought a dif-

ferently located and more stringent form of control. The fear of a cocaine epidemic among British soldiers patronizing prostitutes in the West End of London—a fear which on later investigation proved to have been largely illusory—allowed the passage of drug regulation in 1916 under the Defence of the Realm Act. International drug control in its turn became part of the postwar peace settlement at Versailles. The 1920 Dangerous Drugs Act therefore enshrined a primarily penal approach; control was located in the Home Office rather than in the newly established (1919) Ministry of Health.

British drug policy was henceforward marked by a tension between rival conceptualizations of the drug-addiction issue; drugs as a penal issue versus drugs as a health matter. The 1920s saw this conflict at its height. Britain seemed likely to follow a penal course similar to that of the United States, on whose 1914 act the British legislation was consciously modeled, but British doctors soon reasserted their professional control. By 1926, Britain's ROLLESTON REPORT legitimated a medical approach that could entail medical "maintenance prescribing" of opiates to a patient who would otherwise be unable to function. The Rolleston Report established what became known as the BRITISH SYSTEM of drug control—a liberal, medically based system—albeit one that operated within Home Office control.

This system remained in operation for nearly 40 years, until the rapid changes of the 1960s. The 1920s, 1930s, and 1940s were decades when the numbers of addicts were small and there were few nonmedical users (less than 500). It is generally recognized that the British System of medical control operated *because* of this situation rather than *as the cause* of it. This equilibrium began to break down after World War II (1939–1945), when more extensive recreational, or nonmedical, use of drugs (such as HEROIN and COCAINE) began to spread for a variety of reasons. These included the spread of cannabis (MARIJUANA)—from the new immigrant to the white population, overprescribing of heroin by a number of London doctors, thefts from pharmacies, and the arrival of Canadian heroin addicts. Other drugs—in particular, AMPHETAMINES—also became recreationally popular.

The official numbers of heroin addicts rose rapidly, from 94 persons in 1960 to 175 in 1962; and cocaine users increased from 30 in 1959 to 211 in 1964. Nearly all of these were nonmedical consumers. The average age of new addicts also dropped sharply. Initial government reaction, in the report of the first Brain Committee (1961), was muted; however, the second report (1965), produced when the committee was hastily reconvened, had an air of urgency. Controls were introduced on amphetamines in 1964. The report's proposals (implemented in the Dangerous Drugs Act of 1967) took the prescribing of heroin and cocaine out of the hands of general practitioners and placed it in those of specialist hospital doctors working in drug-dependence units. A formal system was established that notified the Home Office about addicts.

The clinic system established in 1968 did not operate as originally intended. In the 1970s, as the rise in numbers of addicts appeared to stabilize, clinic doctors moved toward a more active concept of treatment, substituting orally administered METHADONE for injected heroin and often insisting on short-term treatment contracts rather than on maintenance prescribing. These clinic policies aided the emergence of a drug black market in Britain in the late 1970s. An influx of Iranian refugees from the Islamic revolution of 1979, bringing financial assets in the form of heroin, also stimulated the market.

The British elections of 1979 returned a Conservative government with a renewed emphasis on a penal response to illicit drugs. Britain participated enthusiastically in the U.S.-led international "war on drugs," but there were also strong forces inside Britain arguing for a more health-focused approach. In 1985, the discovery of acquired immunodeficiency syndrome (AIDS) among injecting drug users in Edinburgh, Scotland, was the trigger for policies that emphasized the reduction of harm from drug use rather than a prohibitionist stance. Nevertheless, in the early 1990s, the tension between penal and health concepts and the interdependence of the two approaches to policy still remained unresolved.

The use of drugs within British society continued to expand in the 1990s. Amphetamines are still second only to cannabis as the most widely used drugs in the United Kingdom, but few users are in contact with drug treatment services or seek any medical help. Services are oriented towards opiate users and black market amphetamine is not expensive, so there is a lower likelihood that financial problems will force users into treatment. Heroin use has also continued to grow. During the 1980s

this emerged in a large number of communities round the country and in a pattern different from that of the 1960s. This new pattern of use mainly involved adolescents and young adults, and the heroin was taken by a new method called "chasing the dragon"—heating heroin on tin foil, with vapors inhaled through a tube. But there was great regional variation, with injecting still popular in some areas. Heroin use has continued to grow; the number of known addicts has grown from about 5000 in 1980 to approximately 50,000 by the late 1990s, with figures still growing at about 20 percent a year. Cocaine use has also risen, but the speed and penetration of crack cocaine into the country has been nowhere near as rapid or as substantial as U.S. commentators had predicted. Surveys suggest that snorting cocaine is more popular than crack or heroin and is on the increase in clubs. Ecstasy (MDMA) use has also received wide media publicity, but surveys suggest it is used less frequently than other "dance drugs," LSD and amphetamine.

British governments, conservative for most of the 90s and governed by the Labour party since 1997, have continued to publish national strategies on drugs, the first of which appeared in the 80s. In 1995, *Tackling Drugs Together: a strategy for England, 1995-1998*, was published and strategies for Scotland and Wales followed. The strategy committed the government to take effective action through law enforcement, accessible treatment and a new emphasis on education and prevention to increase community safety from drug related crime; reduce young people's drug use and reduce health risks and damage associated with drug use. In 1998, the new Labour government published *Tackling Drugs Together to Build a Better Britain: the Government's Ten Year Strategy for Tackling Drug Misuse*, which reiterated these main themes. Former Chief Constable, Keith Hellawell, was appointed "Drug Czar," or national coordinator; his deputy had a background in rehabilitation services.

The relationship between penal and health responses in drug policy has remained central. Arrest referral schemes are common and the government is now to expand pilot treatment and testing orders, which will give an alternative to custody to drug using offenders who agree to undergo treatment. Treatment services in prisons have expanded since the incorporation of the prison health service into the National Health Service: new treatment programs and a through-care service for drug using prisoners will be set up. Mandatory urine testing in prisons has proved controversial. Some policy analysts have argued that U.K. policy is moving to a harsher stance, in effect to compulsory treatment and to a greater emphasis on criminal justice initiatives, and to coercion. The government's unwillingness to accept the conclusions of an independent inquiry into drug policy, which recommended liberalization of the law on cannabis, has been cited as evidence of this. However, there is also official interest, following a House of Lords report, in the medical uses of cannabis and a National Treatment Agency is to be set up, emphasizing the health aspects of drug use. The duality of policy continues.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy; British System of Drug-Addiction Treatment; Opioids and Opioid Control: History*)

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VIRGINIA BERRIDGE

BRITISH JOURNAL OF ADDICTIONS
See Addiction

BRITISH SYSTEM OF DRUG-ADDICTION TREATMENT To many observers from outside the United Kingdom (U.K.), the British System is synonymous with heroin maintenance, with doctors supplying drugs on demand to addicts. To some it has been viewed as an approach of extreme folly; to others it is an effective policy of supreme pragmatism. To those who know and work within it, the British System is somewhat

more complex. Indeed, the extent to which a clearly defined system can be identified has been the focus of debate. Here, we will demonstrate that a particular set of factors have combined in the U.K. to create an evolving system of care for drug takers, which has been responsive both to the changing drug scene and to the individual needs of the drug taker. This review will identify the key characteristics of the British System. Important historical milestones in the development of the system will be identified. Finally the effectiveness of the system will be discussed.

WHAT IS THE BRITISH SYSTEM?

Since a key characteristic of the system has been its evolution during the twentieth century, observers at different stages in this process have had different views as to its nature and purpose. This late twentieth-century view proposes the following five characteristics of the British System:

1. *An Evolving System of Health and Social Care for Drug Takers*. Since the 1920s, the British policy toward addiction has been principally in the form of treatment conducted by medical practitioners. This differed from most other jurisdictions, particularly the United States, where addiction was deemed a deviant and criminal activity under the HARRISON NARCOTICS ACT (1914) through and until the revisions of the late 1960s and early 1970s. While the burden of care for drug takers has expanded over time from the general practitioner to specialist psychiatrist and then back to the generalist (often the general practitioner), the British System has to a large extent been located in the public-health sector, latterly in the National Health Service. Specialist drug-dependence clinics were established throughout the U.K. from the 1960s onward.

   Two important consequences of the emphasis on health rather than correctional services have been (1) the attention to the health and social needs of the drug taker—particularly crucial in the wake of the recent human immunodeficiency virus (HIV) epidemic in injecting drug takers, and (2) the opportunity to influence the development of public-health strategies on a national scale through the existing system of health-care services. This second point has been important in the rapid development of new services, including injecting-equipment exchange schemes in the 1980s as a public-health measure to reduce the spread of HIV.
2. *Control and Monitoring of Drug Takers and Their Physicians*. The extent to which the British System has aimed to exercise control over drug takers has been variable. However, part of the purpose of prescribing drugs in the context of addiction treatment (see below) has been to attract drug takers to have contact with statutory services. An index of drug takers (mainly of HEROIN and COCAINE) known to physicians has been maintained by the Home Office since 1968. This has allowed some indication of the scale of the problem to be known as well as preventing individuals from attending more than one clinic. At the same time, in response to concerns over irresponsible prescribing by certain physicians, only those physicians in possession of a special license issued by the Home Office were entitled to prescribe heroin and cocaine to drug takers. Although the recent (early 1990s) policy has been to encourage the increased involvement of general practitioners in prescribing METHADONE, the prescribing behavior of all doctors in relation to addictive drugs remains closely monitored.
3. *Drug Prescribing*. The overall contribution of drug prescribing as a component of the British System has been important, but overestimated by some. Certainly, the right of physicians to prescribe, and drug takers to receive, addictive drugs in the context of treatment has been a key part of the British System since its inception. At various times, opiates (including injectable heroin and methadone), cocaine, AMPHETAMINES, and BARBITURATES have been prescribed in this context. The aims of such prescribing, as well as the prescribing practices, have varied over time. The principal aim throughout has been to provide a method of detoxification that is as comfortable and medically safe as possible. It has also been accepted at a policy level, however, that some individuals who are either unable or unwilling to stop drugs may require long-term prescribing, with a view to stabilization or maintenance treatment—in some instances with injectable drugs. Since the 1970s, oral methadone (in the form of tablets or linctus—a syrup) has been the opiate prescription of choice

in view of its greater safety and lower resale value on the black market. The prescription of other drugs had largely ceased in this context by the mid-1980s. Heroin prescribing and the prescribing of injectable methadone still have a few proponents, despite a lack of controlled scientific evidence to support their effectiveness (see below). Although there is international interest in the right of British doctors to prescribe injectable heroin, fewer than 200 patients receive such treatment (in the early 1990s). Of more interest is the prescribing of injectable methadone as a potential intermediate step in converting the heroin injector to oral methadone.

Drug prescribing within the British System has been characterized by a greater permissiveness and flexibility than is the case in most other jurisdictions, alongside a continued overall conservative approach by most medical practitioners to prescribing agonist drugs to the drug abuser.

4. *Competition with the Black Market*. A prominent aim at various times in the history of the British System has been that of attracting the drug taker away from the black market and into treatment. The putative benefits of such a scheme would be to remove demand for illicit drugs, leading to elimination of the black market; improve health benefits to the drug user for taking pharmaceutical rather than illicit drugs; and reduce criminality associated with purchasing illicit drugs. While there were relatively few heroin takers in Britain until the 1960s, the continued nonexistence of an imported black market meant that prescribed heroin was the main source of supply, but it actually contributed to a growth in the number of drug takers. Since no convincing evidence has emerged to support the value of this approach, the prescribing of drugs has become based more on individual medical indications than on economic policy.
5. *Flexibility*. Perhaps the most striking feature of the British System has been its capacity to evolve in response to the changing drug problem. Whether this has been the result of deliberate policy or benign laissez faireism is open to debate. The result has allowed a flexible response without overt government intervention in medical practice (beyond the constraints described). Flexibility has been possible at two levels. At the system level, experiments in the provision of a range of services have been possible, including a wide range of drug prescribing-and-injecting equipment exchange schemes. At the individual level, treatments may be tailored to individual needs rather than having the imposition of tightly restricted, prescriptive, and blanket approaches.

MILESTONES IN THE HISTORY OF THE BRITISH SYSTEM

1920

Introduction of the Dangerous Drugs Act following the International Opium Convention at the Hague (1912). This restricts the dispensing of several drugs to physicians, including opiates and cocaine.

1926

The Rolleston Committee publishes its report (see ROLLESTON REPORT), which establishes the right of medical practitioners to prescribe drugs in the context of the treatment of addiction.

1920s–1960s

Small numbers (approx. 400–500) of mainly "therapeutic addicts" and addicted physicians receive opiate prescriptions as treatment for addiction.

1961

The first Brain Committee publishes its report. In reviewing the period since the Rolleston Committee's report it reaffirms the medical practitioner's role and recommends no change in the system.

1960s

A small number of physicians, mainly in London, are prescribing large quantities of heroin leading to a rapid increase in the number of heroin injectors (increasing to 2,000 in number) and growing public alarm.

1965

The Brain Committee is reconvened to consider the increasing problem and publishes its second report. It recommends several restrictions including (1) the establishment of specialized treatment clinics, (2) the licensing of medical practitioners for prescribing, and (3) the Home Office Addicts Index.

1967

The Dangerous Drugs Act implements the recommendations of the Brain Committee and prohibits physicians from prescribing heroin or cocaine to drug takers (except for the purpose of relieving pain caused by organic illness or injury) unless specially licensed by the Home Office. This practically restricts prescribing to the newly established specialist clinics.

1980s

An epidemic of heroin taking occurs on a scale not previously encountered in the U.K.—mainly as a result of new illicit trade routes opening up from the Golden Crescent (Iran, Pakistan, Afghanistan, etc.) to supplement the Far East's GOLDEN TRIANGLE trade. The epidemic is mainly among young Caucasian males in inner city areas throughout the U.K. and leads to a rapid increase in crime (to support the habit). The estimated number of heroin takers increases from around 20,000 in the early 1980s to as many as approximately 150,000 by the end of the 1980s.

1982

The Advisory Council on the Misuse of Drugs (ACMD) publishes its Treatment and Rehabilitation report. The specialist clinics are overwhelmed by the upsurge in demand; a new recognition emerges that drug takers represent a heterogeneous group, many of whom do not require specialist treatment. The report recommends (1) an expanded role for the generalist, (2) the development of nonmedically based treatment approaches, and (3) a requirement for health authorities to monitor the scale of heroin problems in each community. This results in more restrictive opiate prescribing and a broadening of the range of treatment options.

1984

Guidelines for Good Clinical Practice in the Treatment of Drug Misuse are published. This is the first central guidance to physicians since the inception of the system, which indicates the flexibility of practice that physicians had been accorded.

1988/1989

The ACMD publishes two reports on AIDS and Drug Misuse. This major policy review is prompted by the epidemic in HIV infection in injecting drug takers. The reports recommend greater emphasis on attracting and retaining in treatment drug takers unable or unwilling to change their behavior. This results in less restrictive opiate prescribing practices, the introduction of low threshold and user friendly services, and the further expansion of injecting-equipment exchange schemes through the late 1980s and early 1990s.

1991

New Guidelines for Good Clinical Practice is published. These are written by physicians for physicians and aimed at the generalist.

1992

Targets for reductions in drug injecting are introduced as part of the British Government's Health of the Nation white paper—and also formed a part of the AIDS response.

1993

The ACMD's third report on AIDS and drug misuse is published. The constituency of concern is broadened from opiate injectors to those who inject amphetamines and BENZODIAZEPINES. Recommendations include a focus on the impact of hidden drug-taking populations, through outreach, and the introduction of oral methadone programs (along North American and Australasian lines).

HAS THE BRITISH SYSTEM BEEN EFFECTIVE?

Clearly, the question of effectiveness is difficult to answer in the context of a national problem subject to many external and internal influences, within a system that has evolved over many years. Further, it is difficult to compare the effects of policies toward drug problems in various countries: Drug problems are often culturally specific, and attempted solutions that may be acceptable in one setting may be unwelcome or unhelpful in another. On one level, it is clear that the U.K. has not been spared the epidemic rise in heroin taking experienced by other Western industrialized countries; nor has it avoided the spread of HIV in intravenous drug takers—however, the epidemic of HIV has been far less severe than in several other European countries and in the United States. Regional variation in the pattern of the HIV epidemic in the U.K. suggests that the areas where prescribing and specialist clinics were limited, such as in Edinburgh, Scotland, experienced a much more rapid spread—although closer examination reveals this to be insufficient as the sole explanation.

At times, there have been disadvantages to the British System. In particular, a situation where addictive drugs were overenthusiastically prescribed contributed to a worsening of the problem. Further, the U.K. experience with barbiturate and amphetamine prescribing was wholly negative and resulted in its complete discontinuation.

At an individual level, remarkably little controlled research has been carried out to evaluate different prescribing or other treatment approaches, given the opportunity available to do so in the British System. One controlled trial with ninety-six heroin takers involved the random assignment to either injectable heroin or oral methadone maintenance. The results suggested advantages and disadvantages in both treatments. At one year follow-up, more in the methadone group were abstinent than in the heroin group, but more had also returned to illicit drug use in the methadone group.

With the increasing emphasis on treatment in the primary care setting in the 1980s, specialist Community Drug Teams were established with the brief of encouraging the increased involvement of general practitioners. The main advantage of such an approach is, in theory at least, that this should allow greater availability of services than could be provided by specialist clinics alone. During the 1990s, there has been an important but modest increase in the extent of general practitioner involvement, but this still falls short of the goal of universal availability of treatment for drug takers.

Overall, it can be said that the principal benefits of the British System have been (1) to ensure the humanitarian handling of drug takers, through treatment services, and (2) to allow the evolution of a system of care responsive to changing needs—which also has been relatively free from unnecessary governmental constraints.

(SEE ALSO: *Britain, Drug Use in; Injecting Drug Users and HIV; Needle and Syringe Exchanges and HIV/AIDS*)

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D. Colin Drummond
John Strang

BULIMIA NERVOSA Since 1980, bulimia nervosa has been recognized by the American Psychiatric Association as an autonomous eating disorder. The term *bulimia* means "an extreme hunger," but the word is most commonly understood to refer to Bulimia Nervosa. It is characterized by recurrent episodes of binge eating followed by such regular activities as self-induced vomiting, excessive use of laxatives and/or diuretics, fasting or dieting, and vigorous exercise—all of which are directed at weight control. A characteristic feature in the bulimic patient is a persistent concern with weight and body shape. Other psychiatric disorders can accompany bulimia, particularly major depression. The full syndrome affects 1 to 3 percent of the adolescent and young adult female population, but many more experience subclinical variants of the disorder. Bulimia nervosa does occur in males, but such incidence is rare.

This disturbance in eating affects mostly young women—usually women of normal weight—and is often preceded by Anorexia nervosa (restricted eating). The bulimic symptoms may continue for many years with exacerbations and remissions. From the mid-1970s to the mid-1990s, the prevalence of eating disorders appeared to be increasing in industrialized countries. The etiology of bulimia is unknown, although psychological, sociocultural, and biological theories have been proposed. Many consider Western societies' increasing emphasis on thinness, especially among women, to be a contributing influence.

Parallels between bulimia nervosa and substance abuse have been drawn based on an Addiction model, a self-psychology model, and a psychobiological model. According to the *addiction model*, food is the "substance" that is abused in bulimia nervosa. Although there are superficial similarities in phenomenology between binge eating and substance abuse, these similarities are selective and rely on a loose definition of addiction. The *self-psychology perspective* is that both bulimia nervosa and substance abuse arise from a common deficit in psychological functioning. Difficulties regulating affect and tension generate a need for the external distraction provided by food or psychoactive substances, respectively. This model may have some heuristic value but it has not, as of the mid-1990s, received empirical validation. The *psychobiological view* regards eating and drinking as consummatory behaviors with the potential for dysregulation. One possibility is a shared disturbance in the brain neurochemical functioning that regulates drives of appetite. There is some evidence that brain Serotonin function may be disrupted in both bulimia nervosa and Alcohol abuse; however, research in this area has just begun and the validity of this model is unknown as of the mid-1990s.

Among women receiving treatment for substance abuse, estimates of the prevalence of bulimia nervosa range from 8 to 17 percent, and estimates of the prevalence of some eating disorder range from 26 to 47 percent. Similarly, estimates of alcohol abuse among women seeking treatment for bulimia nervosa range from 27 to 49 percent. Thus, substance abuse and bulimia nervosa occur together in young women much more frequently than would be expected for independent disorders. One potential source of this comorbidity lies in genetic risk. Several studies have indicated an overrepresentation of alcohol abuse in the families of women with eating disorders. Another possibility is that certain psychological factors place certain women at risk for the development of either bulimia nervosa or substance abuse. There is some limited evidence for an underlying Addictive Personality in both disorders. As well, women with both

disorders seem to have more difficulties, generally, with impulsive behaviors.

The treatment of bulimia nervosa depends on its severity. Many cases of the eating disturbance resolve on their own. Specific interventions that may be tried include psychodynamic (individual, family, group) therapies as well as cognitive and behaviorally oriented therapies and pharmacological treatments. Modest improvements have been reported with the use of ANTIDEPRESSANT medication. Studies conducted in the late 1990s have shown that ondansetron (a drug commonly used for patients with vomiting associated with chemotheraphy) could be an effective treatment for those with bulimia; this drug was not shown to treat the psychological aspects of the disorder though (Kiss, 2000).

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BUPRENORPHINE Buprenorphine is a semisynthetic OPIATE which is produced from thebaine, a naturally occurring ALKALOID present in the ripe pods of the opium poppy (*Papaner somniferum*). Buprenorphine has an ANALGESIC potency twenty-five to fifty times greater than MORPHINE on a weight basis. However, the analgesic actions of buprenorphine are quite similar to those of morphine and the other opiates after taking into consideration its greater potency. It is assumed that these effects are dependent upon its ability to act at *mu* (morphine) receptors in the brain. Once bound to the receptor, however, buprenorphine only produces a limited effect, and thus it is termed a partial AGONIST. This ability to produce only a partial response may explain why buprenorphine lowers breathing (respiratory depression) less than drugs such as morphine. Because it is a partial agonist, buprenorphine administration to morphine-dependent patients does not elicit significant withdrawal symptoms and can therefore be used as a methadone-like opiate substitute in treatment programs. Another reason for the use of the agent in this respect is its particularly long duration of action. Single doses of buprenorphine can attenuate or prevent many of the actions of morphine for up to thirty hours. Thus, buprenorphine maintenance programs have been proposed to treat opiate addiction.

The interactions of buprenorphine with ANTAGONISTS are interesting. Buprenorphine actions can be readily prevented by antagonists such as NALOXONE, when the antagonist is administered prior to buprenorphine. However, antagonists given after buprenorphine do not readily reverse the opioid actions. This unique pharmacology distinguishes it from traditional opiates such as mor-

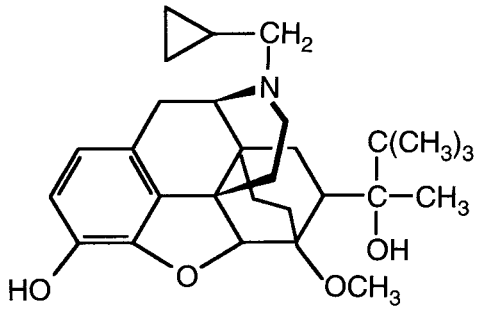

Figure 1
Buprenorphine

phine. Many believe that this observation is due to the prolonged occupation of the receptor by buprenorphine. Once it is bound, other drugs can no longer get to the receptor.

In the early 1990s, it was proposed that buprenorphine might also prove effective in lowering COCAINE use. Some studies in primates showed that buprenorphine lowered the amounts of cocaine taken. Although some small clinical studies in people also suggested a similar effect, more controlled studies did not show a special effect on cocaine use. More extensive work will be needed to determine whether buprenorphine can be useful in the treatment of cocaine abusers.

(SEE ALSO: *Heroin; Treatment/Treatment Types*)

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C

CAFFEINE Caffeine is the world's most widely used behaviorally active drug. More than 80 percent of adults in North America consume caffeine regularly. Average per capita caffeine intakes in the United States, Canada, Sweden, and the United Kingdom have been estimated at 211 milligrams, 238 milligrams, 425 milligrams, and 444 milligrams per day, respectively; the world's per capita caffeine consumption is about 70 milligrams per day. These dose levels are well within the range of caffeine doses that can alter human behavior: As little as 32 milligrams of caffeine, less than the amount of caffeine in most 12-ounce cola soft drinks, can improve vigilance performance and reaction time; and doses as low as 10 milligrams, less than the amount of caffeine in some chocolate bars, can alter self-reports of mood. These data suggest that a large number of people are daily consuming behaviorally active doses of caffeine.

Caffeine-containing foods and beverages are ubiquitously available in and widely accepted by most contemporary societies—yet dietary doses of caffeine can produce behavioral effects that share characteristics with prototypic drugs of abuse: physical dependence, self-administration, and TOLERANCE. Chronic administration of only 100 milligrams of caffeine per day, the amount of caffeine in a cup of coffee, can produce PHYSICAL DEPENDENCE, as evidenced by severe and pronounced withdrawal symptoms that can occur upon abrupt termination of daily caffeine. Under some circumstances, research volunteers reliably self-administer dietary doses of caffeine, even when they are not informed that caffeine is the drug under study; and some evidence indicates that daily use of caffeine produces tolerance to caffeine's behavioral and physiological effects.

CLASS AND CHEMICAL STRUCTURE

Caffeine is an ALKALOID that is often classified as a central nervous system stimulant. Caffeine is structurally related to xanthine, a purine molecule with two oxygen atoms (see Figure 1). Several important compounds, including caffeine, consist of the xanthine molecule with methyl groups attached. A methyl group consists of a carbon atom and three hydrogen atoms. These methylated xanthines, called methylxanthines, are differentiated by the number and location of methyl groups at-

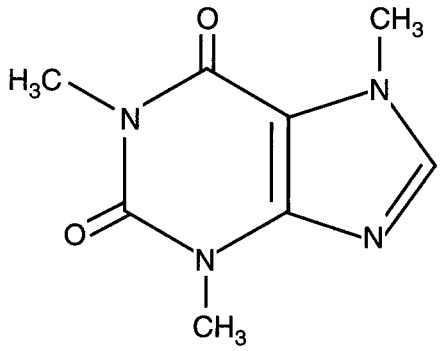

Figure 1
The Caffeine Molecule

tached to the xanthine molecule. Caffeine is a 1, 3, 7-trimethylxanthine. The "tri" refers to the fact that caffeine has three methyl groups. The "1, 3, 7" refers to the position of the methyl groups on the purine molecule. Other important methylxanthines include theophylline, theobromine, and paraxanthine. All these methylxanthines are metabolites of caffeine. In addition, theophylline and theobromine are ingested directly in some foods and medications.

SALIENT FEATURES

Sources. Coffee and TEA are the world's primary dietary sources of caffeine. Other sources include soft drinks, cocoa products, and medications. Caffeine is found in more than sixty species of plants. COFFEE is derived from the beans (seeds) of several species of *Coffea* plants, and the leaves of *Camellia sinensis* plants are used in caffeine-containing teas. CHOCOLATE comes from the seeds or beans of the caffeine-containing cocoa pods of *Theobroma cacao* trees. In developed countries, soft drinks, particularly COLAS, provide another common source of dietary caffeine. Only a portion of the caffeine in soft drinks comes from the kola nut (*Cola nitida*); most of the caffeine is added during manufacturing. Since the 1960s, a marked decrease in coffee consumption in the United States has been accompanied by a substantial increase in the consumption of soft drinks. Maté leaves (*Ilex paraguayensis*), guarana seeds, and yoco bark are other sources of caffeine for a variety of cultures. Table 1 shows the amounts of caffeine found in common dietary and medicinal sources. As can be seen in the range of values for each source in this table, the caffeine content can vary widely depending on method of preparation or commercial brand.

Effects on Mood and Performance. It has long been believed that caffeine stimulates mood and behavior, decreasing fatigue and increasing energy, alertness, and activity. Although caffeine's effects in experimental studies have sometimes been subtle and variable, dietary doses of caffeine have a variety of effects on mood and performance. Doses below 200 milligrams have been shown to improve vigilance and reaction time, increase tapping speed, postpone sleep, and produce reports of increased alertness, energy, motivation to work, desire to talk to people, self-confidence, and well-being. Higher doses can both improve or disrupt performance of complex tasks, increase physical endurance, work output, hand tremor, and reports of nervousness, jitteriness, restlessness, and anxiousness.

DISCOVERY

Caffeine, derived from natural caffeine-containing plants, has been consumed for centuries by various cultures. Consumption of tea was first documented in China in 350 A.D., although there is some evidence that the Chinese first consumed tea as early as the third century B.C. Coffee cultivation began around 600 A.D., probably in what is now Ethiopia.

Caffeine was first chemically isolated from coffee beans in 1820 in Germany. By 1865, caffeine had been identified in tea, maté (a drink made from the leaves of a South American holly), and kola nuts (the chestnut-sized seed of an African tree).

THERAPEUTIC USES

Caffeine is incorporated in a variety of over-the-counter preparations marketed as analgesic, stimulant, cold, decongestant, menstrual-pain, or appetite-suppression medications. As an ingredient in ANALGESICS (painkillers), caffeine is used widely in the treatment of ordinary types of headaches, although evidence for caffeine's analgesic effects is limited: Caffeine may only diminish headaches that result from caffeine withdrawal, but it is also combined with an ergot ALKALOID in the treatment of migraine. Caffeine may have some therapeutic effectiveness in its ability to constrict cerebral blood vessels. The use of caffeine as a central nervous system (CNS) stimulant does have an empirical basis, but there is little evidence that caffeine has appetite-suppressant effects.

Because of various effects of caffeine on the respiratory system, caffeine is used to treat asthma, chronic obstructive pulmonary disease, and neonatal apnea (transient cessation of breathing in newborns)—although other agents, including theophylline, are usually preferred for the treatment of asthma and chronic obstructive pulmonary disease.

Historically, caffeine has been used medically to treat overdoses with opioids and central depressants, but this use has decreased considerably with the development of alternative treatments.

TABLE 1
Caffeine Content in Common Dietary and Medicinal Sources

Source	*Standard Value (in milligrams)*	*Minimum (in milligrams)*	*Maximum (in milligrams)*
Coffee (6 oz./180 ml)			
ground roasted	102	77	186
instant	72	35	211
decaffeinated	4	2	10
Tea (6 oz./180 ml)			
leaf or bag	48	34	58
instant	36	29	37
Cola Soft Drink (12 oz./360 ml)	43	2	58
Chocolate Milk (6 oz./180 ml)	4	2	5
Chocolate Bar (1.45–1.75 oz./40–50 g)	7	5	31
Caffeine-containing Over-the-Counter Medications			
analgesics and cold preparations	32	15	100
appetite suppressants and stimulants	100	50	350

ABUSE

Case reports have described individuals who consume large amounts of caffeine—exceeding one gram per day (1,000 milligrams). This excessive intake, observed particularly among psychiatric patients, drug and alcohol abusers, and anorectic patients, can produce a range of symptoms—muscle twitching, ANXIETY, restlessness, nervousness, insomnia, rambling speech, tachycardia (rapid heartbeat), cardiac arrhythmia (irregular heartbeat), psychomotor agitation, and sensory disturbances including ringing in the ears and flashes of light.

The disorder characterized by excessive caffeine intake has been referred to as caffeinism. There is some suggestion that excessive caffeine consumption can be linked to psychoses and anxiety disorders. Substantial amounts of caffeine are also used by a small percentage of competitive athletes, despite specific sanctions against such use.

Abused drugs are reliably self-administered under a range of environmental circumstances by humans and most are also self-administered by laboratory animals. Caffeine has been self-injected by laboratory nonhuman primates and self-administered orally and intravenously by rats, but there has been considerable variability across subjects and across studies.

Human self-administration of caffeine has been variable, as well; however it is clear that human subjects will self-administer caffeine, either in capsules or in coffee, and even when they are not informed that caffeine is the drug under study. For example, heavy coffee drinkers given repeated choices between capsules containing 100 milligrams caffeine or placebo under double-blind conditions showed clear preference for the caffeine capsules and, on average, consumed between 500 and 1,300 milligrams of caffeine per day. Experimental studies with low to moderate caffeine consumers have found that between 30 and 60 percent of those subjects reliably choose caffeine over placebo in blind-choice tests. Subjects tend to show less caffeine preference as the caffeine dose increases from 100 to 600 milligrams, and some subjects reliably avoid caffeine doses of 400 to 600 milligrams.

TOLERANCE

Chronic caffeine exposure can produce a decreased responsiveness to many of caffeine's effects (i.e., tolerance). This has been observed in both nonhumans and humans. Research with nonhumans has clearly demonstrated that chronic caffeine administration can produce partial tolerance to various effects of caffeine and can produce complete tolerance to caffeine's stimulating effect on locomotor activity in rats. A number of studies also suggest that tolerance to caffeine develops in humans: Daily doses of 250 milligrams of caffeine can increase systolic and diastolic blood pressure, however tolerance quickly develops to these effects

within four days. The stimulating effects of caffeine on urinary and salivary output also diminish with chronic caffeine exposure. Although tolerance appears to develop to some of the central nervous system effects of caffeine, this aspect of caffeine tolerance has not been well explored. Comparisons of the effects of caffeine between heavy and light caffeine consumers provide indirect evidence that repeated (regular) caffeine use diminishes the sleep-disturbing effects and alters the profile of self-reported mood effects. For example, 300 milligrams of caffeine may produce self-reports of jitteriness in people who normally abstain from caffeine but not in regular caffeine consumers. High chronic caffeine doses (900 mg per day) can eliminate the self-reported mood effects (tension, anxiety, nervousness and jitteriness) of 300 milligrams of caffeine given twice a day.

PHYSICAL DEPENDENCE

Evidence of physical dependence on caffeine is provided by the appearance of a withdrawal syndrome following abrupt termination of daily caffeine. Although there have been relatively few demonstrations of caffeine withdrawal in nonhumans, abrupt termination of chronic daily caffeine has been shown to clearly decrease locomotor behavior in rats. Considerably more is known about caffeine withdrawal in humans. Caffeine withdrawal is well documented in anecdotal case reports dating back to the 1800s and in experimental and survey studies from the 1930s to the present. Caffeine withdrawal is typically characterized by reports of headache, fatigue (e.g., reports of mental depression, weakness, lethargy, sleepiness, drowsiness, and decreased alertness), and possibly anxiousness. Descriptions of the withdrawal headache suggest that it develops gradually and can be throbbing and severe.

When caffeine withdrawal occurs, its intensity can vary from mild to severe. Anecdotal descriptions of severe withdrawal suggest that it can be incompatible with normal functioning and include flulike symptoms, fatigue, severe headache, nausea, and vomiting. In general, caffeine withdrawal begins twelve to twenty-four hours after terminating caffeine, peaks at twenty to forty-eight hours, and lasts from two to seven days. Caffeine withdrawal can occur following termination of caffeine doses as low as 100 milligrams per day, an amount equal to one strong cup of coffee, two strong cups of tea, or three soft drinks. Caffeine withdrawal effects can vary within an individual in that a given individual may not experience caffeine withdrawal during every period of caffeine abstinence. The severity of the withdrawal symptoms usually appears to be an increasing function of the maintenance dose of caffeine. Caffeine suppresses caffeine withdrawal symptoms in a dose-dependent manner, so that the magnitude of suppression increases as a function of the administered caffeine dose.

The data described above indicate that the large majority of the adult population in the United States is at risk for periodically experiencing significant disruption of mood and behavior when there are interruptions of daily caffeine consumption.

The nature and time course of effects of terminating daily caffeine consumption is illustrated in Figure 2, a recent experiment involving seven adult subjects. The subjects followed a caffeine-free diet throughout the study and received identically appearing capsules daily. Prior to the study, subjects had received 100 milligrams of caffeine daily for more than 100 days. Placebo capsules were substituted for caffeine without the subjects' knowledge, and subjects continued to receive placebo capsules for twelve days, after which caffeine administration was resumed. The top panel of the figure shows that substitution of placebo for caffeine produced statistically significant increases (asterisks) in the average ratings of headache during the first two days of placebo substitution. Headache ratings gradually decreased over the next twelve days and continued at low levels during the final caffeine condition. The bottom panel of the figure shows that substitution of placebo for caffeine produced similar time-limited increases in subjects' ratings of lethargy/fatigue/tired/sluggish.

ORGAN SYSTEMS

Caffeine affects the cardiovascular, respiratory, gastrointestinal and central nervous systems. Most notably, caffeine stimulates cardiac muscles, relaxes smooth muscles, produces diuresis by acting on the kidney, and stimulates the central nervous system. The potential of dietary doses of caffeine to stimulate the central nervous system is primarily inferred from caffeine's behavioral effects. Low to moderate caffeine doses can produce changes in mood (e.g., increased alertness) and performance

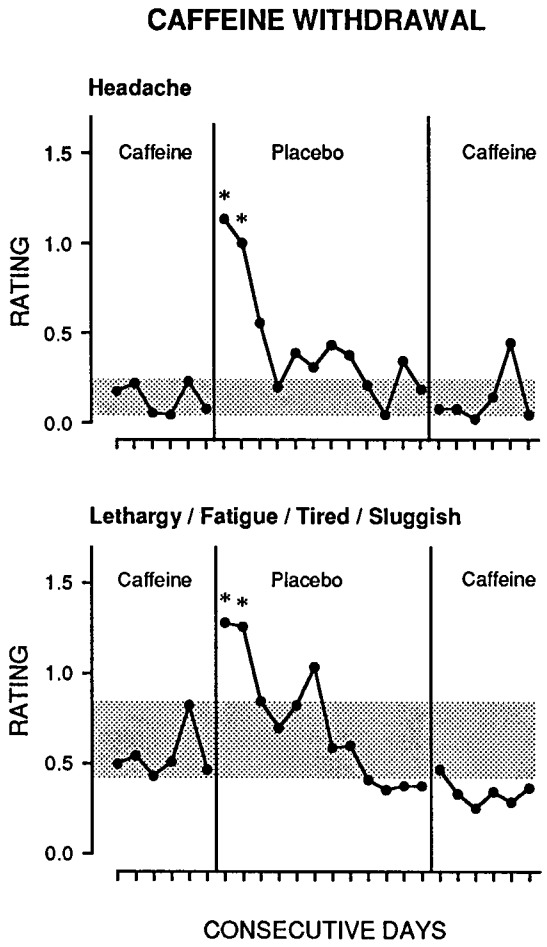

Figure 2
The Termination Effects of Daily Caffeine Consumption
SOURCE: Griffiths et al. (1990). *Low-dose caffeine physical dependence in humans.*

(e.g., improvements in vigilance and reaction time). Higher doses produce reports of nervousness and anxiousness, measurable disturbances in sleep, and increases in tremor. Very high doses can produce convulsions.

Caffeine's cardiovascular effects are variable and depend on dose, route of administration, rate of administration, and history of caffeine consumption. Caffeine doses between 250 and 350 milligrams can produce small increases in blood pressure in caffeine-abstinent adults. Daily caffeine administration, however, produces tolerance to these cardiovascular effects within several days; thus comparable caffeine doses do not reliably affect blood pressure of regular caffeine consumers. High caffeine doses can produce a rapid heartbeat (tachycardia) and in rare cases irregularities in heartbeat (cardiac arrhythmia). Caffeine's effects on peripheral blood flow and vascular resistance are variable. In contrast, caffeine appears to increase cerebrovascular resistance and decrease cerebral blood flow.

Moderate doses of caffeine can increase respiratory rate in caffeine-abstinent adults. Caffeine also relaxes the smooth muscles of the bronchi. Because of caffeine effects on respiration, it has been used to treat asthma, chronic obstructive pulmonary disease, and neonatal apnea (transient cessation of respiration in newborns).

Moderate doses of caffeine can act on the kidney to produce diuretic effects that diminish after chronic dosing. Caffeine has a variety of effects on the gastrointestinal system, particularly the stimulation of acid secretion. These effects can contribute to digestive upset and to ulcers of the gastrointestinal system.

Caffeine increases the concentration of free fatty acids in plasma and increases the basal metabolic rate.

TOXICITY

High doses of caffeine, typically doses above 300 milligrams, can produce restlessness, anxiousness, nervousness, excitement, flushed face, diuresis, gastrointestinal problems, and headache. Doses above 1,000 milligrams can produce rambling speech, muscle twitching, irregular heartbeat, rapid heartbeat, sleeping difficulties, ringing in the ears, motor disturbances, anxiety, vomiting, and convulsions. Adverse effects of high doses of caffeine have been referred to as caffeine intoxication, a condition recognized by the American Psychiatric Association. Extremely high doses of caffeine—between 5,000 and 10,000 mg—can produce convulsions and death.

Extremely high doses of caffeine, well above dietary amounts, have been shown to produce teratogenic effects (birth defects) in mammals. Although there is some evidence to the contrary, dietary doses of caffeine do not appear to affect the incidence of malformations or of low-birth-weight offspring. Although there has been some suggestion that caffeine consumption increases the incidence of benign fibrocystic disease and cancer of the pancreas, kidney, lower urinary tract, and breast, asso-

ciations have not been clearly established between caffeine intake and any of these conditions. Similarly, dietary caffeine has been associated with little, if any, increase in the incidence of heart disease.

Controversies continue over the medical risks of caffeine. Although research has not definitively resolved all the controversies, health-care professionals must make recommendations regarding safe and appropriate use of caffeine. In a recent survey of physician specialists, more than 65 percent recommended reductions in caffeine in patients with arrhythmias, palpitations, tachycardia, esophagitis/hiatal hernia, fibrocystic disease, or ulcers, as well as in patients who are pregnant.

PHARMACOKINETICS

Absorption and Distribution. Caffeine can be effectively administered orally, rectally, intramuscularly, or intravenously; however, it is usually administered orally. Orally consumed caffeine is rapidly and completely absorbed into the bloodstream through the gastrointestinal tract, producing effects in as little as fifteen minutes and reaching peak plasma levels within an hour. Food reduces the rate of absorption. Caffeine readily moves through all cells and tissue, largely by simple diffusion, and thus is distributed to all body organs, quickly reaching equilibrium between blood and all tissues, including brain. Caffeine crosses the placenta, and it passes into breast milk.

Metabolism and Excretion. The bloodstream delivers caffeine to the liver, where it is converted to a variety of metabolites. Most of an ingested dose of caffeine is converted to paraxanthine and then to several other metabolites. A smaller proportion of caffeine is converted to theophylline and theobromine; both of those compounds are also further metabolized. Some of these metabolites may contribute to caffeine's physiologic and behavioral effects.

The amount of time required for the body of an adult to remove half of an ingested dose of caffeine (i.e., the half-life) is 3 to 7 hours. On average, about 95 percent of a dose of caffeine is excreted within 15 to 35 hours. Cigarette smoking produces a twofold increase in the rate at which caffeine is eliminated from the body. There is a twofold decrease in the caffeine elimination rate in women using oral contraceptive steroids and during the later stages of pregnancy. Newborn infants eliminate caffeine at markedly slower rates, requiring over 10 days to eliminate about 95 percent of a dose of caffeine. By 1 year of age, caffeine elimination rates increase substantially, exceeding those of adults; school-aged children eliminated caffeine twice as fast as adults.

MECHANISMS OF ACTION

Three mechanisms by which caffeine might exert its behavioral and physiological effects have been proposed: (1) blockade of receptors for adenosine; (2) inhibition of phosphodiesterase activity resulting in accumulation of cyclic nucleotides; and (3) translocation of intracellular calcium. Only one of these, however, the blockade of adenosine receptors, occurs at caffeine concentrations in plasma produced by dietary consumption of caffeine. Adenosine (an autacoid—or cell-activity modifier), found throughout the body, has a variety of effects that are often *opposite* to caffeine's effects—although caffeine is structurally very similar to adenosine. As a result, caffeine can bind to the receptor sites normally occupied by adenosine, thereby blocking adenosine binding, and preventing adenosine's normal activity. Thus, caffeine's ability to stimulate the central nervous system, and increase urine output and gastric secretions, may be due to the blockade of adenosine's normal tendency to depress the central nervous system and decrease urine output and gastric secretions. The methylxanthine metabolites of caffeine (including paraxanthine, theophylline, and theobromine) are also structurally similar to adenosine and block adenosine binding.

(SEE ALSO: *Addiction: Concepts and Definitions; Tolerance and Physical Dependence*)

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CALCIUM CARBIMIDE Citrated calcium carbimide is a mixture of two parts citric acid to one part calcium carbimide; it slows the metabolism of ALCOHOL (ethanol) from acetaldehyde to acetate, so it is used in the treatment of ALCOHOLISM. It is also known as calcium cyanamide. As an antidipsotropic—an antialcohol or alcohol-sensitizing medication, it has been used for treatment in Canada, the United Kingdom, and Europe since its introduction for clinical use in 1956. Its only therapeutic use is for the treatment of alcoholism. In Canada it is sold under the brand name Temposil. As of 2000, however, calcium carbimide is still not approved for use in the United States.

PHARMACOLOGY

The PHARMACOKINETIC data on the absorption metabolism and elimination of carbimide in humans are incomplete. Since nausea, headache, and vomiting occur because of the rapid absorption of carbimide, for treatment purposes it is formulated as a slow-release tablet. Peak plasma concentrations of carbimide following oral administration in experimental animals occur at 60 minutes; the drug is then metabolized at a relatively rapid rate so that half disappears about every 90 minutes (i.e., an apparent elimination half-life of 92.4 minutes). In humans, an alcohol challenge reaction will occur on an average of 12 to 24 hours after drinking.

Alcohol (ethanol) is normally metabolized first to acetaldehyde, which is then quickly metabolized further so that levels of acetaldehyde are ordinarily quite low in the body (acetaldehyde is toxic). Carbimide produces competitive inhibition of hepatic (liver) aldehyde-NAD oxidoreductase dehydrogenase (ALDH), the enzyme from the liver responsible for oxidation of acetaldehyde into acetate and water. Within two hours of taking carbimide by mouth, ALDH inhibition occurs. If alcohol is then ingested, blood acetaldehyde levels are increased; also mild facial flushing, rapid heartbeat, shortness of breath, and nausea occur with just one drink. As more is drunk, the severity of the reaction increases, with rising discomfort and apprehension. Severe reactions can pose a serious medical risk that requires immediate attention.

DOSAGE AND ADMINISTRATION

In Canada, Temposil is available as round, white 50-mg tablets engraved with the letters "LL" and "U13." The usual dosage is 50 or 100 mg every twelve hours. The drug should never be given to an intoxicated patient and preferably no sooner than 36 hours after the last drink.

Calcium carbimide should be used with caution in patients with asthma, coronary artery disease, or myocardial disease.

In the event of an overdose, the patient should be given pure (100%) oxygen by mask or antihistamines administered intravenously.

SIDE EFFECTS

Unlike disulfiram, carbimide does not have the potential side effect of liver damage. Carbimide, however, exerts antithyroid activity, which can be clinically significant in patients with preexisting hypothyroid disease. According to a 1999 Canadian monograph, other side effects of calcium carbimide include fatigue, skin rashes, ringing in the ears, mild depression, a need to urinate frequently, and impotence. The clinical significance of transient white blood cell increases remains unclear.

USE IN TREATMENT

The rationale for use of carbimide in alcoholism treatment is similar to that of disulfiram. The threat of an unpleasant reaction, which one may

expect following drinking, is sufficient to deter drinking. For alcoholics in treatment who take a drink, the ensuing reaction is unpleasant enough to strengthen their overall conditioned aversion to alcohol. Their reduction of alcohol consumption during carbimide treatment is expected to result in general bodily improvement. A second approach involves the use of carbimide as part of a RELAPSE-PREVENTION treatment, whereby an individual might take it in anticipation of a high-risk situation. As of 2000, scientific evidence supporting the efficacy of carbimide in alcoholism treatment is inconclusive because of a lack of well-controlled clinical trials. No multicenter clinical trials have yet been performed.

(SEE ALSO: *Causes of Substance Abuse: Learning; Disulfiram; Treatment Types: Aversion Therapy*)

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CALIFORNIA CIVIL COMMITMENT PROGRAM Coercive treatment approaches for drug addiction have been utilized consistently throughout the twentieth century, beginning with the morphine maintenance clinics of the 1920s. Federal narcotics treatment facilities were established in Fort Worth, Texas and Lexington, Kentucky in the 1930s. In 1962, the U.S. Supreme Court held, in *Robinson* v. *California* (370 U.S. 660), that a state could establish a program of compulsory treatment for narcotic addiction and that such treatment could involve periods of involuntary confinement, with penal sanctions for failure to comply with compulsory treatment procedures. In 1966, Congress passed the Narcotic Addict Rehabilitation Act (28 U.S.C. sections 2901-2903) that permitted federal judges and prison officials to refer narcotic-addicted probationers and inmates to the Lexington and Fort Worth facilities as an alternative to traditional incarceration. This Act established statutory authority for involuntary inpatient and outpatient treatment and treatment in lieu of prosecution. The Comprehensive Drug Abuse Prevention and Control Act of 1970, more commonly known as the Controlled Substances Act, authorized the diversion of drug-involved offenders from the criminal justice system into drug abuse treatment programs.

The California Civil Addict Program (CAP) was the first true civil commitment program implemented in the United States. In 1961, following a recommendation by the Study Commission on Narcotics, it was placed under the direction of the Department of Corrections and equipped with clear standards for commitment procedures. Addicts convicted of a felony or misdemeanor could be committed to CAP for seven years and then returned to court for disposition of the original charge, or time served in CAP was credited toward the sentence. Addiction was determined by two court-appointed physicians and patients underwent a sixty-day evaluation period (McGlothlin, Anglin, and Wilson 1977).

The program provided both inpatient and outpatient treatment phases and was viewed as a modified therapeutic community. During the initial eight years of CAP (1961-1969), this outpatient program was very stringent adhering to the requirement, "You use, you lose." During the 1970s the program might tolerate infrequent drug use if one's overall behavioral pattern was deemed acceptable (Anglin 1988). Participants in CAP exhibited sustained reductions in drug use, fewer multiple relapses and relapses that were of shorter duration and separated by longer periods of non addiction (Anglin 1988). CAP has an important place in the history of compulsory substance abuse treatment, but the program has been dramatically altered since the late 1970s. As of 1990, the length of the commitment period has been reduced from

seven years to an average of three years. The community phase is often disorganized, ancillary services have been dramatically cut, and there is no treatment service available beyond the minimal 120-hour Civil Commitment Education Program (Wexler 1990).

In 1972, the Treatment to Alternatives Street Crime (TASC) program was created by President Nixon's Special Action Office for Drug Abuse Prevention. TASC, a national program designed to divert drug-involved offenders into appropriate community-based treatment programs, was funded by the National Institute of Mental Health (NIMH) and the Law Enforcement Assistance Administration (LEAA). Federal funding for TASC began to wane beginning in the 1980s until funding was completely withdrawn in 1982. The Judicial Assistance Act of 1984 revived federal endorsement and fiscal support for TASC. This legislation authorized a criminal justice block grant program designed to address drug-related crime and the drug-involved offender. In the more than 100 jurisdictions where TASC currently operates, it serves as a court diversion mechanism or a condition to probation supervision. After referral to community-based treatment, TASC monitors the client's progress and compliance and reports back treatment results to the referring justice system agency. Clients who violate the conditions of their referral are generally returned to the justice system for continued processing or sanctions (Inciardi and McBride 1992).

TASC served as the precursor for the system of 'Drug Courts' currently operating in California and in many other states. A Drug Court is a special court given the responsibility of select felony and misdemeanor cases involving nonviolent drug-using offenders. The program includes random drug testing, judicial supervision, counseling, educational and vocational opportunities and the imposition of sanctions for failure. There are 600 Drug Courts in the nation with about 92 in California. Each is set up utilizing the guidelines of the Federal Office of Drug Court Policy. Clients are responsible for their development and participation in the treatment process. Regular status hearings are held with the judge and the drug court team. After the successful completion of the criminal drug court program, a minimum of 12 months, the drug charge is dismissed. California Drug Courts operate on Federal and State grant money and matching funds from the county where the court is located.

Incarceration of drug-using offenders costs from $25,000 to $50,000 per year. In contrast, the most comprehensive Drug Court System costs an average of $3,000 annually for each offender. The California Drug and Treatment Assessment (CALDATA) estimated a cost of less than $8 per day for outpatient treatment that compares with estimates of $50 to $70 per day associated with jail time. The recent CALDATA study showed a significant reduction in criminal activity during and after treatment, in drug sales and the use of a weapon or physical force.

TASC will continue to expand into the 21rst Century, primarily because it has been recognized by the National Institute on Drug Abuse, the Office of National Drug Control Policy and the Office of Treatment Improvement as an effective program for reducing drug use and related crime (Inciardi and McBride 1992). The courts and their treatment providers provide an early opportunity for treatment and a cost-effective alternative to traditional criminal case processing.

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CANADA, DRUG AND ALCOHOL USE IN Alcohol, tobacco, and cannabis are the most prevalent drugs of abuse in Canada. A 1996 na-

tional survey found that 76.8 percent of Canadian adults aged 15 and over were current drinkers (who had consumed ALCOHOL at least once in the past year); an additional 13.5 percent said they were former drinkers, while only 9.7 percent said they never drank. Per-adult consumption was about 450 drinks per year (7.64 l) of absolute alcohol (ethanol). Overall alcohol consumption has decreased in Canada since the 1980s.

In 1996, 29 percent of adults were current smokers and another 29 percent were former smokers. Overall, the percentage of the population who smoke has been dropping since the 1970s. Practically all TOBACCO is consumed as cigarettes, with daily consumption per smoker estimated at 20.6 (more than one pack) in 1996.

The 1994 national survey found that 23.1 percent of adults had used MARIJUANA or HASHISH at some point, while 7.4 percent had used it the past year. Less than one percent were current COCAINE or CRACK users, and 3.8 percent had used it at some time. Also, 1.1 percent of adults had used LYSERGIC ACID DIETHYLAMIDE (LSD), AMPHETAMINES (speed), or HEROIN in the past year, and 5.9 percent had used them at some point.

High school students and "street" kids reported the use of numerous illegal drugs, such as LSD, HALLUCINOGENS, speed, heroin, glue and other INHALANTS, or made nonprescription use of stimulants, BARBITURATES, and tranquilizers. Most indigenous youth smoke and have rates of alcohol problems and illicit drug use several times higher than the national average. Almost all street youth in Toronto used alcohol and one or more illicit drugs.

The 1996 survey found that 4.5 percent of adults used sleeping pills, 4.3 percent used tranquilizers such as Valium, .9 percent used diet pills or stimulants, 3 percent used antidepressants, and 13 percent used narcotic painkillers such as Demerol, morphine, or codeine. (In Canada, codeine of less than 8 milligrams per tablet is an over-the-counter drug.)

Alcohol, tobacco, and illegal drug use is generally higher among Canadian men than women, but prescription psychoactive-drug use is greater in women. For all types of licit and illicit drugs except tranquilizers and barbiturates, male Canadian students are heavier users than are females.

CONSEQUENCES

A 1993 survey found that 5.1 percent of current drinkers had had a physical health problem due to their drinking at some point. About 2 percent said it had interfered with their friendships or social life, and 2.1 percent said it had affected their home lives or marriages. Finally, 4.7 percent said it affected their financial positions.

In 1996, about 8 percent of current drinkers reported drinking and driving. Of fatally injured drivers who had been tested, 45 percent had positive BLOOD ALCOHOL CONCENTRATIONS (BAC), with 28 percent exceeding 150 milligrams. (The legal level in Canada for impairment is 80 milligrams.)

In 1996, there were 95,877 federal drunk driving offenses, although the number has been declining for several years. Most offenses are for impaired operation of motor vehicles, but about 8 percent are for refusal or failure to provide a breath sample. There were 16,239 people jailed for drunk driving offenses in 1996, about 20 percent of all jailed people.

In 1996, there were 194,916 provincial liquor act offenses, and 14,329 juvenile offenders were convicted in liquor act offenses. However, only 1,201 people were jailed for liquor act offenses, as most are dealt with by fines, suspended sentences, or attendance at detoxification centers.

In 1996, there were 65,106 illicit drug offenses of all types with most (47,002) being for cannabis; there were 11,188 offenses for cocaine and 1,233 for heroin. Almost all convictions were under the Alcoholic Control Act. Offenses under the Food and Drug Act, which covers LSD, stimulants, and nonnarcotic drugs were 1,306.

In 1996, 8,684 people were sent to federal or provincial prisons for drug offenses, the majority of which were for cannabis and cocaine related offenses; they constituted about 16 percent of all federal jail prisoners and 7 percent of provincial jail prisoners.

TREATMENT

In 1996 about 400,000 Canadians were alcohol dependent (2.7% of all adults). The total number treated for alcohol dependence is unknown but probably no more than half have been treated. In 1996, 81,000 cases were treated in hospitals for the

consequences of alcohol problems, such as car accidents, drownings, falls, liver problems, strokes, gastrointestinal problems, and many other causes. There were over 7,000 cases treated in hospitals because of illicit drugs. This included drug psychoses, poisonings, dependency, and various types of accidents.

DEATHS

In 1995, there were 6,701 deaths directly due to alcohol diagnoses. The larger proportions were due to motor vehicle accidents (1,444 persons), suicide (955 persons), liver disease (1,037 persons), alcohol dependence (590 persons), and accidental falls (452 persons). Other causes include various cancers, circulatory problems, and accidents. There were only 804 deaths attributed to illicit drug use in 1995. The majority were due to suicide (329 persons), opiate poisoning (160 persons), and AIDS (83 persons).

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CANCER, DRUGS, AND ALCOHOL The relationship between cancer and drugs, including alcohol, has several aspects. One is the question of carcinogenesis; that is, whether alcohol and other abused substances cause cancer. Another is the relationship between prescription medications and cancer. A third is the complications of cancer treatment in patients with a history of substance abuse.

CARCINOGENESIS

The likelihood of a substance to cause cancer is called carcinogenicity; this is determined in several ways. The first is to see if cells grown *in vitro* (in a test tube) with the potential carcinogen develop cell-structure abnormalities in the new-grown cells. The second is to see if the substance will result in cancers in animals. The third is to look at the clinical course of a disease in a human patient. Finally, the outcomes in a group of people exposed to the substance will be compared to outcomes in those not exposed—either by following the natural course of history in a population through time in cohort studies or in case-control.

In spite of these methods, however, identifying carcinogens is complex and difficult. One reason is the long time delay between use of a carcinogenic chemical and the appearance of cancer symptoms; intervals of 20–30 years are not unusual. As a result, carcinogenic effects are rarely detected in early studies of new medications. Secondly, carcinogenesis appears to be a multistep process involving environmental, nutritional, and genetic factors as well as use of a carcinogenic drug.

Carcinogens are classified as genotoxic or epigenetic. Genotoxic carcinogens act directly on the DNA in human cells, in effect producing abnormal cells. Almost all identified drug carcinogens, however, fall into the second category, the epigenetic carcinogens. These substances encourage the proliferation of latent tumor cells in a tissue or organ.

DRUGS OF ABUSE AND CANCER

Alcohol. Animal data do not show that administering Alcohol alone causes cancer although there is sufficient evidence for the carcinogenicity of acetaldehyde (the major metabolite of alcohol). When alcohol was administered to animals who were also exposed to known carcinogens, the animals who were given the alcohol had a higher rate of tumors of pituitary and adrenal glands, pancreatic islet cells, esophagus, and lungs. They also had higher levels of liver-cell (hepatocellular) carcinomas, liver angiosarcomas, and neoplastic nodules of the liver, as well as benign tumors of the nasal cavity and trachea.

Cancers of the Digestive Tract. Epidemiological studies in humans have shown a causal relationship

between alcohol consumption and cancer of the digestive tract, primarily cancer of the oral cavity, the pharynx (nasopharynx excluded), and the larynx—all two to five times more likely in alcoholics; the esophagus—two to four times more likely; and the liver—liver cancer was increased 50 percent, with primary liver cancer increasing twofold to threefold. These findings persisted, even after adjusting for the effects of smoking, with the relative risk for cancer increasing with the amount of alcohol consumed.

There may be a possible causal link between alcohol, especially beer, and cancer of the large bowel. The risk for colon cancer was increased between 15 percent and threefold depending on the study; that for rectal cancer was increased up to twofold. Unfortunately, these studies did not control for differences in diet.

The mechanism of carcinogenic action appears to be that alcohol acts as a local irritant to the upper gastrointestinal tract, whereas chronic excessive drinking affects the liver, because of the accumulation of the alcohol metabolite acetaldehyde.

Breast Cancer. A strong association exists between alcohol and breast cancer, and there appears to be a dose-response relationship with an apparent relative risk of 1.5 to 2. This relationship holds even after controlling for a number of other factors known to affect breast cancer. As the full etiology (cause) of breast cancer is not yet known, an as-yet-unrecognized factor may account for some of these findings. Overall, it has been estimated that as many as 10 percent of all cancer deaths are due to alcohol.

Tobacco and Illicit Drugs. The role of TOBACCO as a carcinogen is well established and is discussed elsewhere. The role of other drugs as a cause of cancer is still unclear. Some drugs may have a role in cancer development because of mode of administration or degree of carcinogenicity. *In vitro* studies have shown mutagenic properties in a number of drugs—LYSERGIC ACID DIETHYLAMIDE (LSD), OPIUM and its derivatives such as MORPHINE, synthetic narcotics such as METHADONE, and some compounds found in MARIJUANA. There have been clinical reports of cancers in the respiratory tract, primarily the lungs, in heavy marijuana users, of the nasal passages in cocaine users, and in a number of organs in LSD users. Higher rates of esophageal cancer have been reported in opium smokers.

PRESCRIPTION DRUGS AND CANCER

Very few drugs that are suspected of causing cancer in humans are used in contemporary medical practice. Those that are, however, fall into two significant categories: alkylating agents that are used to treat cancer; and birth control pills and other hormone preparations.

Alkylating agents. These antineoplastic drugs include busulfan (Myleran), used to treat leukemia that has not responded to other drugs; cyclophosphamide (Cytoxan), an immunosuppressant used to treat ovarian cancer and malignant lymphoma; melphalan (Alkeran); ifosfamide (Ifex), used to treat testicular cancer and Ewing's sarcoma; cisplatin (Platinol), a drug derived from platinum; and chlorambucil (Leukeran), used in the treatment of leukemia and Hodgkin's disease. All of these drugs have serious side effects ranging from liver toxicity to fibrosis of the lungs, as well as carcinogenic potential. Cyclophosphamide in particular increases a patient's risk of secondary cancers for several years after it has been discontinued.

Synthetic Hormones and Steroid Preparations. The natural and synthetic estrogens used for contraception and for postmenopausal hormone replacement have been associated with an increased risk of uterine cancer. As of the late 1990s, these estrogens are usually combined with progestins, which appear to lower this risk. Diethylstilbestrol (DES), which is used to treat menopausal symptoms and advanced cancers of the breast, may cause vaginal or cervical cancer in women whose mothers took DES during pregnancy. Oxymetholone (Anadrol), a steroid related to testosterone, is reported to increase the risk of liver cancer.

SUBSTANCE ABUSE AND CANCER TREATMENT

On the one hand, NARCOTIC and psychoactive drugs have an important role in cancer treatment. Cancer patients have used *Cannabis sativa* (marijuana) to reduce the nausea associated with chemotherapy. LSD has been used in treating psychological disturbances associated with cancer. Although it was once feared that cancer patients would become addicted to opioids given for pain control, a recent study showed that of 11,882 cancer patients

with no prior substance abuse history, only four became addicts after treatment with opioids.

On the other hand, preexisting abuse of these same substances complicates cancer treatment. A history of substance abuse may shorten a cancer patient's life expectancy and undermine the effectiveness of palliative care. Ongoing substance abuse disrupts the patient's relationships with physicians and other caregivers. As of 2000, the National Cancer Institute has issued guidelines for the clinical management of cancer patients with substance abuse histories. These guidelines include evaluation and treatment of comorbid psychiatric disorders, evaluation of the patient's tolerance of drugs, and monitoring of hospital inpatients.

(SEE ALSO: *Complications: Immunologic*)

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MANUELLA ADRIAN
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CANNABIS SATIVA This is the botanical name for the HEMP plant that originated in Asia. It is the basis of the hemp industry as well as the source of the widely used intoxicant TETRAHYDROCANNABINOL (THC), the active agent in MARIJUANA, HASHISH, GANJA, and BHANG.

HISTORY

The use of *Cannabis sativa* has been recorded for thousands of years, beginning in Asia. It was known to the ancient Greeks and later to the Arabs, who, during their spread of Islam from the seventh to the fifteenth centuries, also spread its use across the Levant and North Africa. Some 200–300 million people are estimated to use *Cannabis* in some form worldwide. Thus, it is not only one of the oldest known but also one of the most widely used mind-altering drugs.

Since the 1960s, the rise in its use in the United States has been enormous and associated with the youth movement and countercultural revolution. Although the drug was in use before that time, it was popular only in some ethnic and specialized groups (e.g., jazz musicians). By the 1990s, some 30–40 million Americans are estimated to have used it and a substantial number use it regularly—although since 1979 the number of youngsters initiated into its use has been declining after a steep rise with an increasingly lower age of first use.

BOTANY

Cannabis sativa grows in the tropical, subtropical, and temperate regions. It is generally considered a single species of the mulberry family (Moraceae) with multiple morphological variants (e.g., *C. indica* or *C. americana*). It is an herb of varying size; some are quite bushy and attain a height of 10 to 15 feet (3 to 4.6 m). Due to genetic differences, some plants produce strong fibers (but little THC) and others produce a substantial quantity of THC but weak fibers. The fiber-producer is grown commercially for cloth, rope, roofing materials, and floor coverings; this was cultivated as a cash crop in colonial America for such purposes (Hart, 1980). During World War II, when it appeared that the United States might be cut off from Southeast Asian hemp, necessary to the war effort, the plants were cultivated in the mid-western states. Some of them continue to grow wild today,

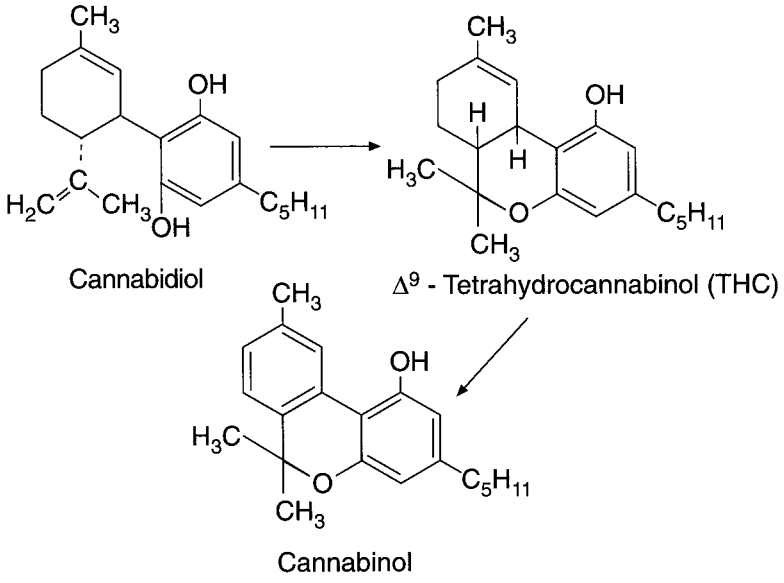

Figure 1
Biosynthetic Pathway of Cannabinoids

but since they are of the fiber-producing variety, they contain little drug content.

The drug-producing variety is widely cultivated in societies where its use is condoned. Illegal crops are also planted, some in the United States. The choice parts are the fresh top leaves and flowers of the female plant. The leaves have a characteristic configuration of five deeply cut serrated lobes. When they are harvested, they often resemble lawn cuttings—which accounts for the slang term "grass."

CHEMISTRY

The collective name given to the terpenes found in *Cannabis* is cannabinoids. Most of the naturally occurring cannabinoids have now been identified, and three are the most abundant—cannabidiol (CBD), tetrahydrocannabinol (THC), and cannabinol (CBN). The steps from CBD to THC to CBN represent the biosynthetic pathway in the plant. THC is an optically active resinous material that is very lipid-soluble but water-insoluble; these physical properties make pharmacological investigations difficult, since various nonpolar solvents must be used. Although many other materials have been found in this plant, the cannabinoids are unique to it and THC is the only one with appreciable mental affects. THC is believed to be largely, if not solely, responsible for the effects desired by those who use *Cannabis* socially. Virtually all the effects produced by smoking or eating some of the whole plant can be attained by using THC alone.

USE AS A SOCIAL DRUG

Cannabis grows so easily that it is called a weed. In the United States, where it remains illegal, it is possible for those who wish to use it as a social drug to grow their own supply. The ease of cultivation keeps the price of imported illicit marijuana down, which helps account for some of its widespread use. Such cultivation is, however, as illegal as possession of the drug obtained from illicit "street" sources.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy; High School Senior Survey*)

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LEO E. HOLLISTER

CASUAL DRUG USE *See* Addiction: Concepts and Definitions

CATECHOLAMINES The catecholamines are a series of structurally similar amines (e.g., DOPAMINE, epinephrine, NOREPINEPHRINE that function as hormones, as NEUROTRANSMITTERS, or both. Catecholamines are produced by the enzymatic conversion of tyrosine, sharing the chemical root of 3,4-dihydroxyphenylethanolamine. The three major catecholamines (mentioned above) derive from sequential enzymatic reactions-tyrosine is converted to dihydroxyphenylacetic acid (dopa); dopa, which is not an end product but a common intermediate (and the medication of choice for Parkinson's disease), is converted to dopamine; dopamine is converted to noradrenaline (also called norepinephrine); and noradrenaline is converted to adrenaline (also called epinephrine). These substances are the neurotransmitters for the sympathetic neurons (nerve cells) of the autonomic nervous system, as well as for three separate broad sets of brain neuropathways.

FLOYD BLOOM

CATHINONE *See* Khat

CAUSES OF SUBSTANCE ABUSE This section contains articles on some of the many factors thought to contribute to substance use, abuse, and dependence. It includes discussions of *Drug Effects and Biological Responses*, *Genetics Learning*, and an article on the *Psychological (Psychoanalytic) Perspective. Sociocultural* causes and *Vulnerability*, are discussed in several articles throughout the Encyclopedia, for example, *Ethnicity and Drugs*, *Families and Drug Use*, *Poverty and Drugs*. See also the article *Disease Concept of Alcoholism and Drug Addiction* and the section on *Vulnerability*.

Drug Effects and Biological Responses Although many indirect factors lead to an individual abusing drugs, a person's response to the effects of the drugs themselves contribute both to their use and abuse. These drug effects should be considered in relation to four phases of drug use: (1) initiation-consolidation, (2) maintenance, (3) repeated withdrawal and relapse, and (4) postwithdrawal. During the initiation-consolidation phase, behaviors that lead to the taking of a drug are gradually strengthened through operant and classical conditioning processes and by biochemical changes in the brain. The drug effects include a cascade of discriminative or internally appreciated drug cues (i.e., subjective effects). The presence of these cues often leads to associated autonomic responses and reports of urges in humans. These responses and urges may result in an unfolding of a sequence of behavioral and physiological events leading to continued drug consumption.

After a pattern of chronic drug use is established, individuals may become tolerant to certain effects of a drug. In addition, they may experience withdrawal effects when they stop taking a drug. Withdrawal effects are often opposite to the drug-induced state and usually involve some form of dysphoria—a state of illness and distress. Over time, withdrawal effects become associated with stimuli in the environment, as was the case for the euphoric and other direct effects of the drug. Because of operant and classical conditioning processes, these associated stimuli can then produce conditioned effects that are often characterized as urges or cravings, and that may trigger relapse.

The underlying NEUROTRANSMITTER systems within the brain, subserving these behavioral features of drug effects, are just beginning to be understood. Early RESEARCH on the neural substrates of reward in general used electrical brain stimulation as the reward. For example, Olds (1977) found that rats would press a lever to receive a brief electrical pulse to the hypothalamus; rats would press this lever to such an extent that they did not engage in consummatory reward activities such as eating and drinking. Subsequent research indicated that activation of certain systems in the brain, namely the mesolimbic and nigrostriatal dopaminergic systems, were most sensitive to brain stimulation reinforcement. Several theories have been suggested to explain the importance of the brain reward system for the survival of species (Conrad, 1950; Glickman & Schiff, 1967; O'Donahue & Hagmen, 1967; Roberts & Carey, 1965).

Further research demonstrated that most drugs of abuse lower the threshold for this brain stimulation reward, thus suggesting that such drugs may activate the same, or similar, reward pathways (see Koob & Bloom, 1988). As will be seen, furthermore, the reinforcing effects of the drugs them-

selves—that is, effects that lead individuals to take the drugs—are directly mediated by these REWARD systems. The fact that many drugs induce activation of these systems may indicate a mechanism underlying the addiction-related effects of drugs of abuse.

COCAINE AND OTHER STIMULANTS

COCAINE is an indirect catecholamine agonist that acts by blocking the reuptake of monoamines, including DOPAMINE (DA), NOREPINEPHRINE (NE), and serotonin (5-HT). During the process of reuptake, the previously released neurotransmitter is actively transported back from the synaptic cleft into the presynaptic terminal of the neuron where the neurotransmitter was produced and released (Pitts & Marwah, 1987). In contrast to cocaine, AMPHETAMINE acts not only by inhibiting uptake, but also by releasing catecholamines from newly synthesized storage pools from the presynaptic terminal of the neuron (e.g., Carlsson & Waldeck, 1966).

Amphetamine and cocaine are both potent PSYCHOMOTOR stimulants. They produce increased alertness and energy and lower ANXIETY and social inhibitions. The acute reinforcing actions of the stimulants are primarily determined by their augmentation of DA systems. With prolonged consumption: (1) acute TOLERANCE becomes substantial, and (2) the individual starts to regularly consume higher and many more doses if the resources are available. Over time, in high-dose regimens, the behavioral pattern of use becomes stereotyped and restricted. In settings of low availability, the individual focuses on the acquisition and consumption of the drug. These effects of stimulants occur within weeks or months of continued use. The individual may also start "bingeing" during this period. A binge is characterized by the readministration of the drug approximately every ten to twenty minutes, resulting in frequent mood swings (i.e., alternations of highs and lows). Cocaine binges typically last twelve hours, but may last as long as seven days.

It has been proposed that cocaine abstinence consists of a three-phase pattern: crash, WITHDRAWAL, and extinction (Gawin & Kleber, 1986; Gawin & Ellinwood, 1988). The crash phase immediately follows the cessation of a binge and is characterized by initial depression, agitation, and anxiety. Over the first few hours, drug craving is replaced by an intense desire for sleep. During this time, the individual may use ALCOHOL, BENZODIAZEPINES, or OPIATES to induce sleep. Following the crash, hypersomnolence (excessive sleep) and hyperphagia (excessive appetite) develop. Following the first few days of hypersomnolence and hyperhagia, other symptoms emerge that are the opposite of the effects of cocaine—withdrawal symptoms. During this withdrawal period, which lasts three to ten days, individuals experience decreased energy, limited interest in their environment, and anhedonia. They are also strongly susceptible to RELAPSE and starting another binge cycle (Gawin & Ellinwood, 1988; Gawin & Kleber, 1986; Jaffe, 1985). This phase is followed in time by the extinction phase, in which relapse to cocaine use is prevented. During the extinction phase, brief periods of drug CRAVING also occur. These episodes of craving are thought to be triggered by conditioned stimuli that were previously associated with the drug. If the individual experiences these cues without the associated drug effects—that is, resists relapse—then the ability of these cues to elicit drug cravings should diminish over time, which in turn should lessen the probability of relapse (Gawin & Ellinwood, 1988).

As already noted, acute administration of cocaine produces profound inhibition of dopaminergic uptake (Fuxe, Hamberger, & Malmfors, 1967). The relation between cocaine dose and DA levels is linear; therefore, larger amounts of cocaine result in higher extracellular DA levels. These levels of DA are thought to underlie the reinforcing effects of cocaine (Gawin & Ellinwood, 1988). Because both cocaine and amphetamine result in enhanced dopaminergic neurotransmission, thereby producing elevated extracellular levels of catecholamines, these elevated neurotransmitter levels would presumably have local time-dependent inhibitory effects on the enzyme tyrosine hydroxylase, which is responsible for controlling their rate of synthesis. Therefore, this substrate-inhibitory mechanism might compensate for the increased catecholamine levels and activity by decreasing their synthesis. Galloway (1990) found that cocaine, in a way that was consistent with this proposition, decreased DA synthesis in a dose-dependent manner in various brain regions.

Chronic, intermittent stimulant use (e.g., 1–2 injections per 24 hrs) produces other behavioral

effects besides euphoria and increased energy: (1) stimulant psychosis, which is characterized by paranoia, anxiety, stereotyped compulsive behaviors, and HALLUCINATIONS, and (2) sensitization or "reverse tolerance." Sensitization refers to the fact that the effects of cocaine are progressively enhanced. Although sensitization has been demonstrated in animal studies, it is not clear whether it occurs in humans. There are nevertheless several possible explanations for sensitization. First, because cocaine blocks dopaminergic, uptake, chronic cocaine use could somehow harm the functioning of the dopamine uptake mechanism; the evidence regarding this possibility is equivocal (Zahniser et al., 1988b). Second, sensitization could also be the result of enhanced dopaminergic release, similar to that found to be chronic after amphetamine administration (Castaneda. Becker, & Robinson, 1988). Akimoto, Hammamura, & Otsuki (1989) found enhanced DA release in the striatum one week following chronic cocaine administration. Similar data has been obtained by others (Kalivas et al., 1988; King et al., 1993; Pettit el al., 1990). Cocaine levels in blood and cerebrospinal fluid have also been reported to be elevated in chronically treated subjects (Reith, Benuck, & Lajtha, 1987); however, these increases cannot account for most of the change in DA release (Pettit et al., 1990). Furthermore, some researchers report no consistent effects in this regard. Third, there could be changes in autoreceptor sensitivity following chronic cocaine administration. Autoreceptors for particular neurotransmitters are those receptors that reside on the same NEURON that releases the NEUROTRANSMITTER. The autoreceptors on the somatodendritic area of neurons regulate impulse flow along the neuron, whereas autoreceptors on the terminal regions of the neuron regulate the amount of neurotransmitter released per impulse and neurotransmitter synthesis (Cooper, Bloom, & Roth, 1986). Sensitization could, therefore, be the result of decreased autoreceptor sensitivity. Such subsensitivity would result in either increased impulse flow, if somatodendritic autoreceptors were altered, or increased neurotransmission/synthesis, if terminal autoreceptors were altered. The net effect, in either case, would be an increase in dopaminergic neurotransmission. There is some evidence of decreased somatodendritic autoreceptor sensitivity twenty-four hours after the cessation of chronic cocaine administration (Henry, Greene, & White, 1989). However, seven days after termination of daily cocaine injections, when cocaine-induced sensitization is still fully present, somatodendritic autoreceptors are no longer reduced in sensitivity (Zhang, Lee, & Ellinwood, 1992). Evidence regarding changes in terminal autoreceptor sensitivity is mixed. Dwoskin et al. (1988) found that terminal autoreceptors were supersensitive, not subsensitive, to a DA agonist twenty-four hours following chronic cocaine use. Henry et al. (1989) also found that terminal autoreceptors were supersensitive to DA following chronic daily cocaine injections. Although autoreceptor supersensitivity cannot explain sensitization, it is a possible mechanism underlying the previously described anhedonia and anergy experienced by cocaine abusers during the withdrawal phase. Fourth and last, there could be an increase in the number or sensitivity of postsynaptic DA receptors. The evidence regarding this hypothesis is also mixed (Zahniser et al., 1988a). For example, Peris et al. (1990) found an increased number of postsynaptic D2 receptors in the NUCLEUS ACCUMBENS one day following cessation of chronic cocaine administration; however, after one week the number of receptors had returned to normal levels. In contrast, there is some evidence that postsynaptic DA receptors are decreased following chronic cocaine use. Volkow et al. (1990) found lower uptake values for [18 F]n-methylspiroperidol in human cocaine users who had been abstinent for one week, as compared with normal subjects. Uptake values were similar, however, for normal subjects and cocaine users who had been abstinent for one month.

In contrast with these results, Yi and Johnson (1990) have reported that chronic intermittent cocaine use impairs the regulation of synaptosomal 3[H]-DA release by DA autoreceptors, thus suggesting a subsensitivity or down-regulation of release-modulating DA autoreceptors seven days after chronic cocaine administration. The differences in the results of the Yi and Johnson (1990) and the Dwoskin et al. (1988) studies may be due to differences in the administration schedules or in the procedures used to measure autoreceptor sensitivity.

In contrast with the changes induced by intermittent but chronic drug administration, a regimen that involves the chronic administration of steady-state levels of drug results in decreased DA overflow when striatal brain slices are perfused with cocaine. This result may be due to the development of su-

TABLE 1
Effects of Chronic Cocaine and Amphetamine Administration on Dopaminergic Functioning

	Amphetamine		*Cocaine*	
	Day 1	Day 7	Day 1	Day 7
Autoreceptor Sensitivity	sub	super	sub	super
Receptors	decreased	decreased	unclear	unchanged
Biosynthesis	reduced	reduced	unchanged	unchanged
Uptake Sites	decreased	decreased	unchanged	unchanged

persensitive autoreceptors. Autoreceptor supersensitivity would result in decreased dopaminergic activity. There is some support for this hypothesis from research involving the chronic administration of amphetamine. Lee and colleagues (Lee, Ellinwood, & Nishita, 1988; Lee & Ellinwood, 1989) found that twenty-four hours after withdrawal from a week of continuous administration of amphetamine, all indicators of autoreceptor activity demonstrated a pronounced subsensitivity. Similar results have been found following the continuous infusion of cocaine (Zhang et al., 1992). However, by the seventh day of withdrawal (a period associated with anergia, irritability, and "urges" in human stimulant abusers), nigrostriatal somatodendritic autoreceptors progress from an initial subsensitivity to a supersensitive state, whereas terminal autoreceptors are normosensitive. The changes in sensitivity of receptors clearly depend on the way the drug is administered and which receptors are evaluated. The evidence, moreover, is not always consistent.

There is also evidence that chronic cocaine administration produces neurotoxicity—i.e., actual destruction of neural tissue—although there are conflicting results and the relationship of this neurotoxicity to the addiction process is unclear. For example, Trulson and colleagues (1986) demonstrated decreased tyrosine hydroxylase activity sixty days after chronic cocaine treatment (see also Trulson & Ulissey, 1987), thereby indicating decreased DA synthesis. (Tyrosine hydroxylase is the rate-limiting step in the biosynthesis of DA; Cooper el al., 1986.) Similarly, Taylor and Ho (1978) found that chronic administration of cocaine decreased tyrosine hydroxylase activity in the caudate, but Seiden and Kleven (1988) were unable to replicate the findings of Trulson. As contrasted with the inconclusive results on cocaine, research involving amphetamine is much clearer. First, chronic METHAMPHETAMINE administration reduces the number of DA uptake sites (Ricaurte, Schuster, & Seiden, 1980; Ricaurte, Seiden, & Schuster, 1984). Second, DA and tyrosine-betahydroxylase levels are reduced for extended periods following chronic amphetamine administration (Ricaurte et al., 1980, 1984). Third, there is evidence of neuronal degeneration, chromatolysis, and decreased catecholamine histofluorescence (Duarte-Escalante & Ellinwood, 1970).

As with cocaine's effects on DA reuptake, cocaine also blocks 5-HT reuptake. Since activation of 5-HT postsynaptic receptors affects neurotransmission in neurons that release DA, this blockade prolongs the inhibitory effects of 5-HT on dopaminergic neurotransmission (Taylor & Ho, 1978). However, cocaine also inhibits the firing rates of dorsal raphe 5-HT neurons (Cunningham & Lakoski, 1988, 1990). Thus, acutely the net effect of cocaine on 5-HT neurotransmission in the nucleus accumbens will depend on the relative contributions of uptake inhibition, which would increase synaptic 5-HT, and inhibition of neuronal firing, which would decrease synaptic 5-HT. Broderick (1991) reported that acute, subcutaneous injections of cocaine resulted in a dose-dependent increase in DA levels, as measured by dialysis of the nucleus accumbens. This suggests a decrease in 5-HT levels that may result from activation of somatodendritic 5-HT autoreceptors located in the dorsal raphe nucleus. Acute cocaine administration has indeed been reported to almost completely inhibit the basal firing rate of dorsal raphe serotonergic neurons.

As with the effects of chronic amphetamine administration on the functioning of DA systems,

chronic methamphetamine administration has been shown to induce pronounced long-term changes in tryptophan hydroxylase activity, as well as in 5-HT content and number of uptake sites (Ricaurte et al., 1980). The effects of chronic cocaine on serotonergic functioning are less well established. For example, Ho et al. (1977) found decreased levels of 5-HT following chronic cocaine administration. Seiden and Kleven (1988). however, failed to find any effects of chronic cocaine on the biosynthesis of serotonin.

Some of these discrepancies can be reconciled by the fact that different chronic dosing regimens produce different changes in 5-HT systems. For example, Cunningham and colleagues found that daily injections of cocaine resulted in an increased sensitivity of dorsal raphe somadendritic 5-HT autoreceptors to cocaine's inhibitory effects as measured by electrophysiological techniques (Cunningham & Lakoski, 1988, 1990). These results are consistent with the behavioral data of King and colleagues (1993a), who found that daily cocaine injections produced an enhanced inhibitory effect of NAN-190 on cocaine-induced locomotion and an enhanced excitatory effect of 8-OH-DPAT on locomotion. In contrast with these results, the continuous infusion of cocaine via an osmotic minipump results in a decreased sensitivity of dorsal raphe somadendritic 5HT autoreceptors and a decreased excitatory effect of 8-OH-DPAT on locomotion (King, Joyner, & Ellinwood, 1993b; King et al., 1993b).

Interestingly, the depletion of 5-HT or reduction of 5-HT neurotransmission is associated with impulsive behavior. For example, Linnoila et al. (1983) found that violent offenders with a diagnosis of PERSONALITY DISORDER associated with impulsivity had lower levels of 5-hydroxyindoleacetic acid (5-HIAA, the metabolite of 5-HT) than other offenders. Bouilliouc et al. (1978, 1980) reported suppressed 5-HIAA levels in the cerebrospinal fluid (CSF) of XXY-chromosome (aggressive) institutionalized criminals. Lithium has been successfully used to treat violent offenders (Sheard, 1971, 1975; Sheard et al., 1976; Tupin et al., 1973). Lithium treatment produces increases in CSF 5-HIAA in humans (Fyro et al., 1975; Sheard & Aghajanian, 1970), and central nervous system (CNS) 5-HT in nonhumans (Sheard & Aghajanian, 1970; Mandell & Knapp, 1977); this indicates that increases in 5-HT are associated with decreases in violent and aggressive behavior. After extensively reviewing the literature, Brown and Linnoila (1990) concluded that low levels of CSF 5-HIAA are related to disinhibition of aggressive/impulsive behavior and not to antisocial acts in and of themselves. The transition to high-dose cocaine use might be considered impulsive behavior because the individual is focusing on the immediate, short-term advantages of drug consumption while ignoring the long-term advantages of drug abstinence. Hence, the 5-HT receptor supersensitivity, and resulting inhibition of 5-HT neurotransmission, may be a contributing factor to the development of the high-dose, bingelike pattern of cocaine abuse.

OPIATES

The OPIATES are derived from the POPPY plant and have been used for centuries. A number of types of endogenous opiate RECEPTORS have been identified and their locations mapped. There are high concentrations of opiate receptors in the caudate nucleus, nucleus accumbens, periventricular gray region, and the nucleus arcuatus of medobasal hypothalamus (Pert, Kuhar, & Snyder, 1975, 1976). These areas may be differently involved in the reinforcing, aversive, and dependence-producing effects of the opiates. Furthermore, different receptor subtypes may mediate the different effects of the opiates.

The opiates produce ANALGESIA, changes in mood (e.g., euphoria and tranquility), drowsiness, respiratory depression, and nausea (Jaffe & Martin, 1990). These drugs also reduce motivated behavior; there is a decrease in appetite, sexual drive, and aggression. Intravenous administration of opioids results in initial effects of flushing of the skin and sensations in the abdominal regions that have been likened to a sexual orgasm (Jaffe, 1990).

With continuous use of opioids, marked tolerance develops to some, but not all, of the effects of these drugs. Tolerance to opioids is generally characterized by a shorter duration of effect and attenuated analgesia, euphoria, and other CNS-depressant effects; however, there is less tolerance to the lethal effects of opiates. Therefore, if an individual administers ever larger doses to obtain the same effect (e.g., the rush or high), this may increase the probability of a lethal overdose (Jaffe, 1990).

Although the course and severity of withdrawal symptoms following opiate abstinence depend on which opiate was used, the dose and pattern of consumption, the duration of use, and the interdose interval, the opiate withdrawal syndrome follows the same general progression. Approximately eight to twelve hours after the last dose, individuals experience yawning, lacrimation, and rhinorrhea; twelve to fourteen hours after the final dose, they may fall into a fitful, restless sleep and awaken feeling worse than when they went to sleep. With the continuation of opiate withdrawal, they experience increasing dysphoria, anorexia, gooseflesh, irritability, agitation, and tremors. At the peak intensity of the withdrawal symptoms, they may experience exacerbated irritability, insomnia, intense anorexia, weakness, and profound depression. Common symptoms include alternating coldness and intense skin flushing and sweating, vomiting and diarrhea (Jaffe, 1990). This pattern of symptoms indicates that during the initial withdrawal phase there is a generalized CNS hyperexcitability. Thus, the addicted opiate abuser continues to recycle opiate use to both avoid or terminate the wtihdrawal symptoms, and to reexperience the euphoric effects. This powerful combination of euphoria, tolerance, and withdrawal can lead to profound levels of addiction.

Studies have found that rats and monkeys will self-administer opioids, thus indicating that these drugs serve as reinforcers (Koob & Bloom, 1988). Chronic opioid administration results in physical dependence, as demonstrated by the presence of a withdrawal syndrome following drug cessation. Most clinicians hold the classic position that PHYSICAL DEPENDENCE (i.e., avoidance of withdrawal symptoms) is a major motivating factor in opiate self-administration, but evidence indicates that reinforcement and withdrawal are separate processes. Bozarth and Wise (1984) demonstrated that rats will self-administer morphine into the ventral tegmental area without the presence or development of any apparent withdrawal symptoms. Chronic administration of morphine into the periaqueductal gray area, however, produces signs of a strong withdrawal syndrome.

Several lines of evidence indicate that dopaminergic neurotransmission may partially mediate the reinforcing effects of opiate administration. First, injection of met-enkephalin into the ventral tegmental area results in increases in DA release in the nucleus accumbens (Di Chiara & Imperato, 1988). Second, although opiates generally produce sedation, low doses of systemic morphine increase locomotor activity (Domino, Vasko, & Wilson, 1976). Third, injections of morphine into the ventral tegmental area produce circling behavior (Holmes, Bozarth, & Wise, 1983); injections of opiates into the ventral tegmental area produce increased locomotion, as with systemic injections of opiates, thereby suggesting increased dopaminergic transmission (Blaesig & Herz, 1980). Fourth, selective lesions of the dopaminergic system decrease opiate self-administration, although not to the extent of affecting cocaine self-administration (Bozarth & Wise, 1985). Fifth, rats learn to self-administer opiates directly into the ventral tegmental area (Bozarth & Wise, 1981), rats also inject opiates into the nucleus accumbens and the lateral hypothalamus (Goeders, Lane, & Smith, 1984). Sixth, administration of the D1 antagonist SCH 23390, but not the D2 antagonists sulpiride and spiperone, block the reinforcing effects of morphine.

Ettenberg et al. (1982) found no effect of alpha flupenthixol, primarily a D2 antagonist, on heroin self-administration, although the same doses decreased cocaine self-administration. Similar results have been reported by others using other dopaminergic antagonists (De Wit & Wise, 1977). Thus, both place preference and self-administration procedures indicate that opiates are not reinforcing through D2 receptors, which are vital to stimulant reinforcement. These results indicate that opiate reinforcement is at least partially independent of the D2 stimulant type of reinforcement, yet they do act through a dopaminergic mechanism to induce a significant part of their effects.

Chronic administration of opiates produces several behavioral and neurochemical effects that may be related to their reinforcing effects. First, chronic administration of MORPHINE results in the augmentation of the behavioral effects of low doses of morphine. In other words, subjects undergoing chronic opiate administration become sensitized to the behavioral effects of morphine (Ahtee, 1973, 1974). Second, chronic morphine administration results in decreased DA turnover in the striatum and limbic system during withdrawal (Ahtee & Attila, 1980, 1987). Third, in mice withdrawn from morphine, the synthesis and release of DA are attenuated (Ahtee et al., 1987); similar results have been obtained with human heroin addicts in which CSF

homovanillic acid concentrations were decreased (Bowers, Kleber, & Davis, 1971).

These results indicate that during chronic morphine administration there is a down-regulation of the dopaminergic system and a neuroadaptation to this depletion. During withdrawal from opiate administration there is an augmentation of dopaminergic mechanisms. Indeed, during withdrawal rats are sensitized to the behavioral effects of apomorphine (Ahtee & Atilla, 1987), and small doses of morphine increase striatal homovanillic acid levels more in withdrawn than in control rats, thereby indicating that the dopaminergic system is sensitized at this point (Ahtee, 1973, 1974). Thus, some of the withdrawal symptoms (e.g., irritability and dysphoria) may be mediated by changes in dopaminergic functioning.

Acute administration of opiates increases the synthesis of 5-HT and the formation of 5-HIAA, and these effects are eliminated by the administration of opiate antagonists (Ahtee & Carlsson, 1979), thus suggesting that opiate administration results in increased serotonergic functioning. Indeed, acute administration of dynorphin-(1–13), while it decreases striatal dopamine, actually increases striatal serotonin (Broderick, 1987). This increased serotonergic functioning may contribute to the "post-consummatory calm" produced by opiate drugs: Increasing serotonergic functioning would tend to inhibit incentive-motivated behaviors and produce a calm, tranquil state. Indeed, the atypical anxiolytic drug buspirone exerts its anxiety-reducing effects via serotonergic activation.

During withdrawal from chronic opioid administration, 5-HIAA levels are decreased (Ahtee, 1980; Ahtee et al., 1987). This pattern of serotonin results could well cause increased impulsivity and a higher probability of relapse, similar to that described earlier in relation to the psychomotor stimulants.

In summary, like cocaine, the opiates are consumed because of their reinforcing properties. These reinforcing properties are the result of activation of endogenous opiate receptors; furthermore, activation of the dopaminergic system modulates the reinforcing effects of opiates. During chronic opiate administration, subjects become physically dependent. There is an increase in dynorphin levels that may mediate some of the aversive aspects of the withdrawal syndrome (e.g., decreased dopaminergic functioning). Furthermore, during chronic administration, there is functional down-regulation of both the dopaminergic and serotonergic systems. Upon withdrawal from opiates, there is a subsequent supersensitivity of the dopaminergic system. This dopaminergic supersensitivity may be involved in opiate craving and general irritability during withdrawal.

(SEE ALSO: *Addiction: Concepts and Definitions; Brain Structures and Drugs; Opioids: Complications and Withdrawal; Research: Animal Model; Tolerance and Physical Dependence; Wikler's Pharmacologic Theory of Drug Addiction*)

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Genetic Factors Why do some people become alcoholics or drug addicts, while others who try the same substances, even to excess, avoid the snare of dependence? Could there be a genetic basis for such behavior? Given the strong familial patterns of substance abuse that often occur, the answer may be yes—in some cases. Evidence points toward at least one inheritable form of ALCOHOLISM; a genetic vulnerability to drug abuse has not been confirmed by research, although it may exist.

Researchers know that many environmental and psychological factors are implicated in addictions, including a dysfunctional family, poor academic performance, delinquent behavior, and perhaps most importantly, easy access to drugs. The abuser's personality is also a factor, and alcoholics, in particular, often exhibit similar traits: an inability to handle frustration, extreme sensitivity, poor self-image, and isolation. While some of these qualities may be inherited, they do not prove a direct genetic link to alcohol or drug abuse—they simply act as a petri dish in which self-destructive behavior can develop.

Physiology, on the other hand, is determined directly by genetic inheritance, and it is in this arena that scientists have begun to discover what may be an inborn predispostion to some forms of substance abuse-particularly alcoholism. Heredity may also influence an individual's response to drug ingestion by either heightening pleasant sensations or suppressing unpleasant ones, such as the "flushing" reaction to alcohol (which can include elevated skin temperature, increased pulse rate, headache, and nausea) and the queasiness caused by opioids. An individual's response to drugs may also be determined by genetically influenced differences in the receptor proteins on which the drugs act, differences in the enzymes that metabolize these drugs, or differences in the proteins that remove these drugs from their sites of action.

GENETIC PREDISPOSITION

Researchers suspect that a tendency to alcoholism can be inherited, and studies of twins, adopted children of alcoholics, and half siblings of alcoholic parents have confirmed these suspicions, although a direct genetic link has yet to be found. Studies have shown that the first-degree relatives of alcoholics are fourteen times more likely to be addicts themselves. Even when the children of alcoholic parents are adopted into new families, their risk of becoming alcoholics is still far higher if one of their biological parents was an alcoholic. All of these statistics are especially significant for males, who seem more than twice as likely as females to inherit drug and alcohol dependence.

Animal studies indicate that an inherited difference in nerve cell membranes may be a factor in alcoholism. Alcohol lowers the viscosity of the nerve-cell membrane, which may interrupt neural excitation or interfere with the membrane proteins involved with neurotransmission. Studies performed on two genetically different strains of mice showed that alcohol-sensitive mice had nerve-cell membranes that were more susceptible to the viscosity-lowering effects of alcohol than mice that were less susceptible to alcohol (Goldstein, 1982).

Another genetic factor may be the alteration of membrane-bound neuronal enzymes, such as sodium-potassium ATPase, an ion pump that is responsible for the movement of sodium-potassium ions through the cell membrane. Alcohol inhibits the sodium-potassium ATPase, leading to speculation that there may be a genetic predisposition to alcoholism in people who show an atypical resistance to the alcohol-induced inhibition of this enzyme.

Alcohol also affects the proteins that make up RECEPTORS, including the NMDA receptor, which mediates neuronal excitation following release of the NEUROTRANSMITTER glutamate and is the major excitatory pathway in the brain, and the GABA receptor, the major inhibitory pathway in the central nervous system. Changes in either of these receptor proteins could predispose an individual to alcoholism by altering their response to the drug.

The A1 allele. Alcohol interferes with the release of the neurotransmitters NOREPINEPHRINE and DOPAMINE. One promising area of research indicates that some *severe* forms of alcoholism (and possibly drug abuse) may occur in subjects who have up to 30 percent fewer DOPAMINE receptors in their brains than usual; this deficiency is caused by a variant gene for the dopamine D2 receptor, called the A1 allele.

People with this trait may be far less able than most to find enjoyment in everyday activities and have much greater difficulty coping with the stresses of life. One researcher, in fact, has tied the presence of the A1 allele to a condition he calls the "reward-deficit syndrome" (Blum et al.). Possession of the A1 allele is identified with a range of impulsive behaviors, possibly caused by a brain chemistry that spurs people to substance abuse and other self-destructive behaviors in the search for neurochemical satisfaction.

Because alcohol and many other drugs release a flood of dopamine into the brain, addicts may turn to alcohol and drugs to get the feelings they crave. A smaller-than-usual number of dopamine receptors may also diminish or suppress the unpleasant sensations many people feel when they become intoxicated and heighten the inebriation, which increases the likelihood that the substance will be used again and in greater quantities.

Predisposition against substance abuse. Genetic predisposition may also *inhibit* the development of alcoholism because heredity may determine the way people metabolize alcohol. The major chemical pathway for the metabolism of alcohol requires two enzymes: alcohol dehydrogenase, which converts alcohol to acetaldehyde; and acetaldehyde dehydrogenase, which converts acetaldehyde to acetic acid. Acetic acid is then converted by a series of enzymes to carbon dioxide and water.

Recent studies indicate that there is a genetic difference in the enzymes involved in the alcohol metabolism of Asians. In a significant proportion of Asians, alcohol dehydrogenase is more active than in Caucasians; it rapidly converts alcohol to acetaldehyde. In these same Asian populations, there is a genetic deficiency in acetaldehyde dehydrogenase, so that acetaldehyde accumulates and produces the unpleasant flushing reaction described above. This same rapid and unpleasant accumulation of acetaldehyde is what alcoholic patients risk when they take the drug DISULFIRAM (Antabuse) to deter them from drinking. The acetaldehyde dehydrogenase deficiency may actually deter many Asians from abusing alcohol. Asians that do become alcoholics do not exhibit the acetaldehyde dehydrogenase deficiency common in the general Asian population.

PSYCHIATRIC DISORDERS

In addition to a physiology that changes their metabolization of and reaction to drugs and alcohol, certain psychiatric diagnoses are regularly overrepresented among alcoholics and drug dependents. These include depression, anxiety disorders, and ANTISOCIAL PERSONALITY, all of which may be inherited. When these complications are factored in, it becomes difficult to distinguish genetically based substance abuse from risky, self-destructive behavior caused by mental illness.

SAMPLE AND CONTROL VARIABLES

Many factors affect a person's susceptibility to drug abuse, which makes studying the genetic bases for addiction difficult, particularly where drug addiction is concerned. Studies must match control and drug-abusing populations adequately, and the sample size must be large enough to detect small genotype changes from background influences. Individual variables (such as severity of dependence) need to be consistent between research groups. It's also difficult to determine whether an individual currently in the control group will express addictive behavior in the future. For these and other reasons, scientists have been unable to prove a definitive link between an individual's genetic makeup and a tendency to substance abuse, although they strongly suspect its existence, especially in the more severe forms of alcoholism and drug dependence.

(SEE ALSO: *Conduct Disorder; Complications: Mental Disorders; Epidemiology of Drug Abuse; Women*

and Substance Abuse; Vulnerability As Cause of Substance Abuse)

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Learning The role played by learning factors in drug and alcohol abuse has recently received much attention. Two basic learning mechanisms are thought to be activated when an organism repeatedly self-administers a psychoactive substance. First, classical conditioning processes are engaged when environmental stimuli signal the upcoming effects of the drug. Second, operant conditioning occurs as an organism learns that particular behaviors lead either to a drug reward or to punishment. The effects of these two processes presumably interact, and they are thought to influence repeated drug use and/or relapse to drug use following a period of abstinence.

Classical conditioning occurs when an organism learns about a contingency between two events in the external environment. The most common situation involves learning that a biologically neutral event (the conditioned stimulus, CS, such as a light or a tone) signals the upcoming occurrence of a biologically relevant event (the unconditioned stimulus, US, such as the effects of a drug or the WITHDRAWAL syndrome from absence of a drug). As a result of this signaling relationship, the CS produces conditioned responses (CRs), related to the US in use. In the area of drug use, a number of investigators have suggested that environmental events that signal upcoming withdrawal or drug use in humans elicit CRs—which motivate further drug taking (Baker, Morse, & Sherman, 1987).

Operant conditioning involves learning about contingencies between behaviors and their outcomes. A typical operant conditioning situation sets up contingencies between three different events—a response; the outcome of that response (the reward or reinforcer); and the stimulus situation in which that response—outcome relationship is established (the discriminative stimulus). Drugs of abuse function as potent reinforcers for human addicts, since a variety of behaviors are directed solely toward their attainment and use. Consequently, understanding the rules governing the acquisition of operant behaviors directed toward drug reinforcers may be critical to understanding addiction.

Classical and operant conditioning processes may be activated simultaneously during drug seeking and self-administration. Events that have consistently signaled drug use may eventually come to evoke CRs in the form of craving—urges to use the drug. In this way, signals of drug use may act as discriminative stimuli motivating the drug user to begin drug-seeking behavior. For example, walking past a known dealer might act as a CS for a heroin addict, evoking the CR of craving for HEROIN. This craving response might then increase the likelihood of behaviors that are rewarded by the desired drug effects—buying and preparing heroin.

OPERANT CONDITIONING WITH DRUG REINFORCERS

A large body of data shows that virtually all drugs of dependence in human beings act as reinforcers for animals in operant-conditioning situations. Typical studies on the reinforcing properties of drugs involve rats or monkeys fitted with venous catheters, through which a drug can be administered directly. Responses directed toward an object, such as a lever, result in infusions of the drug.

The basic finding of such studies has been that a wide variety of abused drugs—including COCAINE, MORPHINE, heroin, *d*-AMPHETAMINE, pentobarbi-

tal, and ALCOHOL—all serve to establish and maintain operant behaviors in animals. Other drugs with a lesser abuse potential in humans—such as aspirin, tricyclic antidepressants, hallucinogens, and opioid mixed agonist/antagonists—fail to support responding.

The degree to which a given drug of abuse reinforces behavior appears to depend more on the schedule of reinforcement of the drug than on its intrinsic properties. A schedule of REINFORCEMENT refers to the pattern of access provided to the reinforcing event. For example, ratio schedules require an animal to make some predetermined number of responses before a reinforcer is given. Yet interval schedules are set up so that responses are effective at producing a reinforcer only after a delay following the previous one. Reinforcers in ratio schedules depend solely on the number of responses made; therefore, these schedules typically result in higher response rates than interval schedules in which responses made too early are ineffective. Because reinforcement schedules largely determine the rate of responding in a given situation, the abuse potential of the various drugs cannot be reliably assessed by comparing how quickly animals respond for each substance.

Other techniques for making such comparisons are available, however, and one technique for comparing the reinforcing properties of various substances involves calculating for each a so-called breaking point under a fixed ratio schedule of reinforcement. A fixed ratio schedule requires that an animal make a fixed number of responses (the ratio) for each reinforcement received. For a given drug dose, the breaking point is reached when a ratio too high to support responding is required. The breaking point achieved with the highest tolerable dose of a drug is often taken to be an index of that drug's reinforcing properties. Drugs with the highest breaking point are considered to be the most reinforcing and hence to have the highest abuse potential. Of drugs studied with such a procedure, cocaine appears to have the highest breaking point. For example, in some experiments, animals have been willing to press a lever up to 12,000 times for a single dose of cocaine.

Choice experiments provide a second means for comparing the reinforcing properties of two different drugs. Animals in such designs are typically given a choice between two responses, each of which leads to infusions of a different drug. A preference for one response is taken to indicate a preference for the drug associated with that response. A finding of particular interest from such studies has been that cocaine appears to be preferred not only to a number of other drugs but also to nondrug rewards such as food and social contact (Johanson, 1984)—but it is important to vary the other reinforcers as well. Animals will choose less cocaine when the amount of food provided is greater or tastier.

Far fewer systematic data on the reinforcing properties of drugs have been collected with human subjects. A number of experiments have shown that human subjects will work for tokens exchangeable for OPIOIDS, alcohol, pentobarbital, DIAZEPAM (Valium), and *d*-amphetamine. In addition, drug-abusing individuals will reliably produce arbitrary responses in a laboratory for immediate access to their drugs of choice. For example, heroin addicts will repeatedly push a button to receive heroin injections and cocaine users will choose to perform responses leading to cocaine injections over responses that yield injections of saline (Henningfield, Lukas, & Bigelow, 1986).

In sum, a body of both animal and human data now exists that documents the way drugs of abuse can act as potent reinforcing events. The pattern of drug use exhibited by an individual user, however, appears to depend as much on the schedule of drug availability as on the particular properties of the chosen drug. Therefore, predicting patterns of drug taking by humans will require a better understanding of the parameters of drug availability that exist in the real world.

CLASSICAL CONDITIONING OF DRUG-RELATED CUES

Conditioned Withdrawal Model. A number of investigators have advanced the idea that stimuli that reliably signal drug use elicit CRs that motivate further drug use. For example, Wikler (1980) noted that drug-free heroin addicts participating in discussions of drug use appeared to go through episodes of withdrawal as a result. Withdrawal refers to the unpleasant symptoms experienced by drug abusers following the abrupt cessation of drug use. Since the individuals discussed by Wikler had not used heroin for a long time, their withdrawal symptoms could not have been the result of recent termination of the drug. Wikler proposed instead

that events that reliably signal the onset of naturally occurring drug withdrawal become CSs capable of evoking withdrawal symptoms on their own. On future occasions, the mere presence of drug-related stimuli evoke conditioned withdrawal states that compel the person to counter these unpleasant feelings with drug use.

A second model that also invokes conditioning was put forth by Siegel (1979), who proposed that stimuli paired with drug use come to evoke conditioned compensatory responses, which oppose the direct effects of the drug. As these drug-opposite responses grow in size, over repeated conditioning experiences, they increasingly oppose the effects of the drug. Therefore, addicts should find that, over time, higher doses of the drug should be necessary to achieve a given effect. This pattern is indeed observed, and it is known as drug tolerance. According to Siegel's model, drug-related cues encountered in the absence of drug taking produce drug-opposite responses, which are not canceled by the direct effects of the drug. These drug-opposite responses are then thought to represent what the user experiences as withdrawal symptoms.

According to Siegel's model, conditioning can motivate drug use in two ways. First, the withdrawal symptoms generated by drug-related stimuli following a period of abstinence can lead to drug use aimed at relieving these unpleasant effects. Second, tolerance to the effects of a drug may motivate a user to increase his or her level of use in an attempt to maintain a fixed level of drug effect.

Siegel's model has not gone unchallenged. The primary problem appears to be that signals for drug use do not always produce drug-opposite responses. Instead, such signals sometimes produce responses that resemble the direct effects of the drug. The conditions determining whether CRs produced by drug-related stimuli are drug-like or drug-opposite have not been fully worked out. Yet drug-like responses (e.g., drug-induced euphoria) to environmental stimuli may act to motivate drug use as well. Some researchers have asserted that it is the memory of drug-induced euphoria that is the major factor contributing to continual drug use and relapse.

Conditioned Incentive Model. Stewart, deWit, and Eikelboom (1984) have proposed that conditioned drug stimuli provide the impetus for further drug use by producing mild drug-like effects, which whet the appetite of the user (the priming effect). Thus drug-related events cause drug use by prompting the user to anticipate the pleasurable consequences. Like the models of Siegel and Wikler, this theory proposes that events signaling drug use become conditioned stimuli that encourage the drug user to initiate drug-seeking behaviors. The models differ only in their characterization of the CR elicited by the drug-related events.

Some evidence for this model lies in the observation that many animals show drug-like responses to stimuli paired with drug use, particularly when stimulant drugs such as cocaine or *d*-amphetamine are used. Furthermore, many researchers have found that animals that have stopped responding for a drug reinforcer may resume responding following a small unearned dose of the drug (a priming dose). Environmental signals for drug use may act in the same way as these priming doses. Some research suggests that the stimuli associated with rewards can elicit release of the neurotransmitter dopamine in the reward center of the brain, an event that is produced by the effects of most rewarding drugs.

Human Data. Since the 1970s, investigators have collected data from a number of sources to document that stimuli associated with drug use in humans acquire conditioned properties. Evidence for this statement has come from three primary sources:

- self-reports by addicts about the conditions under which they experience craving and withdrawal
- attempts to establish drug conditioning in the laboratory
- assessments of responding to cues thought to have become drug CSs in the natural environment

Self-reports of Conditioned Effects. Many drug-abuse patients report drug craving and withdrawal when faced with drug-related stimuli in their home environment. Wikler (1980) reported that drug-free heroin addicts who returned to their home (addiction) environment following a period of treatment experienced symptoms of heroin withdrawal. O'Brien (1976) took a more systematic approach, interviewing heroin addicts and constructing a hierarchy of real-world events that result in withdrawal feelings and craving for drug use. Such reports encouraged the idea that events that signal drug self-administration in the home environment

come to evoke conditioned responses, which motivate further drug use. Consequently, investigations have attempted to study this phenomenon under controlled laboratory conditions.

Laboratory Conditioning Studies. O'Brien and his colleagues (1986) found that neutral stimuli paired either with opiate administration or with opiate withdrawal appeared to elicit conditioned withdrawal reactions. A number of later studies using alcohol as the unconditioned stimulus have also tended to find drug-opposite responses elicited by the experimental CS, although drug-like effects have been observed as well.

Studies of conditioning in the laboratory permit the conclusion that such conditioning can occur as a consequence of drug-taking behavior by addicts. However, the contingency between potential CSs and drug effects in the natural environment is undoubtedly less precise than that programmed in the laboratory. Therefore, affirming the role of drug-related stimuli in drug use requires a direct assessment of the effects of such events.

Cue-Assessment Studies. To determine whether events associated with drug use in the natural environment acquire conditioned properties, many studies have recreated typical drug-related stimuli in the laboratory. In such studies, subjects are exposed to audiotapes, videotapes, slides, and paraphernalia with drug-related content, while physiological and self-report responses are obtained. Responses to such drug-related stimuli are then usually compared with the responses subjects make when they are exposed to comparable stimuli lacking a drug-specific content. So far, the majority of cue-assessment studies have been carried out with either opiate-abusing subjects or alcoholics, although there have also been some studies of cocaine abusers.

For opiate users, exposure to visual stimuli and paraphernalia typically associated with opioid use have been found to produce subjective reports of craving for opiates and withdrawal, as well as other physiological changes associated with withdrawal, including drops in skin temperature and skin resistance and increases in heart rate. Other studies have shown that opiate users given a medication that blocks the effects of the opiates (an opiate ANTAGONIST) initially report pleasurable sensations after only injection—even of placebo (saline solution). After several repetitions of the placebo, the injections begin to become aversive, including elements of withdrawal. Opiate-related stimuli, then, appear to evoke both subjective and physiological responses related to drug use; it appears likely that such responses have a basis in prior conditioning.

Studies with alcoholics have yielded similar results. In a typical experiment, the reactions of alcoholics to the sight and smell of an alcoholic beverage are compared with responses evoked by a nonalcohol (placebo) drink. The results show greater craving/urges to drink induced by the alcohol cue than by the control stimulus. Such data suggest that alcohol-related cues acquire the ability to evoke conditioned responses.

Classical/Operant Conditioning Interaction. As mentioned earlier, much of the work on drug conditioning contains the implicit notion that classical and operant learning effects combine to motivate drug use (or craving, even if no drug use actually occurs). The most common idea is that drug CSs evoke craving and withdrawal states, which motivate the performance of drug-seeking behaviors. These behaviors are reinforced, in turn, by the effects of the drug. Although much evidence suggests that psychoactive drugs of abuse have powerful reinforcing properties—and that signals of those drugs elicit conditioned responses—the question remains as to whether these classically conditioned responses actually motivate drug-seeking behaviors.

One technique used in a study to examine this issue involved asking opiate abusers to describe the conditions under which they had relapsed to drug use. After some probing, many patients were able to describe specific events or stimuli that triggered craving and withdrawal leading to use. Such reports, however, suffer from the difficulty that retrospective self-reports from individuals reluctant to analyze their own behavior may be inaccurate.

Because of the ethical limitations imposed on providing drugs to drug abusers, little laboratory work has been done to examine whether drug-related cues increase drug self-administration. Some data have been collected by Ludwig, Wikler, and Stark (1974), however, who found that alcoholics in a barlike environment worked harder at an alcohol-rewarded task than did subjects in a laboratory setting. This result supports the idea that alcohol-related stimuli impact directly on the motivation to drink alcohol. Yet, no nonalcoholic subjects were studied, preventing the conclusion that alcoholics are uniquely susceptible to alcohol-

related events. Because the assumption that responses to drug-related stimuli motivate actual drug use is central to learning models, more studies using drug taking as a dependent measure are clearly needed.

Treatment Implications of Learning and Conditioning Theories. If classical and operant conditioning motivate drug use, then substance-abuse treatments should aim at reducing the impact of these learning effects. The most commonly discussed interventions include aversion training, extinction, and behavioral alternatives.

AVERSION THERAPY or training involves teaching subjects that stimuli and responses that once terminated in drug effects will lead to unpleasant outcomes. The most common technique has been to pair self-administrations of a drug with electric shock or with chemically induced sickness (e.g., Antabuse). Although there are some anecdotal reports of successful treatment using such therapy, systematic data are not abundant. In addition, such aversion training suffers from the disadvantage that patients are unlikely to continue to administer punishments to themselves once outside treatment. Since the treatment setting is clearly different from the home environment, subjects may simply learn that drug-taking behavior is reinforced at home but punished in the clinic.

Extinction training consists of exposing subjects repeatedly to drug-related stimuli and responses, to break the association between these events and the effects of drug use. Operant extinction procedures require subjects to perform repeatedly drug-use behaviors in the absence of a drug reinforcer. This can be accomplished by having subjects administer their drug of abuse in the usual way—while they are maintained on medication blocking the drug's effects. In this way, drug responses go unreinforced, because the drug effects are missing. Extinction of classically conditioned stimuli typically requires subjects to view repeatedly drug-related scenes and handle paraphernalia without using the abused substance. Such training has the advantage of not requiring subjects to accept punishment. Nevertheless, subjects might show extinction in the context of the clinic but continue to experience conditioned effects that lead to drug use in the unaffected home environment.

Behavioral alternatives training represents a third approach to reducing the impact of conditioning on drug abuse. This technique involves teaching subjects to avoid drug-related situations or to make alternative responses in the presence of drug-related stimuli. These new responses are designed to compete with the drug-seeking behaviors usually elicited by drug-related cues. Rather than try to eliminate craving produced by drug cues, this treatment attempts to give the patient ways to avoid cues plus alternatives to drug use. Behavioral alternatives to drug use range from simple time-out periods, to forming images inconsistent with drug use, to acting in ways that reduce the chances of use or cue exposure (e.g., going out to eat). These approaches are now commonly designated as COGNITIVE THERAPIES or RELAPSE-PREVENTION approaches. The advantage of these procedures over aversion therapy and extinction lies in the greater potential for patients to use their training in the clinic to deal with high-risk situations in the real world.

Whether these particular conditioning interventions provide lasting help to substance abusers remains to be seen—but data exist to suggest that in alcoholics, opiate addicts, and cocaine users, these cognitive therapies and relapse-prevention techniques do have value. They reduce the probability of relapse.

(SEE ALSO: *Addiction: Concepts and Definitions; Conditioned Tolerance; Naltrexone in Treatment of Drug Dependence; Wikler's Pharmacologic Theory of Drug Addiction*)

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STEVEN J. ROBBINS

Psychological (Psychoanalytic) Perspective The psychological study and understanding of substance abusers has tended to be difficult, controversial, and complicated. Part of this derives from the nature of addictive illness; the acute (short-term) and the chronic (long-term) use of drugs and ALCOHOL cause individuals to seem pleasure oriented, self-centered, and/or destructive to self and others, thus making them difficult to approach, understand, or treat. In other respects, the controversy or lack of understanding derives from competing ideas or schools of thought that debate (if not hotly contend) whether substance abuse is a disease or a symptom, whether biological and genetic factors are more important than environmental or psychological ones, and/or whether substance abuse causes or is the result of human psychological suffering. Furthermore, in recent years psychological factors were minimized, because we entered the era of biological psychiatry/psychology, in which empirical interest in brain structure and function (down to the microscopic and molecular level) has predominated over interest in the person, the person's mind, and subjective aspects of human psychological life that govern our emotions and behavior. Although one cannot ignore that substances of abuse are PSYCHOACTIVE—powerful chemicals that act on the brain—there is a tendency to lose sight of the total person whose ways of thinking, feeling, and behaving (including subjective feelings about self and others) are equally and profoundly affected both by that chemistry and by the subjective effects produced by those psychoactive substances.

Clearly, biological, genetic (i.e., hereditary), and sociological factors are important in the development of drug abuse and dependence. Such factors are best studied by empirical methods, and modern technology—including the computer—has yielded new and valuable data since the late 1960s to explain aspects of addictive behavior. It is also noteworthy that during this period (a time when substance abuse has been most prevalent, studied, and treated), clinical work with substance abusers has yielded data and findings of equal importance and validity, and this work has focused on some of the important subjective psychological factors that also explain aspects of addictive behavior—some which empirical methods alone do not adequately fathom or explain.

I will present here a psychological understanding of drug abuse and dependence based on the perspective gained from clinical work with alcoholic and drug-dependent individuals. In psychology and clinical psychiatry, it is referred to as the *case method* of study of human psychological prob-

lems. Guided by psychodynamic principles (the assumptions of which will be explained), this article will review what three decades of clinical work and case study with substance abusers has yielded on some of the main psychological influences that make likely or compelling the dependence on, and continued use and relapse to, drugs and alcohol.

ASSUMPTIONS

A psychodynamic perspective of human psychological life problems rests on the principle that we are all more or less susceptible to various forms of human psychological vulnerabilities—at the same time, we are also more or less endowed with human psychological strengths or capacities to protect against these vulnerabilities. Without ignoring hereditary factors, especially those that affect temperament, a psychological, and in this case psychodynamic perspective attempts to understand psychological forces at work (for example, drives and feelings) that operate within the individual at the same time that there is a corresponding interest in the psychological structures and functions that observably (and just as often, less obviously) operate to regulate or control our drives, feelings, and behavior.

A psychodynamic approach to human psychology greatly depends on a developmental perspective or an appreciation of the psychological forces, structures, and functions as they develop and change over one's lifetime. Psychodynamic clinicians are especially interested in the way individuals are influenced in the earliest phases of development by parents (and other caregivers), and then in the development of relationships with other children and peers, and later in the life cycle in relationships with adults and small and large groups—all of which shape our life views and experiences, as well as our attitudes, values, and characteristic ways of reacting and behaving.

Based on these assumptions, clinicians have the opportunity, most usually in the context of treating patients, to study and understand how the degree of developmental impairments (or strengths) has predisposed toward (or protected against) psychological and psychiatric dysfunction, including addictive vulnerability. In my experience, and that of my associates, we believe that modern psychodynamic-clinical approaches are as relevant and useful for studying and treating substance-dependent individuals as they are for the many other patients who benefit from this perspective.

The psychological study and understanding of addictive illness necessarily requires the condition of abstinence (being free of drug/alcohol use). Again, there is considerable debate about the duration of abstinence required before meaningful or valid psychological inferences can be made about individuals with addictive disorders. In my experience, however, the confounding effects of acute and chronic drug/alcohol use are variable, and it is often surprising that within days or weeks—but certainly within several months of abstinence—how much can be learned about a person's makeup and psychology that predisposed him or her to use and become dependent on substances. This point about the requirement for a period of abstinence from drugs and alcohol is important to emphasize, otherwise it can be and is rightfully argued that what appear to be the psychological causes of dependence on psychoactive substances are actually the result of such a dependence. Fortunately, in recent years, the combination of modern detoxification approaches, psychoeducational/rehabilitation/Relapse Prevention programs, Twelve-Step groups, and individual and group psychotherapeutic approaches, have been increasingly successful in establishing and maintaining abstinence. This, in turn, has made psychological treatments and understanding increasingly possible.

PSYCHOLOGICAL SUFFERING AND SELF-CONTROL

A clinical-psychodynamic perspective suggests that human psychological suffering and problems with self-control are at the heart of addictive disorders. In fact, it is probably safe to say that to understand the psychology of addictive behavior is to understand a great deal about human psychological problems of suffering and control in general. The suffering that influences addictive behavior occurs at many levels, but it principally evolves out of susceptibilities involving people's self-esteem, relationships, emotions, and capacities to take care of themselves. Individuals who find various or particular drugs appealing (including alcohol) or who become dependent on them, discover that, short-term, the drug action or effect relieves or controls their distress—that is, such drugs are used to self-medicate distress. Although

problems with self-esteem and relationships are important parts in the equation of addictive behavior, it is mainly the problems with how substance-dependent individuals experience, tolerate, and express their feelings and their problems with self-care that makes addictive behavior so malignantly likely and compelling.

Problems with emotions and self-care painfully and repetitiously become involved with attempts to control suffering and behavior. This process includes such self-defeating coping patterns as action, activity, psychological defensiveness (e.g., denial, boastful or arrogant postures, attitudes of invulnerability and toughness), and, ultimately, the use of drugs and alcohol. What originally was a "solution" for suffering and self-regulation—where substances were used for relief or control—turns into a problem where there is a progressive loss of control of one's self, the drugs or alcohol employed to combat one's difficulties, and possibly life itself.

THE SELF-MEDICATION HYPOTHESIS

The self-medication hypothesis specifically refers to some individuals who, by dint of temperament or developmental factors, experience and find that certain painful feelings (or affects) are intense and unbearable and that the specific action or effect of one of the various classes of abused drugs (e.g., analgesics, depressants, or stimulants) relieves their psychological pain and suffering. The self-medication hypothesis also implies that the particular drug or class of drugs preferred is not random. Rather, it is determined by how that class of drugs with its specific actions interact with emotional states or particular painful feelings unique to the individuals who use or select their "drug-of-choice."

This is only one aspect of addictive suffering—namely that emotions are experienced in the extreme and that addictive-prone individuals feel too much pain, so resort to particular drugs to *relieve* their suffering. Another aspect of addictive suffering, to be covered subsequently, is that emotions are just as often absent, nameless, and confusing and that such individuals experience pain of a different type; they consciously feel too little of their distress and do not know when or why they are bothered (e.g., feeling empty, void, or cut off from emotions), and drugs or alcohol in these instances are used to change or *control* their emotions or suffering. In the first instance the operative motive is the relief of suffering; in the second, it is the control of suffering.

The self-medication hypothesis rests on the observation that patients, if asked, will indicate that they prefer or discover that one class of drugs has more appeal than another. Still, the drugs preferred by an individual are not the ones that are always used. Drugs that are actually used are just as often the result of other factors, such as cost and availability.

The three main classes of drugs that have been studied are the OPIOID analgesics (pain relievers), depressants or SEDATIVE-HYPNOTICS (soothing, relaxing, or sleep-inducing drugs), and STIMULANTS (activating or energizing drugs). The main appeal of opioids (e.g., HEROIN, MORPHINE, oxycodone) is that they are powerful subduing or calming agents. Besides calming or subduing physical pain for which they were originally intended, opioids are also very effective in reducing or alleviating distressing or disruptive emotions. Beyond its calming influence on physical and emotional pain in general, however, I have found that the main and specific action of opioids, namely as an anti-rage or anti-aggression drug, makes them especially appealing and compelling for those who struggle within, and with others, with feelings of intense anger, AGGRESSION, and hostility. Such a state of affairs is not uncommon for people who, in their early life development or in later life experiences, have suffered major trauma, neglect, or abuse. Such individuals, when they first use opioids, discover the extraordinary calming and soothing effects of these drugs on their intense anger and rage—and thus they become powerfully drawn or attached to them.

Whereas opioid-dependent people have much difficulty controlling their feelings, especially anger and rage, those who prefer or who are dependent on depressants generally have the opposite problem—namely they are too controlled or too "tightly wrapped" around their feelings. As is the case with other substance abusers, developmental life experiences, in this case often involving distrust and traumatic disappointment, have had a special influence on their experience of emotions. In the case of those who prefer depressants, they are the ones who have special difficulties experiencing emotions involving loving or caring feelings, interpersonal depen-

dency, and closeness; in psychological terms, they are defensive and repressed around these emotions and have difficulty in experiencing or expressing them. Depressants (e.g., alcohol, Seconal, Xanax) have appeal for these people, because such drugs help them to relax their defenses and release them from their repressions. Mainly, such drugs briefly (the short- or quick-acting depressants) produce a sense of safety and an inner sense of warmth, affection, or closeness that otherwise these people cannot experience or allow.

Finally, stimulants (i.e., AMPHETAMINES and COCAINE are the most popular and widely used) have appeal for those who suffer with overt and/or subtle states of depression, mania, and hyperactivity—in which problems with activation, activity, and energy are common. For example, ambitious driven types—for whom performance, prowess, and achievement are essential—find such drugs especially appealing on two counts: (1) stimulants are uplifting when they become depressed if their goals and ambitions, often unrealistic, fail them; (2) stimulants are facilitating and make action and activity easier when such people are on the upswing, making it easier for them to be the way they like to be when they are performing at their best. Stimulants cast a wide net of appeal because, in addition, they counter feelings of low energy, low activity, and low self-esteem in those suffering with overt or less overt (unrecognized or atypical) depression. Finally, those individuals suffering with attention deficit-hyperactivity disorder (ADHD), often subclinical or not recognized, are also drawn to and become dependent on stimulants, because of the paradoxically opposite calming effect that stimulants have for people with this disorder—much like hyperactive children who respond well to the prescribed stimulant Ritalin.

SELF-REGULATION VULNERABILITIES

To explain why people became addicted, early psychodynamic theory placed great emphasis on subconscious and unconscious factors, pleasure and aggressive instincts or drives, and the symbolic meaning of drugs. To some extent, the stereotype of substance abusers as pleasure-seeking destructive characters (to self and others), in part, still persists and derives from these early formulations. Albeit useful and innovative at the time, much of this early perspective is now outdated, counterempathic, and does disservice to understanding the motives of addicted and alcoholic individuals.

In contrast, the self-medication hypothesis has evolved from contemporary psychodynamic theory, which has placed the centrality of feelings (or affects) ahead of drives or instincts and has emphasized the importance of self-regulation, involving self-development or self-esteem (i.e., self-psychology), relationship with others (i.e., object-relations theory), and self-care (i.e., ego or structural psychology/theory). These contemporary psychodynamic findings have evolved since the 1950s, based on the works of investigators such as Weider and Kaplan, Milkman and Frosch, Wurmser, Krystal, Woody and associates, Blatt and associates, Wilson, Dodes, and Khantzian.

Although the self-medication hypothesis has gained wide acceptance as an explanation for drug/alcohol dependency, it is not without its critics and it fails to deal with at least two fundamental problems or observations that it does not explain or address. First, many individuals suffer with the painful feelings and emotions that substance abusers experience, but they do not become addicted or alcoholic. Secondly, the self-medication hypothesis fails to take into account that addicted and alcoholic individuals suffer as much if not more as a result of their drug/alcohol use, and this might appear to contradict the hypothesis that substances are used to relieve suffering.

Many of these criticisms, inconsistencies, and apparent contradictions are better understood or resolved when addictive problems are considered more broadly—in terms of self-regulation vulnerabilities or as a self-regulation disorder. For humans, life is the constant challenge of self-regulation, as opposed to the release, relief, or control of instincts and drives as early theory suggested. What is in need of regulation involves feelings, the sense of self (or self-esteem), relationships with others, and behavior. Those prone to addictive problems are predisposed to be so, because they suffer with a range of self-regulation vulnerabilities. Their sense of self, including self-regard, is often shaky or defective from the outset of their lives. A basic sense of well-being and a capacity for self-comfort and self-soothing is very often lacking or underdeveloped from the earliest phases of development. Subsequent development of self-esteem and self-love, if

it develops at all, remains shaky and inconsistent, given the compromised sense of self from which self-regard evolves. Needless to say, a poor sense of self or low self-esteem (which usually originates in a compromised or deficient self-other parenting relationship), ultimately affects subsequent self-other relationships and profoundly affects one's capacity to trust or to be dependent upon or to become involved with others.

It should not be surprising, then, that for some the energizing and activating properties of stimulants help self-doubting reticent individuals to overcome their depressive slumps and withdrawal; or that the soothing, relaxing effects of depressants help individuals who are restricted and cut off from others to break through their inhibitions and briefly experience the warmth and comfort of human contact that they otherwise do not allow or trust; or yet still, that those whose lives are racked by anger and related agitation would find a drug like heroin (an opioid analgesic) to be a powerful containing calming antidote to their intense and threatening emotions, which disrupt them from within and threaten most of their relationships with others. These examples, and those previously covered in relation to self-medication motives that govern drug use and dependency, help demonstrate the how and why of specific drug effects—which often become so compelling that they may consume the lives of some users.

In my experience, the regulation of *feelings* (or affects) and *self-care* become the two most compelling self-regulatory problems; they combine to make dependence on substances more likely than any other self-regulation factors. Focus on these two factors explains clearly why most people who suffer subjective painful emotions do not necessarily become addicted as well as why so many substance abusers persist in using debilitating substances despite the great suffering that ensues from their abuse.

It should be noted that in this article I have stressed psychological factors and have not pursued how the regular use of addictive drugs causes TOLERANCE AND PHYSICAL DEPENDENCE; the drugs, then, are used to remain "normal." It is not insignificant however, that the emotional pain involved in physical WITHDRAWAL just as often is an exaggerated form of the pain that the drug-of-choice originally relieved. This aspect of drug use and relapse are covered elsewhere in this encyclopedia.

As I have indicated, substance abusers suffer in the extreme with their emotions—they feel too much or they feel too little. When there is too much, we have described how drugs can relieve the intense unbearable feelings that addicts and others experience. Where there is too little and people are (or seem to be) devoid of, cut off from, or confused by their feelings (e.g., alexithymia, disaffected, or affect deficits), addicts prefer to counter the helplessness and loss of control caused by their lack of feelings. Instead, they choose to use drugs to change and control their feelings, even if it causes them more distress. They exchange feelings that are vague, confusing, and out-of-control, for drug-induced feelings that they recognize, understand, and control, even if such are painful and uncomfortable. Therefore, the factors of *relief* and *control* dominate people's motives for depending on drugs—even if these people have to endure the pain that their dependence on drugs also entails.

Finally, *deficits in self-care* (again deriving from early-life developmental problems) make it likely that certain individuals will become involved with hazardous activities and relationships that lead to drug experimentation, use, and dependence. Self-care deficits refer to a major self-regulation problem, wherein individuals feel and think differently around potential or actually dangerous situations and activities, including those that involve drug/alcohol experimentation and use. Where most of us would be apprehensive or frightened or would anticipate some guilt and shame, addictive and alcoholic-prone individuals show little or no such worry. Studying these patients' pre- and postaddictive behavior patterns very often reveals similar unfeeling, unthinking, fearless behavior in conducting other aspects of their lives—for example, preventable accidents, health-care problems, and financial difficulties seem evident and common. Being out of touch with, or not feeling, their feelings (that is, their "affect deficits" or "dis-affected state") contributes to their self-care problems and thus makes it more likely that they would engage in the dangerous pursuit of drug/alcohol abuse, where others with better self-care functions would not (even in those instances where the unbearable psychological suffering and states of distress are like those experienced by addicts). In this respect, painful or unbearable feelings, alone, are not sufficient to cause substance abuse or dependence. Rather, it is when individuals lack adequate self-care capaci-

ties *and* experience intense suffering that conditions exist for addictive behavior to develop or be likely.

(SEE ALSO: *Addiction: Concepts and Definitions; Causes of Substance Abuse: Learning; Complications: Mental Disorders; Conduct Disorder and Drug Use; Coping and Drug Use; Disease Concept of Alcohol and Drug Abuse; Vulnerability: Psychoanalytic*)

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E. J. KHANTZIAN

CENTER ON ADDICTION AND SUBSTANCE ABUSE (CASA) The Center on Addiction and Substance Abuse at Columbia University (633 Third Avenue, New York, NY 10017) was established in 1992 as a not-for-profit entity affiliated with the university. It was founded by Joseph A. Califano, Jr., a former secretary of health, education, and welfare in the Carter administration, and Herbert D. Kleber, M.D. CASA has been funded by major grants from the Robert Wood Johnson Foundation, as well as other foundations, private companies, and government agencies. Califano, who has had a long-term interest in substance-abuse problems, was a vigorous advocate of smoking cessation programs when he was a member of the Carter cabinet. In organizing CASA, he assembled a board of directors that includes many prominent people in politics, industry, academia, ADVERTISING, and the media. Kleber, a well-known drug abuse researcher and former deputy director of the Office of National Drug Control Policy under William Bennett and President George Bush, is executive vice president and medical director. Califano is president and chairman of the board.

The work of the center initially emphasized analysis of available data on the social and ECONOMIC COSTS of substance abuse (ALCOHOL, TOBACCO, and illicit drugs). The center then moved on to creating national demonstration projects, major policy papers on key issues in the substance abuse field, and research projects on treatment.

CASA has demonstration programs at thirty-eight sites in twenty-five cities and sixteen states. These demonstration programs include CASA START, a program to help thirteen- to eighteen-year-old children who are using alcohol, tobacco, or illicit drugs or who are at high risk of doing so. It involves collaboration between schools, police departments and community organizations, and is aimed at preventing substance abuse, improving school performance, and reducing delinquency among these children. CASA WORKS FOR FAMILIES is the first comprehensive national demonstration designed to help drug and alcohol addicted mothers on welfare achieve self-sufficiency. It is a sixteen million dollar, three-year demonstration that combines in a single course of treatment and training: drug and alcohol treatment, literacy and job training, parenting and social skills, violence prevention, health care, family services, and a gradual move to work. The program is being tested

in eleven sites in nine states and will serve more than one thousand women and their children. More sites are planned.

Reports put out by CASA include: *Substance Abuse and the American Adolescent*; *Substance Abuse and the Mature Woman*; *Substance Abuse and America's Prison Population*; *Substance Abuse on the College Campus*; *Substance Abuse and Sex*; *Substance Abuse and Learning Disabilities*; and *Substance Abuse in Rural America*. Other projects include CASA's National Commission on Sports and Substance Abuse, which will produce a comprehensive analysis of substance abuse and sports in America; the National Evaluation of Substance Abuse Treatment (NESAT), which follows two thousand individuals in close to two hundred treatment programs from intake up to one year or more afterwards, to evaluate the effectiveness of drug and alcohol treatment programs; CATS, the Cocaine Alternative Treatment Study, which looks at the effectiveness of acupuncture as a treatment for cocaine in over 500 patients at six university sites across the U.S.; and an analysis of drug courts and their effectiveness.

Through articles both in the popular press and scientific journals, press conferences, and testimony before congressional committees, CASA conducts a continuing campaign to raise public awareness about the pervasiveness of and the SOCIAL COSTS OF SUBSTANCE ABUSE. The priorities for the organization are to explain to the American people the social and economic costs of substance abuse and its impact on their lives, to identify what can be done, which prevention and treatment programs work and for whom, and to encourage individual institutions to take responsibility to prevent and combat substance abuse.

(SEE ALSO: *Economic Costs of Substance Abuse; Social Costs of Substance Abuse*)

JEROME H. JAFFE
REVISED BY HERB KLEBER

CENTRAL NERVOUS SYSTEM *See* Brain Structures and Drugs; Limbic System; Neurotransmission

CENTRE FOR ADDICTION AND MENTAL HEALTH The Centre for Addiction and Mental Health is a clinical, research, and teaching facility that is associated with the University of Toronto. The Centre is one of only four mental health and addictions facilities to receive the Centre of Excellence designation by the World Health Organization (WHO). It was created in 1998 through the merger of the Queen Street Mental Health Centre, the Donwood Institute, the Clarke Institute of Psychiatry, and the Addiction Research Foundation. The Centre's central research and clinical facilities are located in Toronto and 12 additional offices are found in communities throughout the province of Ontario.

The Centre's underlying goals are to advance the understanding of mental health and addiction and to utilize that knowledge in their treatment programs. It aims to prevent problems, find more effective treatments for mental illness and addictions, and improve the quality of life for persons who are afflicted with mental illness or struggling with addictions. It is a patient-centered institution that provides services that are sensitive to gender, age, race, culture, religion, and sexual orientation. The Centre is a unique entity that is recognized for its ability to:

- Integrate biological, clinical, and social research;
- Translate research results into treatments and tools for practical use;
- Blend treatments for alcohol and drug addiction and mental health disorders;
- Conduct leading-edge research; and
- Provide continuity of care in one location.

The Centre is involved with many programs to strengthen the capacity and quality of mental health and addiction services in Ontario. One program, the Assertive Community Treatment Team, uses a multi-disciplinary team to provide intensive treatment of chronic mental illness in diverse communities. Another program educates family physicians, who have patients with addiction and mental health disorders, through a series of continuing education seminars. The Centre aids the smaller, more distant Ontario communities by sending "fly-in" consultation teams or through video conferencing with patients or health care providers. In addition, the Ontario Tobacco Research Unit is housed at the Centre and provides funds for sur-

veillance, evaluation, and research into nicotine addiction.

The Clarke Institute of Psychiatry is the primary research site of the Centre. Funding is provided by the Ontario Ministry of Health, research grants, and donations. Psychiatric research programs include biochemistry, endocrinology, epidemiology, genetics, molecular biology, neurochemistry, neuroimaging, positron emission tomography (PET), and transgenics. Major areas of research include:

- Investigating the biological aspects of mental illness and addiction;
- Studying nicotine addiction;
- Investigating treatments for depression;
- Investigating treatments for children with behavioral problems;
- Improving the treatment of schizophrenia;
- Studying pathological gambling; and
- Studying the issues of mental health and the homeless.

The Centre publishes the *Journal of Addiction and Mental Health*, a magazine that provides information and news items relevant to mental health professionals and other interested parties. The readership includes professionals, patients, consumers, and family members. Initially aimed at a Canadian readership, the Centre hopes to expand the *Journal of Addiction and Mental Health* to attract an international readership. Also published by the Centre is the *Balanced Scorecard* which provides an overview of the performance of the Centre.

Key personnel at the Centre include: Dr. Paul Garfinkel, president and chief executive officer; Jean Simpson, executive vice president and chief operating officer; Dr. Georgiana Beal, chief of nursing practice and professional services; Dr. David Goldbloom, physician-in-chief; Dr. Patrick Smith, Vice president, addiction programs; Jean Trimnell, vice president; mental health programs; Dr. Franco Vaccarino, vice president, research; Carolyn Nutter, vice president, community health and education; and Mary Deacon, president and executive director, CAMH foundation.

The Centre for Addiction and Mental Health, is located at 33 Russell Street, Toronto, Ontario, Canada M5S 2S1, (416) 535-8501, http://www.camh.net.

BELINDA ROWLAND

CHAIN OF CUSTODY *See* Drug Testing

CHEMICAL DEPENDENCE *See* Addiction: Concepts and Definitions

CHEWING TOBACCO *See* Tobacco: History of; Treatment: Tobacco, an Overview

CHILD ABUSE AND DRUGS In the United States, on average, a child is abused every 13 seconds. Because of social awareness, reported child abuse has increased dramatically since the 1980s. Some states have experienced a 20 percent increase in reported child abuse between 1990 and 1991. The American Public Health Association (APHA) estimates that 1.7 million children are abused or neglected annually in the United States. This means that by 1992 a total of about 63.5 million (2.8%) of children under 18 years of age were abused.

Reported cases in the 1990 National Child Abuse and Neglect Data System totaled about half the APHA estimate, or only 893,856. This total comprised 227,057 victims of physical abuse; 403,430 victims of neglect; 138,357 victims of sexual abuse; 59,974 victims of emotional maltreatment; and 68,207 other. These figures represent only the reported cases—the proverbial tip of the iceberg. Research suggests that as many as 10 percent of children may be sexually abused and even more children physically abused or neglected. In addition, each year a higher percentage of U.S. children are being raised in poverty, often by overstressed and drug-abusing parents.

INCIDENCE AND PREVALENCE OF DRUG ABUSE

Although the casual use of drugs is decreasing in the United States, reported drug abuse is increasing in women of child-bearing age. While it is believed that more children are being raised by alcohol-, tobacco-, or other drug (ATOD)-abusing parents, the scope of the problem is undetermined. Longitudinal studies of children of ATOD-abusing parents are currently underway by the U.S. Centers for Disease Control (CDC), the NATIONAL INSTITUTE ON

DRUG ABUSE (NIDA), and the NATIONAL INSTITUTE ON ALCOHOLISM AND ALCOHOL ABUSE (NIAAA). The Children of Alcoholics Foundation estimates that in the U.S. population about one in eight were raised in homes with one alcoholic parent. Studies suggest that as many as 11 percent of newborns are drug-exposed in utero. About six million women of childbearing age are marijuana users and 10,000 children per year are born to women using opiates. Polydrug use and frequent use of alcohol and other drugs by parents increases the difficulty of researching any causal relationships between a specific drug and child abuse.

The National Committee for the Prevention of Child Abuse (NCPCA) estimates that 10 million U.S. children are raised by ATOD-abusing parents or caretakers and at least 675,000 children every year are seriously abused by ATOD caretakers. ATOD-abusing women have higher fertility rates and more multiple births than non-ATOD-abusing women. Reasons for these repeated pregnancies in drug-abusing women may include lack of sex education and birth control, irregular menstruation, carelessness when using drugs, peer pressure and cultural norms, the desire to replace lost children, the need for increased welfare payments, the enjoyment of being pregnant (decreased depression), and having an infant to love them. These interesting findings should be studied further to determine their validity and related psychological, sociological, and biological causal mechanisms.

RELATIONSHIP OF CHILD MALTREATMENT AND ATOD ABUSE

Do addicted parents abuse their children more? Do both addicted and nonaddicted parents abuse their children more when using alcohol or drugs? Unfortunately, clear empirical research is lacking on the relationship of child abuse and alcohol abuse or child abuse and drug abuse. The validity of existing research is threatened by problems, such as unclear definitions of ATOD abuse, lack of control groups or longitudinal causal studies, and inappropriate statistical and research design techniques for separating causation and coincidence (Bays, 1990). Nevertheless, a relationship does exist between child abuse and ATOD abuse—and definitely between ATOD abuse and child neglect. Similar risk factors for child abuse exist for both child-abusing parents and substance-abusing parents, such as poor parenting skills, family disorganization, involvement in criminal activity, and a disproportionately high incidence of mental and physical illness. Several types of child abuse and neglect involving children of drug abusers are reviewed below.

Prenatal Drug Exposure. A number of states legally define *in utero exposure* to alcohol and other drugs as child abuse. States with excessively punitive laws requiring child removal are rapidly changing these laws. For example, in California, the law mandating that infants be removed from detected drug-abusing mothers at birth has been modified; in San Francisco, a positive urine toxicology alone cannot be the only reason for removal. In many states, medical or social-services personnel are mandated to report such cases to protective-services workers; this can result in the avoidance of prenatal care by ATOD-abusing pregnant women. For this reason, social-services employees may hesitate to notify authorities. Additionally, notification of authorities can appear to be (and may in some cases be) racially biased and discriminatory if more poor women of color are referred. In one Florida study only one percent of white but ten percent of African-American drug-abusing pregnant women were reported to child-protective services.

Alcohol and other drugs can cause teratogenic effects—resulting in abnormalities in the fetus. Isolating the specific effects of individual drugs has been complicated by the large proportion of women who are polydrug abusers and by additional factors of poor nutrition, disease, stress, and lack of prenatal care. Alcohol and tobacco are the drugs most commonly used by women during pregnancy; as many as 1 percent of births may be affected by FETAL ALCOHOL SYNDROME (FAS). Some researchers assert that FAS may be the major cause of mental retardation. Characteristics of FAS include facial anomalies, retarded growth, and abnormalities of the heart, kidneys, ears, and skeletal system. The long-term effects of FAS are still being studied but appear to include reduced intelligence, attention deficits, learning disorders, hyperactivity, impulsivity, and more antisocial behaviors than the norm.

The perinatal and long-term effects of other drugs have been studied—such as COCAINE, METHAMPHETAMINE, MARIJUANA, OPIATES, and PHENCYCLIDINE (PCP). Although a number of immediate problems are apparent, including drug

withdrawal and developmental delays, with good postnatal environments many of these children can overcome their in utero exposure if structural damage is not severe. Some researchers have reported that even when cocaine-exposed infants were reared in adoptive homes from birth, some showed neurological deficits. Significant central nervous system (CNS) damage occurs with cocaine exposure. The major effects at birth of most drugs, however, including alcohol and tobacco, are preterm deliveries of low-birthweight infants, indicative of growth retardation that may affect both brain and physical development. Sudden infant death syndrome (SIDS) is also two to twenty times higher in infants exposed to cocaine and opiates.

Few longitudinal studies have tracked the impact of drug exposure on children. The longest follow-up study is of prenatal opiate-exposed children evaluated at ten years of age, and it is very difficult to separate the impact of a poor postnatal environment from prenatal drug exposure (unless the children are adopted). The few longitudinal studies conducted of prenatal drug-exposed infants have found almost no long-term developmental problems directly related to their drug exposure. A few cross-sectional studies of children of drug abusers have found clinically significant negative impacts on their emotional, academic, and behavioral status. These studies suggest that the greater the degree of maternal drug abuse, the greater the negative impact on the child's mental and behavioral status as measured by standardized clinical measures.

The quality of the infant's postnatal environment as actively constructed by the mother or caregiver appears to be the most significant factor in determining the impact of drugs on the drug-exposed or nondrug-exposed infant. Studies find that children born to drug-abusing mothers can look normal or be resilient to their in utero exposure to drugs if they are provided a nurturing environment that includes responsiveness to their needs, stimulation, and early childhood education.

Postnatal Exposure to Drugs. Children can be hurt by ingesting or inhaling alcohol, tobacco, and other drugs. In 1978, PCP was the second most common cause of poisoning in young children at Los Angeles Children's Hospital. Four major ways exist for children to become intoxicated: passive inhalation, accidental self-ingestion, being given drugs by a minor, and deliberate poisoning by an adult. In addition, infants can ingest alcohol, nicotine, and other drugs through breast milk. Passive inhalation of tobacco is recognized as a health hazard to children; however, passive inhalation of CRACK (freebase cocaine), PCP, marijuana, or hashish also has negative effects. Children living with parents who manufacture synthetic DESIGNER DRUGS in their homes, such as methamphetamine, are exposed to hazardous toxic chemicals. Some ATOD-abusing parents allow their children to drink alcohol or use the drugs they find lying around the house. Some parents deliberately give their children alcohol or other drugs (i.e., tincture of opium) to reduce their crying, sedate them, or to induce intoxication to amuse the parents. any relatively healthy child with unexplained neurological symptoms, seizures, or death may have been exposed to drugs.

Physical Abuse. Until recently, child-welfare agencies did not routinely screen for alcohol and drug abuse in caregivers of abused children. Because only about 40 percent of public child-welfare agencies and 71 percent of private child-welfare agencies even inquire about caregiver substance use, little is known about the incidence of substance abuse in child abuse cases. The Child Welfare League of America (CWLA) reported, after a 1990 survey of its 547 member agencies, that 37 percent of the children served by state agencies and 57 percent of children served by private agencies were estimated to be affected by ATOD family problems. A review of the literature found five studies suggesting a strong overlap between physical abuse and parental alcoholism. Physical and sexual abuse has been reported in 27 percent of alcoholic families and 19 percent of opiate-addicted families. Serious neglect was even more common (30.5%). Overall, 41 percent of children of addicts were found to be physically abused or neglected. In 1987, 50 percent of all reported child abuse and neglect cases in New York City were associated with parental drug abuse and 64 percent of cases were associated with parental alcohol and drug abuse. Of all child fatalities, 25 percent had related to a positive child drug toxicology (an overdose, OD).

ATOD abuse is frequently implicated when the courts remove a child from the home. A 1986 Illinois study indicated that 50 percent of all outplacements were from substance-abusing families and 68 percent of these parents refused ATOD treatment. Children growing up in abusive environ-

ments have increased, unfulfilled dependency needs, low self-esteem, distrust of others, and problems with aggression and anxiety.

Child Sexual Abuse. A high percentage of drug abusers report that they were sexually abused as children. Child molesters are often intoxicated when the abuse occurs. Alcohol's influence on the brain allows a disinhibition of socially proscribed behaviors, including incest and the sexual molestation of children. A 1986 review of the research suggests that alcohol is involved in about 30 to 40 percent of child sexual-abuse cases, particularly when girls are abused. A 1988 study found 48 percent of fathers who had committed incest were alcoholic but 63 percent of fathers were drinking at the time of the abuse. Because of the high heritability rate of male-limited alcoholism (the most severe type of alcoholism associated with early drinking and antisocial behavior in males), sexually molested children may be more genetically vulnerable to ATOD-abused antisocial behavior. Thus, the cycle of childhood sexual abuse is perpetuated over generations, because of the overlap between the two types of abuse.

Childhood sexual abuse is a major risk factor for greater psychological distress, dissociative experiences, depression, eating disorders, relationship, trust, and intimacy difficulties, post-traumatic stress response, psychotic disorder, and heavy drug abuse. A very high percentage of drug abusers at inpatient and residential treatment programs report being sexually abused as children. When direct questions were asked of the clients, men's reports of childhood sexual abuse increased from four percent to 16 percent for adult males, and to 42 percent for adolescent males. Reports by women increased from 20 percent to 75 percent of adult women, and to a high of 90 percent for adolescent women. Other studies indicate that between 25 and 44 percent of female drug abusers report childhood sexual abuse compared to 15 percent of nonaddicted women.

Psychological conflicts arising from childhood sexual abuse are often a hidden factor contributing to drug abuse and relapse. Sexually molested children are reported to experience boundary inadequacy, resulting in difficulty establishing and enforcing the personal, psychological, or social boundaries necessary to maintain a sense of the self that is separate from other people. Hence, survivors of childhood sexual assault often do not see themselves as individuals separate from the desires or demands of others. The concept of refusing another person access to their bodies (and in later life, to their privacy, time, physical space, and possessions) has not been incorporated into their sense of identity. This leaves the survivors vulnerable to subsequent violations or coercive tactics throughout their lives. It could also lead adult survivors to become perpetrators who abuse their own or other children because of their own boundary inadequacies.

Risk Factors for Child Abuse by Substance Abusers. Child-welfare authorities consider parental substance abuse to be a major risk factor for child abuse. Under the influence of alcohol and other drugs, adults are less inhibited and have reduced judgement and emotional control. Uppers (stimulants such as cocaine, methamphetamine, PCP, and amphetamines) can cause anxiety, irritability, paranoia, and aggressiveness. Downers (depressants such as alcohol, opiates, sedatives, and barbiturates) have also been related to depression, irritability, and loss of control while disciplining children. It has been suggested that organic brain damage, hypoglycemia, and sleep disturbances caused by alcohol exacerbates child abuse by alcoholics. ATOD-abusing parents are often irritable and angry because of neurochemical imbalances caused by persistent drug abuse. Some researchers attest that these neurochemical imbalances can last for several years after detoxification. Furthermore, neurotransmitter imbalances, which can be either biologically inherited or lifestyle-induced, may precede parental drug abuse and lead to self-medication with drugs. For example, excessively aggressive adolescent human and monkey males have been found to have lower levels of serotonin. Alcohol and carbohydrates increase brain levels of serotonin. Low levels of serotonin are associated with depression and eating disorders. Doctors prescribe serotonin-uptake inhibitors, such as fluoxetine (Prozac), to reduce mental disorder like depression and bulimia.

Psychosocial risk factors for child abuse include the following:

1. *Modeling Physical and Sexual Abuse and Violence* as seen in the child's home as enacted by adults or the abuser's friendship groups, or as portrayed in popular media (movies, television, radio). Drug abusers often belong to subcultures where violence is common. Children raised in

violent homes are more likely to become abusers as adults, thus perpetuating the cycle of violence.

2. *Family Violence and Conflict*. High levels of family conflict found in drug-abusing families can lead to family violence. Absence of empathy and support among family members in the home environment increases the risk of child abuse and family violence. Ironically, women who are victimized by their spouses have pregnancy rates 2.3 times higher than national averages. Children growing up in abusive homes experience increased anxiety, powerlessness, and self-deprecation, which may lead to ATOD abuse and, in turn, to aggression, conflict, and physical/sexual abuse of others.
3. *Poor Parenting Skills*. Drug-abusing parents or caretakers have been found to have less adequate parenting skills, spend less time with their children, have unrealistic developmental expectations that can lead to excessive punishment, and lax, overly severe, or inconsistent discipline. Verbal abuse in the form of threatening, chastising, belittling, and criticizing are common. Studies have found that drug-abusing parents, whether in recovery or not, are able to increase their parenting skills after participating in a 14-week parent-training program (The Strengthening Families Program).
4. *Poverty and Stress*. Many children of drug-abusing parents or caretakers are raised in poverty. Money that would normally be available for food, clothing, transportation, medical and dental care, and to provide social and educational opportunities for the children is often diverted into purchases of tobacco, alcohol, and drugs. Crack-addicted parents sometimes use food stamps and welfare checks to purchase crack. Lack of money to handle daily crises elevates the usual level of life stressors and increases parental anger and irritability. Unemployment, which frequently results in low self-esteem, can lead to increased child abuse.
5. *Mental Disorders*. Approximately 90 percent of drug abusers have other mental disorders, such as depression, bipolar-affective disorder, narcissism, ANTISOCIAL PERSONALITY, organic brain disease, and psychosis. Mental disorders of this nature can have a severe impact on a person's ability to parent and can lead to child abuse. Parents suffering from antisocial personality and narcissism are less empathic toward their children's suffering. It is harder to decenter from their own perspective, needs, and emotions in order to consider the child's feelings. Depression, bipolar disorder, and psychosis can cause parents to become angry, irrational, and abusive. Parents with personality disorders are less likely to internalize societal taboos against child abuse and sexual abuse.
6. *Physical Illness and Handicaps*. Physical illness and physical handicaps can reduce the patience parents need to handle the stress inherent in dealing with children. Physical illness is more common in ATOD-abusing families because of their lifestyle and lack of preventive health care. Intravenous drug abusers and their children have higher rates of common infections, as well as increased exposure to diseases transmitted through the blood (HIV/AIDS and hepatitis), sexually transmitted diseases (syphilis, gonorrhea, and herpes), and tuberculosis.
7. *Criminal Involvement*. Drug-abusing parents are at high risk for criminal involvement by nature of their use alone or by the need to obtain considerable sums of money to support their habit. Prostitution, theft, and drug dealing are reported in about half of all drug-abusing parents. Arrest and incarceration may increase the stress on the family and can reduce inhibitions to sexual abuse upon reunification of the family.

Children of ATOD abusers frequently are more difficult to parent because of the increased prevalence of ATTENTION DEFICIT DISORDER (ADD), hyperactivity, CONDUCT DISORDERS, and learning disorders. Some of these difficult temperament characteristics are caused by in utero exposure to drugs, some by genetic inheritance, and others by lack of nurturing and inconsistent parenting. Regardless of the cause, children of ATOD-abusing parents are frequently the most difficult to raise, even if they are raised by unstressed, happy, healthy, non-ATOD-abusing parents.

PROPOSED RESPONSES

Reasonable evidence exists to indicate that children who are raised by ATOD-abusing parents are at increased risk of abuse and neglect, as well as of subsequent addiction and delinquent behaviors. Additional research is needed on the long-term ef-

fects of reported physical or sexual abuse. Because of the high overlap between child abuse and drug abuse, ATOD treatment agencies should routinely ask their clients if they have been or are being physically or sexually abused. It is also important that child-welfare agencies routinely determine whether caregiver or family member ATOD abuse is contributing to the maltreatment of children.

Because it is not possible to remove all children from risky family environments, additional research is needed on ways to protect children. Caregivers and professionals can help maltreated children to avoid abuse or become more psychologically resilient to future ATOD abuse. Some children are resilient to negative outcomes, even though they were exposed to drugs in utero or lived with drug-abusing parents. Some of these children were really never exposed to the same degree of negative influences because they were sheltered by a caring adult who addressed their needs. The emerging literature on resiliency processes and mechanisms should be reviewed and used to inform resiliency research with children of drug abusers and to make prevention interventions more effective.

Negative outcomes primarily appear to be related to the physical and emotional abuse and neglect typically endured by children of drug-abusing parents. Even children of drug-addicted mothers can be resilient to their high-risk environments if their mothers realize the negative impact of their chaotic street lives on their infants and work to improve their parenting skills. This may include finding external supports to learn parenting skills, such as parent-and-family-skills training programs, locating good early-childhood education for the child and outside child care, and possibly even considering foster care or adoption. Research has shown more positive outcomes for drug-exposed infants if the mothers were willing to use whatever external social supports were necessary to provide the best opportunities for learning and emotional growth for the child. Such mothers clearly were able to understand and empathize with their children's needs and were willing to separate from their infants for short or long periods, if necessary, for the welfare of their children.

(SEE ALSO: *Childhood Behavior and Later Drug Use; Coping and Drug Use; Family Violence and Substance Abuse; Poverty and Drug Use*)

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KAROL L. KUMPFER
JAN BAYS

CHILDHOOD BEHAVIOR AND LATER DRUG USE Social scientists can point with confidence to risk factors from childhood that predict drug use and deviance in adolescence. In general, the findings indicate that certain childhood personality traits, family experiences, and ecological factors strongly affect adolescent drug-using behavior.

A child who is irritable and easily distracted, who throws temper tantrums, fights often with siblings, and engages in predelinquent behavior is more likely than others to use drugs in adolescence. Other investigators have also found childhood AGGRESSION to be a most powerful predictor of adolescent drug use and deviance.

Poor childhood impulse control and a difficult temperament have been related to adolescent marijuana use. When problematic factors continue into adolescence, both the use of illicit drugs and the psychopharmacological effects of some drugs may then actually serve to exacerbate and enlarge the adolescent's feelings of irritability and aggressive-

ness—as evidenced by continuing temper tantrums, aggression, and delinquent behavior.

In addition to childhood personality traits that predict future drug use, family experiences can also serve as predictors. In a longitudinal study dealing with the early childhood precursors of adolescent drug use, researchers found that greater mother involvement with the child protected against later drug use. It has also been found that children who became frequent drug users had mothers who were cold and who gave them little encouragement. The connection between peer factors during childhood and later drug use has received little empirical study, although peer factors during adolescence are of demonstrable importance and have long been known to be so. Childhood ecological factors, such as relatively low socioeconomic status, are related to greater adolescent drug use.

Factors from childhood do not directly affect adolescent drug use; rather, they are mediated by the factors of adolescence. More specifically, the risk factors of childhood are associated with the risk factors of adolescence—and these, in turn, become related to drug use.

The risk factors of adolescence include aspects of unconventionality (such as rebelliousness); difficulty in the parent-child mutual attachment relation (such as low parental affection and identification); and the adolescent's associating with deviant peers. More specifically, findings indicate that nonachieving aggressive children—those with difficulty in emotional control and those who have received little economic and psychosocial support—are most likely to be rebellious adolescents, to have a conflictual, nonaffectionate relationship with their parents, and to be associated with deviant peers. Adolescent rebelliousness, difficulty in the parent-child attachment, and associating with deviant peers are the factors related to greater drug use in adolescence.

PREVENTION AND TREATMENT

Because the risk factors of both childhood and adolescence come from different domains, a multifactorial approach to drug-use prevention and treatment is essential. Moreover, since childhood drug-prone personality characteristics and adverse childhood experiences seem to determine adolescent risks for drug use, logic and social responsibility dictate that one intervene early in the development of a child at risk. Where needed or warranted, early intervention may facilitate the development of later drug-resistant personality traits—a positive parent-child bond and association with nondeviant peers—and, consequently, the result should be lower drug use in the adolescent.

As children grow up, there are also several later points and distinct psychological domains at which it is possible to ameliorate drug use. During adolescence, for example, a decrease in the risk factors that relate to personality, family, or peers may also result in less to no drug use.

(SEE ALSO: *Child Abuse and Drugs; Conduct Disorders; Coping and Drug Use; Families and Drug Use; Family Violence and Substance Abuse; Poverty and Drug Use*)

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CHINA *See* Asia, Drug Use In; Golden Triangle

CHINESE AMERICANS, ALCOHOL AND DRUG USE AMONG In 1980, the Chinese-American community, with a population of 812,178, comprised the largest subpopulation of Asian/Pacific Islanders in the United States. During the 1980s, the population of Chinese Americans nearly doubled—1,618,973 according to the 1990 data from the U.S. Bureau of the Census (although the Filipino-American community had by then become the largest Asian subgroup). The largest numbers of Chinese Americans reported in the 1990 census are in the states of California (704,850), New York (284,144), Hawaii (68,804), Texas (63,232), New Jersey (59,084), Massachusetts (53,792), and Illinois (49,936). The Chinese-American ethnic community actually consists of people from many countries, and recent waves of immigration especially contribute to the heterogeneity of this ethnic group. Chinese immigrants have come to the United States from British Hong Kong, the People's Republic of China, the Republic of China (Taiwan), and from various countries in Southeast Asia, Latin America, and the Caribbean. Approximately 63.3 percent of the Chinese-American respondents to the 1980 census had been foreign (non-U.S.) born.

ALCOHOL

In China, historically, alcohol was sanctioned for religious ceremonies, especially ancestor worship. Today, in China and among Chinese immigrants, alcohol is commonly served at celebrations and banquets, and some people consume alcohol at meals—beer, wine, brandy, or whiskey. Drinking-centered institutions, however, are absent (Hsu, 1955; Singer, 1972; Wang, 1968). In Chinese tradition, moderate drinking is believed to have medicinal effects, but excessive use is believed to bring on "nine-fold harm" (Yu & Liu, 1986/87) and is condemned in folk culture as one of the four vices. Many hypothesize that cultural influences are important in shaping drug-use patterns as well as beliefs about drug use. Some research ties cultural beliefs to differences in drinking patterns, despite similarities in availability (Glassner & Berg, 1980; Mizruchi & Perrucci, 1962).

Chinese cultural beliefs regarding the religious and medicinal benefits of moderate drinking and the harm associated with excessive use may control drinking patterns in China, but when people move into a new cultural setting, their alcohol use may be influenced by the extent to which they adopt the values of the surrounding culture. Sue (1987) states that alcohol abuse is more congruent with American than Chinese values, since Chinese values are antithetical to alcohol abuse. This "acculturation hypothesis" (Austin & Lee, 1989) has received mixed support with respect to the experience of Chinese Americans. This suggests that more investigation is necessary to help determine which influences result in the retention of cultural values and which result in adaptation to the new culture.

OPIUM

OPIUM is thought to have been introduced to China by Arab traders during the ninth century. Initially, it was taken internally as medicine (Singer, 1974). Not until the mid-seventeenth century was the practice of smoking opium (usually in pipes) introduced by the Portuguese. Little of the opium poppy (*Papaver somniferum*) was actually grown or used in China before the sixteenth century. By the eighteenth century, however, opium had become a profitable cash cargo—from British India to China's ports, where foreigners were allowed only confined access to trade—for the Portuguese, Dutch, and English—and then after 1810 for the Americans (Goodie, 1963). Smoking opium had become so widespread and so debilitating in China that its sale was forbidden by imperial decree as early as 1729 and its importation was prohibited in 1800. The emperor's declarations were not universally honored, however, and much disagreement existed on how to deal with opium addictions, the drain of silver to foreigners, and the tribute system of then-developing foreign relations (Fairbank, Reischauer, & Craig, 1965).

Meanwhile, an illicit opium trade continued to grow—for example, from approximately 5,000 chests imported to Canton in 1821 by British traders to approximately 30,000 chests by the late 1830s (Fairbank, Reischauer, & Craig, 1965). Efforts in an anti-opium campaign were stepped up, and hostilities between China and Britain eventually led to the Opium Wars. Britain had asserted that it was not bound by the trade restrictions imposed on Canton, and Britain won the wars. As a result, Hong Kong, a major port and center for all kinds of trade, was ceded to Britain in 1842. Illicit opium remained an important export until 1911, at

which time the British Parliament forbade its shipment to China. By this time, however, cultivation of the opium poppy was flourishing in China, and markets for MORPHINE, HEROIN, and other narcotic concentrates were growing. Although opium dens provided an atmosphere and opportunity for drug use by individuals or as a social activity, in China opium smoking remained one of the four vices.

Much of the research on ALCOHOL and other drug use has grouped all Asians and Pacific Islanders together. Only two studies have compared Asian groups, and they have suggested significant differences among them. In a 1981 study conducted in Los Angeles, Kitano and Chi (1986–1987) found differences in alcohol consumption patterns among respondents from four groups of Asians: Chinese, Japanese, Korean, and Filipino. Most of the respondents were from thirty to sixty-one years old. Except in the Japanese sample, the majority were foreign born and most had an average annual income of 20,000 to 30,000 dollars. Among these four groups, the following identified themselves as abstainers: 31.2 percent of Chinese males and 68.8 percent of females; 32.8 percent of Japanese males and 33.8 percent of females; 34.5 percent of Filipino males and 80.0 percent of females; and 45.8 percent of Korean males and 81.6 percent of females.

The lowest prevalence of heavy drinking was reported by Chinese Americans (14% male, 0% female), followed by Koreans (25.8% male, 0.8% female), Filipinos (29.0% male, 3.5% female), and Japanese (28.9% male, 11.7% female). Most of the male heavy drinkers were in the age category 26–35 among Chinese, in the age category 36–45 among Koreans, and evenly divided among age categories for Japanese and Filipinos.

Kitano and Chi found that among Chinese Americans in their Los Angeles sample those most likely to drink at any level were men, under the age of forty-five, and of relatively high social and educational background. They found that parental drinking and going to or giving parties were the most important variables distinguishing drinkers from abstainers among their Chinese adult male sample (Chi, Kitano, & Lubben, 1988). Going to bars and having friends who drank were also significant factors.

CONCLUSION

More rigorous surveys are still needed to obtain an accurate picture of alcohol- and other drug-use patterns among Chinese Americans. Since this is a heterogeneous group, future studies should take into account whether people in the sample are U.S. or foreign born, their country of origin, their degree of acculturation, and other demographic characteristics that will provide a better basis for comparison with other groups.

Although it has long been asserted that responses to drug problems should be sensitive to cultural diversity, until recently little research has focused on drug use among people other than blacks or whites in the United States. Such research would be useful for developing culturally appropriate interventions.

(SEE ALSO: *Ethnic Issues and Cultural Relevance in Treatment; Ethnicity and Drugs; Papaver somniferum*)

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CHLORAL HYDRATE Chloral hydrate is one of the oldest sedative agents still in use. It was made by the German chemist Liebig in 1832 and introduced into general use in 1869 as a substitute for LAUDANUM, an alcoholic solution of OPIUM. Chloral hydrate differs from the BARBITURATES in that it is a simple molecule composed of two carbon atoms, three hydrogen atoms, two oxygen atoms, and three chloride atoms. It is the famous (or infamous) substance added to alcohol to make a *Mickey Finn*, a drink known to cause those who drink it to become unconscious. Because it shares many effects of other central nervous system depressants, it can be used to treat the alcohol withdrawal syndrome. Chloral hydrate was a popular sedative for elderly patients because its effects occur quickly, last only a short time, and leave no nagging hangover effect. However, it is inconvenient to use (up to 2 grams must be taken by mouth) and, after the introduction of the BENZODIAZEPINES, its use has decreased.

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CHLORDIAZEPOXIDE Chlordiazepoxide (brand name Librium) is a member of the BENZODIAZEPINE family of drugs currently used to treat insomnia, anxiety, muscle spasms, and some forms of epilepsy. It was the first benzodiazepine to be used in clinical practice in the 1960s, as an alternative to PHENOBARBITAL or MEPROBAMATE, in treating psychoneuroses, anxiety, and tension. Its advantage over BARBITURATES and other central nervous system depressants is that it is less toxic, especially after an overdose.

In addition to the previously mentioned uses, chlordiazepoxide is frequently used to treat the seizures or DELIRIUM TREMENS (DTs) that appear during alcohol withdrawal. In the late 1990s, Dr. Michael Mayo-Smith conducted a meta-analysis to determine if benzodiazepines effectively prevent delirium in patients experiencing DTs. Although benzodiazepines were shown to be effective, this study was not conclusive since chlordiazepoxide was the only benzodiazepine tested, and further testing is needed on other benzodiazepines before an overall claim can be made (Johnson et al., 1997).

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CHOCOLATE An ingredient of many popular treats—candies, sweets, baked goods, soft drinks, hot drinks, ice cream, and other frozen desserts. It is prepared, often as a paste, from the roasted crushed seeds (called cocoa beans) of the small South American cacao tree called *Theobroma cacao* (this is not the shrub known as the COCA PLANT, which produces COCAINE, *Erythroxylon coca*).

The cacao tree has small yellowish flowers, followed by fleshy yellow pods with many seeds. The dried, partly fermented fatty seeds are used to make the paste, which is mixed with sugar to pro-

Figure 1
Cacao Leaves and Pod

duce the chocolate flavor loved throughout the world. Cocoa butter and cocoa powder are other important extracts from the bean. Cocoa beans were introduced to Europe by the Spanish, who brought them back from the New World in the sixteenth century. They had first been used by the civilizations of the New World—Mexicans, Aztecs, and Mayan royalty—in a ceremonial unsweetened drink and as a spice in special festive foods, such as molé. They were first used in Europe by the privileged classes to create a hot, sweet drink. By the seventeenth century, cocoa shops and COFFEE shops (cafés) became part of European life, serving free TOBACCO with drinks and thereby increasing trade with the New World colonies.

Chocolate produces a mild stimulating effect caused by the THEOBROMINE and CAFFEINE it contains. Both are ALKALOIDS of the chemical class called xanthines. Theobromine in high doses has many effects on the body, and it is possible to become addicted to some xanthines, such as caffeine. Nevertheless, some people are so attracted to the flavor that compulsive or obsessive use has resulted in the newly coined term *chocoholic*. Some scientists are researching the phenylethylamine in chocolate as the factor that encourages compulsive chocolate ingestion.

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CHROMOSOME Chromosomes are structures in the nucleus of the cell that contain the DNA or hereditary material which form genes. Genes are the commonly known units of heredity, and some may contribute to a tendency toward addiction in ways that are not yet understood. Each chromosome is an elongated structure that is clearly visible during cell division. Humans possess twenty-three pairs including the sex chromosomes. A male has an X and a Y sex chromosome, whereas a female has two X sex chromosomes. One of each pair comes from each parent.

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CHRONIC PAIN Chronic or persistent pain is defined as pain that lasts for longer than six months. Chronic pain can stem from cancer, illness, injury, or postsurgical changes. Often, persons with chronic pain suffer from syndromes that cannot be confirmed by laboratory tests. These chronic pain syndromes include central pain syndromes, fibromyalgia, headache, lower back pain, myofascial pain syndrome, neuropathic pain, and phantom limb pain. Frequent locations of chronic pain include the back, head, joints, chest, abdomen, and extremities. Chronic pain is common and its sufferers are more likely to have anxiety or depression, have poor perception of their health, decreases in their quality of life, and experience a disruption of their livelihood than those who are not in pain.

In most cases, there is no cure for the chronic pain so treatment is aimed at pain control and rehabilitation. Unfortunately, chronic pain is often ineffectively treated because physicians can be reluctant to prescribe strong, potentially addictive medications. The ineffective pain treatment is compounded by commonly associated conditions such as depression, insomnia, fatigue, and a decrease in general physical functioning. Therefore, treating the pain alone is not sufficient.

The optimal approach to the chronic pain sufferer is an interdisciplinary team that may be comprised of a pain management physician, nurse specialist, psychologist, physical therapist, pharmacist, and vocational counsellor. The physician conducts a thorough assessment of the patient and determines the appropriate medical interventions. The psychologist conducts a thorough psy-

chological assessment, educates the patient on techniques to reduce pain, and tends to any associated mental health illnesses. The nurse specialist acts as a case manager and educator. The physical therapist ascertains the patient's physical endurance, flexibility, and strength and conducts the physical rehabilitation process. The vocational counsellor identifies and devises strategies to allow the patient to return to work. In addition to dispensing medications, the pharmacist will review past and present use of medicinal agents and educate the patient on the proper use of medications.

PHARMACOLOGICAL MANAGEMENT OF CHRONIC PAIN

In the treatment of chronic pain, drugs (analgesics) are usually administered in a stepwise fashion beginning with mild, relatively safe agents and progressing to stronger agents as necessary. In 1986, the World Health Organization (WHO) proposed a stepwise plan, frequently called the ANALGESIC LADDER, for the oral treatment of cancer pain. This plan provides adequate pain relief for up to 90 percent of cancer patients but may have limited success for other chronic pain patients. Step one of the ladder is recommended for patients with mild pain and consists of nonopioid analgesics, step two is for moderate pain and consists of mild opioids, and step three is for severe pain and consists of strong opioids. More than one analgesic may be used at a time for an added effect, a procedure called adjuvant therapy.

Nonopioid analgesics consist of acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs). Acetaminophen is an effective analgesic that has a minimal side effect profile. Nonsteroidal anti-inflammatory drugs have both analgesic and anti-inflammatory properties. Examples of nonsteroidal anti-inflammatory drugs are aspirin, ibuprofen, naproxen, meclofenamate, piroxicam, and more recently celebrex and vioxx. Major side effects associated with the use of nonsteroidal anti-inflammatory drugs include kidney toxicity, bleeding disorders, and stomach disorders.

Opioid analgesics are available in different strengths. Examples of opioid analgesics are morphine, fentanyl, methadone, and meperidine. The side effects of opioids may be much more serious than those seen with nonopioid analgesics. Side effects include respiratory depression, alterations in consciousness (e.g. drowsiness, sedation, confusion), nausea, vomiting, constipation, urinary retention, and itching.

Other medications used in the treatment of chronic pain include antidepressants and anticonvulsants. Nerve blocks, injection of anesthetics into trigger points, or injection of steroids into the epidural space of the spinal cord may also be utilized. Implantable methods are utilized as treatments of last resort. These methods involve implanting drug delivery systems or electrodes into specific areas of the spinal cord.

TOLERANCE, DEPENDENCE, AND ADDICTION

The continued use of opioids leads to tolerance, in which increasingly higher doses of drug must be used to obtain the original level of pain relief. Tolerance develops slowly, occurring over a period of months to years. Cross-tolerance to other opioids develops, although to a lesser extent. Tolerance can be differentiated from physical dependence and addiction.

Physical dependence is a characteristic of opioid use because of the mode of action. It reflects a state of neurological adaptation to the drug. With physical dependency, discontinuation of opioid use leads to withdrawal symptoms (e.g. sweating, tearing, rapid heart rate, nasal discharge, abdominal cramps, nausea, and vomiting). To prevent withdrawal symptoms, patients on long term opioid use are gradually weaned off the medication. Physical dependence on opioids does not lead to addiction, although it may compel the patient to seek opioids to relieve symptoms of withdrawal.

For chronic pain patients taking opioids, tolerance and physical dependence are not indicators of addiction. Addiction is not a characteristic of opioid use, rather, it is dependent upon the user. In fact, the medical use of opioids is only very rarely associated with addiction. The agonist-antagonist class of opioids (buprenorphine, butorphanol, nalbuphine, pentazocine, and dezocine) has a low abuse potential.

Any patient taking opioids to treat chronic pain can meet the criteria for addiction set forth by the American Psychiatric Association in the *Diagnostic and statistical manual of mental disorders: DSM-IV*. Therefore, it is very difficult to diagnose addiction in chronic pain patients who are taking opi-

oids. Chronic pain patients who are being ineffectively treated could display the drug-seeking behavior that is characteristic of addiction, a phenomenon called *pseudoaddiction*. Alternatively, the patient receiving effective pain treatment may take extreme measures to insure an adequate supply of medication. This behavior is termed *therapeutic dependence*.

Suggestive signs of addiction within the context of opioid therapy for chronic pain include:

- Loss of control over opioid use;
- Preoccupation with the use of opioids despite adequate pain control; and
- Continued use of opioids even with their adverse consequences.

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CIGARETTE COMPANIES *See* Advertising and Tobacco Use

CIGARETTES AND SMOKING *See* Nicotine; Tobacco, History of; Treatment: Tobacco, An Overview

CIRRHOSIS *See* Complications: Liver

CIVIL COMMITMENT The term commonly used for compulsory treatment is *civil commitment*. Typically, civil commitment serves as an alternative to incarceration (prison) by providing compulsory, court-ordered treatment for chronic drug abusers, especially antisocial addicts responsible for committing a large number of criminal acts. It is generally believed that narcotic addicts must be brought into a supervised environment for an extended period of time for any treatment to be meaningful. Civil commitment is a useful strategy for diverting into treatment those who ordinarily would not seek assistance voluntarily, and it has been shown to suppress daily narcotic use and criminal involvement (Leukefeld & Tims, 1988).

HISTORICAL CONTEXT

The concept of compulsory treatment for drug abusers in the United States was proposed shortly after Congress passed the Harrison Act of 1914. By 1919, the Narcotics Unit of the U.S. Treasury Department had convinced Congress to establish a chain of federal "narcotics farms," where heroin users convicted of federal law violations could be incarcerated and treated for addiction (Inciardi, 1988). The first of such farms, established in 1935, was the U.S. Public Health Service Hospital in Lexington, Kentucky. Three years later, a sister hospital was established in Fort Worth, Texas. The goal of the facilities was to use vocational and psychiatric therapy to help free the addicts of their psychological dependence on drugs, to treat withdrawal illness, and to correct mental and social problems. Follow-up studies from Lexington in the 1940s indicated that addicts treated under legal coercion with posthospital supervision had better outcomes than voluntary patients, primarily because voluntary patients rarely completed the treatment program (Maddux, 1988). However, later studies failed to support these early positive findings. During the 1950s, hospital staff members recommended the enactment of a federal civil commitment law for narcotic addicts, but legal counsel in the Department of Health, Education, and Welfare considered such a law unconstitutional.

Then in 1962, President John F. Kennedy convened a White House Conference on Narcotic and Drug Abuse where nearly all members approved the civil commitment of narcotic addicts. Civil commitment was advocated as protection for society and rehabilitation for the individual. A compulsory treatment program aimed at the federal offender was enacted by the NARCOTIC ADDICT REHABILITATION ACT (NARA) of 1966. By that time, about twenty-five states had laws permitting civil commitment, and a few major programs were enacted in response to public fears of growing drug-related street crime (Inciardi, 1988). California, in 1961, launched its Civil Addict Program

(CAP), the first large-scale civil commitment program to be implemented in the United States. Because of its relative success, in 1966, New York's Narcotic Addiction Control Commission (NACC) established the largest and costliest civil commitment program in history—the NEW YORK STATE CIVIL COMMITMENT PROGRAM.

CIVIL COMMITMENT PROGRAMS

The federal NARA and the California and New York compulsory treatment programs had a similar intent: They made it possible for the necessary legislation to be enacted and for commitment procedures to be carried out. They served to control and rehabilitate the compulsive drug abuser by providing secure treatment environments as an alternative to regular incarceration in correctional facilities (Leukefeld & Tims, 1988). Eligible addicts convicted of a crime could be committed by the court or could choose commitment over incarceration. Addicts not involved in criminal proceedings could commit themselves voluntarily or could be involuntarily institutionalized upon the petition of another individual (such as a peace officer) (McGlothlin, Anglin, & Wilson, 1977). Integral to each of these programs was supervised aftercare with antinarcotics testing. Length of commitment terms ranged from three years in NARA to seven years in CAP.

Although nearly all of NARA's civil commitment programs were generally considered unsuccessful, the funding for community programs contained within the same legislation did provide seed money for the nationwide establishment of some of today's basic drug-treatment programs in the community (Maddux, 1988). It was also believed that the New York State Civil Commitment Program failed, and the process of dismantling it began in 1971 (Inciardi, 1988). The failure of this program is partly attributable to the fact that it was administered by the social welfare agency of New York State, which had little experience controlling addicts. In contrast, CAP, California's program, was deemed at least moderately effective in modifying behavior, because it was implemented through the California Department of Corrections, which had trained personnel who were familiar with treating substance abusers (Anglin, 1988).

Follow-up studies of the California program found that participants exhibited reductions in daily heroin use as well as in property crime and antisocial activities. Although many patients did become readdicted at some point, their relapses were typically of shorter duration and less frequent than those not involved in treatment (Anglin, 1988). The general conclusion drawn from these studies was that civil commitment, when adequately implemented, might be an effective means of reducing narcotic addiction and of minimizing adverse, addiction-related behavior. Repeated interventions are typically required, however, because drug dependence is a chronic condition marked by relapses (Leukefeld & Tims, 1988).

LIMITATIONS OF CIVIL COMMITMENT

Civil commitment helps get drug abusers into treatment and keeps the abusers in treatment for an extended length of time. Outcome studies have generally shown that the success of treatment is directly related to the amount of time spent in treatment and that long-term supervision upon return to the community, with objective monitoring (DRUG TESTING), is an essential component of a successful program. Furthermore, civil commitment often makes treatment available before a crime is committed, and it provides clear treatment goals rather than only providing punishment (Leukefeld & Tims, 1988). Still, such a program as civil commitment has serious limitations. It is costly and can overwhelm facilities unless adequate funding, facilities, and staff have been made available. Many addicts are considered unwilling or unsuitable for participation. External coercion can bring drug users into treatment, but it cannot assure that as patients they will participate in treatment. Even with the advent of intensive interventions designed to engage the patients, some patients simply participate passively (Maddux, 1988). Finally, the scope of civil commitment is restricted by constitutional guarantees of individual liberty. The question remains: Within a free society, to what extent should the government curtail the civil liberties of a compulsive drug user?

Today, there is an increasing tendency to see civil commitment as control rather than treatment and it serves only a limited number of addicts who are sufficiently problematic in their behavior to warrant commitment. The use of such measures as civil commitment in a better coordinated and expanded fashion, however, could produce significant

individual and social benefits (Anglin, 1988). Civil commitment may also gain more popular support as a means for dealing with intravenous drug users who are at risk for contracting or transmitting AIDS.

(SEE ALSO: *Coerced Treatment for Substance Offenders; Treatment Alternatives to Street Crime* [*TASC*])

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HARRY K. WEXLER

CLINICAL TRIALS NETWORK In an effort to find the most effective treatments for drug addiction, the National Institute on Drug Abuse (NIDA) has established a clinical trials research network to test new pharmacological and behavioral treatments in diverse patient populations. Clinical trials have been used for diseases such as cancer and AIDS as a fast, effective, and safe way to test new treatments. Also, as with other diseases, there are a number of effective treatments for addiction. However, the efficacy of these new treatments has been demonstrated primarily in specialized treatment research settings, with somewhat restricted patient populations. As a consequence, few of these new drug-abuse treatments are being applied on a wide-scale basis in real-life practice settings

In response, NIDA has established the National Drug Abuse Treatment Clinical Trials Network (CTN). The CTN is based on a model used successfully by other NIH institutes, including the National Cancer Institute, the National Heart Lung and Blood Institute, and the National Institute of Allergy and Infectious Diseases. The CTN provides a research infrastructure to test whether new and improved treatment components are effective in real-life settings with diverse patient populations.

THE CTN STRUCTURE

NIDA has established the first six nodes of the CTN in various regions of the country. Each node or functional unit of the CTN is affiliated with a research-based organization and a number of drug-abuse treatment programs in the community. The CTN brings together researchers and practitioners as partners to conduct full-scale testing of promising new medications and behavioral treatments in a wide range of community drug-abuse treatment settings with patients from a variety of ethnic and social back-grounds. (The nodes to date include nodes in New England, the Delaware Valley, the Mid-Atlantic, the Northwest, the Pacific region, and New York.) Each of these centers is linked with at least five community treatment programs in its region. CTN research is carried out in the community-based treatment setting. Each node works with the other nodes and with NIDA to conduct multisite and cross-regional clinical trials research.

THE MISSION OF THE CTN

The overall goal of the CTN is to improve the quality of drug-abuse and addiction treatment throughout the nation using science as the vehicle.

Toward this end, the mission of the CTN is threefold:

(1) Conduct studies of behavioral, pharmacological, and integrated behavioral and pharmacological treatment interventions in multisite clinical trials to determine effectiveness across a broad range of community-based treatment settings and diversified patient populations.
(2) Transfer the research results to physicians, providers, and their patients to improve the quality of drug abuse treatment throughout the country using science as the vehicle.
(3) Provide advice on changing policies to ensure the delivery of effective therapies in community-based treatment programs.

CURRENT AND FUTURE DIRECTIONS

Three science-based treatment research protocols will start in 2000, including two behavioral therapies developed to enhance treatment outcomes, and one that will test a new medication for use in opiate detoxification. Several other protocols are currently being developed. All treatment components to be tested have been shown to be effective in controlled research environments.

When complete, it is expected that the network will consist of twenty to thirty nodes consisting of regional research treatment centers linked to ten to fifteen community-based treatment programs that represent the variety of settings and patient populations prevalent in that particular region of the country. The CTN will help ensure that treatment research in drug abuse and addiction meets the needs of the wider community, including minorities, women, children, adolescents, and underserved populations. The CTN will also be useful to other aspects of NIDA's research portfolio. For example, multi-site clinical trials with diverse patient populations will provide a valuable resource to researchers interested in elucidating genetic and environmental determinants of vulnerability. Ultimately, increased understanding of the roles played by genetics, environment, and their interaction in shaping an individual's susceptibility to drug addiction will lead to a variety of more targeted drug abuse prevention and treatment approaches. For more information, visit NIDA's website at www.nida.nih.gov.

For more information about NIDA's National Drug Abuse Treatment Clinical Trials Network, visit the NIDA website at www.drugabuse.gov.

ALAN I. LESHNER

CLANDESTINE LABORATORIES *See* Amphetamine Epidemics; Colombia as Drug Source

CLASSIFICATION OF DRUG TYPES *See* Drug Types

CLONE, CLONING A clone is a group of organisms that derive from a single ancestor and are genetically identical. A clone can be a group of mammals such as sheep, or a group of cells in culture.

Cloning cells is a powerful tool in biology and medicine, since growing large quantities of identical cells allows for a large harvest of the various identical and useful components of these cells. It is possible to construct genetic components in the laboratory, place them in cells, and then have the cells grow and multiply to produce large quantities of the components.

Cloning is an essential technique in modern molecular biology; it is used widely in studying genetic effects in the drug-abuse field. Cloning much larger organisms such as cows and sheep is expected to have a major impact in that production of the best of any species can theoretically be accomplished by cloning. This is an important goal in agriculture today.

MICHAEL J. KUHAR

CLONIDINE While not itself life-threatening, the opioid WITHDRAWAL syndrome is extremely unpleasant and contributes to further opioid use and relapse. HEROIN addicts report that the acute withdrawal syndrome begins in approximately eight hours after their last injection and

includes the following: craving for the drug, anxiety, perspiration, perspiration with hot and cold flashes, tearing of the eyes and nose, restlessness, problems sleeping, problems falling asleep, goose bumps, aching bones and muscles, loss of appetite, nausea, vomiting, diarrhea, abdominal cramps, spontaneous yawning, and a group of symptoms called flu-like.

During the later years of the nineteenth century and early years of the twentieth, some cures for this opioid withdrawal syndrome have been far worse than the withdrawal itself—with some actually causing death. Soon after it became available in the mid-nineteenth century, injectable MORPHINE was proposed as a treatment for opium eating; then heroin or COCAINE were, in the late nineteenth century, proposed as cures for morphine addiction. From the mid-twentieth century until the 1970s, most medical treatment of the opioid withdrawal syndrome involved either gradual reduction of the dose of opioid or the substitution of METHADONE, followed by its gradual reduction. In 1978, Gold and coworkers proposed that the nonopiate antihypertensive clonidine could be an effective nonopiate treatment for opiate withdrawal distress. The scientific basis for the proposition that clonidine would be useful was based on the hypothesis that the opiate withdrawal syndrome was caused by hyperactivity or hyperexcitability of a specific brain nucleus composed of noradrenergic neurons, called the locus coeruleus (LC). There was considerable neuroscientific research to support this withdrawal hypothesis and the rationale for the efficacy of clonidine.

Since 1977/78, clonidine has been tried in numerous inpatient and outpatient opioid addict populations worldwide and studied by researchers in numerous well-controlled studies. In virtually all studies, clonidine has been shown to be a safe and effective nonopioid treatment that could control several aspects of opioid withdrawal. Clonidine, while having opiate-like effects in reversing several aspects of opiate withdrawal, is not an opiate and is therefore not subject to the burdensome regulatory CONTROLS that have been placed on the use of opioids. Clonidine has its most demonstrable effects on autonomic elements of opioid withdrawal: sweating, gastrointestinal complaints (cramps, diarrhea, nausea), and elevated blood pressure. It does not have substantial capacity to alleviate muscle aches, insomnia, or craving for opioids.

Research and clinical experiences since the original discoveries have (1) supported the notion of LC hyperactivity as one of the neural substrates for the opioid withdrawal syndrome; (2) supported the efficacy of clonidine—establishing clonidine detoxification as one of the standard treatments for adult opioid addicts—and extended it to neonates (newborns) and to alcohol, nicotine, and other drug withdrawals that share a preponderance of behaviours with opioid withdrawal; (3) demonstrated that abstinence could be maintained by some opioid addicts and that others could benefit from antagonist therapy with NALTREXONE, thanks to clonidine or accelerated clonidine-naltrexone detoxification; (4) led to considerable progress in the understanding of the critical cellular event causing LC hyperactivity in opioid withdrawal and hypoactivity in the presence of clonidine or opioid agonists; and (5) led to the rapidly expanding clinical armamentarium available to treat addicts on the basis of rodent and primate studies.

CLONIDINE SHORTENS DETOXIFICATION

Detoxification of opioid addicts with clonidine has been used to facilitate the transition from chronic opioid administration to naltrexone (a long-acting opioid ANTAGONIST) or to drug-free status. Naltrexone possesses opioid-blocking action at all opioid-receptor sites in the body and brain, rather than having an affinity for a specific type of opioid receptor. It is a useful medication for those patients willing to take it to prevent relapse. When recovering addicts take naltrexone, they make opioid effects unavailable to themselves. The affinity of naltrexone for the receptors is such that they are unable to feel the effects of heroin, methadone, or other exogenous opioids. While the original clonidine treatment protocol of Gold and his colleagues (1978a, b) facilitated the initiation of naltrexone by avoiding the extra ten-day wait required after the last methadone, an accelerated detoxification protocol has been developed using naltrexone and clonidine simultaneously. Since clonidine reduces precipitated withdrawal as well as the withdrawal that results from simply discontinuing chronic opioid administration, total withdrawal and naltrexone induction time has been shortened to six days with little loss in success rate (Charney, Heninger, & Kleber, 1986).

OTHER NEW USES

Clonidine has been tried with varying success in a number of medical problems where the behaviors, signs, and/or symptoms resemble those seen in opiate withdrawal or following LC electrical or chemical stimulation to a certain degree. Clonidine has also been tried in humans on the basis of noradrenergic hyperactivity in generalized and panic ANXIETY; obsessive-compulsive symptomatology; Gilles de La Tourette's syndrome; mania; ATTENTION DEFICIT DISORDER; narcolepsy; neuroleptic-induced akathisia; ALCOHOL withdrawal and NICOTINE withdrawal; and phaeochromocytoma. Clonidine's ANALGESIC effects have been rediscovered and given orally, transdermally (skin patches), epidurally (into the area around the spinal canal), and parenterally (by injection)—to decrease anesthetic requirements and to effect less respiratory depression than OPIOIDS alone.

CONCLUSION

Using clonidine for withdrawal distress allows the brain to reestablish normal homeostatic patterns when given as part of a long-term recovery program. It allows patients sufficient motivation to achieve and sustain drug-free existence.

(SEE ALSO: *Opioid Complications and Withdrawal*)

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MARK S. GOLD

CLUB DRUGS Club drugs is a term that encompasses those drugs commonly abused within the context of the club and rave scenes which have developed over the past decade in the United States and Europe. Club drugs are a diverse group in terms of pharmacology, psychological effect, and toxicity. They form a unified grouping because of the context in which they are used, the clubs and raves that define turn-of-the-century youth culture. Because of the diversity and pleasure seeking inherent to the club world, no list of club drugs can pretend to be comprehensive but most lists include drugs like MDMA, GHB, KETAMINE, ROHYPNOL, METHAMPHETAMINE and LSD. Most of these drugs are perceived by users as relatively benign compared to "older" drugs like COCAINE. As might be expected, this perception is often not borne out in reality.

Many observers of the history of drug use in the United States have noted the cyclical nature of patterns of drug use. Few today recall that the cocaine epidemic of the 1980s was in fact the second cocaine epidemic in this century, the previous one having ended in the 1930s. Indeed, the lack of a cultural memory of the lessons of the previous epidemic no doubt played a role in the reemergence of the belief that cocaine was a "safe drug" in the 1960s and 70s, a belief that had been popular in the early 1900's until certain less palatable realities began to sink in. Club drugs seem to fit this pattern insofar as the lack of direct experience with the negative consequences of their use (MDMA for instance was not recreationally used before the 1980s) imparts the belief that they are a safe means of entertainment. History suggests that such a view is unlikely to stand the test of time.

MDMA is new as psychoactive drugs go; it only emerged as a recreational drug in the mid-1980s. It is an AMPHETAMINE derived HALLUCINOGEN, sometimes described as an empathogen or entactogen due to the enhanced feelings of emotional and physical closeness to others it generates in many users. Although it has a reputation as a benign

"love drug," MDMA has contributed to hundreds of deaths in its short time as an abused drug. It has been linked to seizures as well as kidney and cardiovascular failure. MDMA has produced long-term neurotoxicity in animals and a number of frequent human users have exhibited cognitive and emotional deficits.

Both rohypnol and GHB have gained notoriety as "date-rape" drugs due to their criminally abused propensity for impairing memory and inducing unconsciousness. Again, both are fairly new to the world of recreational drug use, although rohypnol belongs to the same class of drugs, the BENZODIAZEPINES, as VALIUM, a drug with a well known history of abuse. These drugs are especially dangerous when used with ALCOHOL, which exacerbates their depressant effects often leading to stupor, respiratory depression, and in some cases coma and death. Like alcohol, GHB and rohypnol seem to cause an increase in violent behavior in some users. These drugs have been linked to such a disproportionate number of negative events that many countries have opted to increase restrictions on their use.

Methamphetamine is an exception to the rule that club drugs are new; it has a long and well-documented history of abuse and toxic effects. Its appearance on the club scene seems to be linked to its low cost and the present negative perception of cocaine as an alternative PSYCHOSTIMULANT. Methamphetamine is substantially more toxic to the brain and liver than cocaine, while sharing some of cocaine's potentially lethal effects on the cardiovascular system. Amphetamine use has also been linked to toxic psychosis.

Ketamine is a dissociative anesthetic formerly used in humans but now largely restricted to veterinary use. Ketamine shares its major site of action with PHENCYCLIDINE (PCP) and, like the latter drug, can produce many of the symptoms of psychosis in humans including hallucinations and indifference to pain or death. Given that chronic PCP use has been associated with the development of long-term psychosis, it seems likely that this may prove to be a risk with ketamine as well. Ketamine is thought to have few other toxic effects.

LSD is another drug with a well-known history of misuse and abuse. Its major dangers lie in its hallucinogenic properties, which may cause users to physically harm themselves or others. LSD also seems to aggravate depression and psychosis. Outside of its intense psychological effects LSD has few, if any, physiological side effects even when taken at doses well in excess of those used recreationally.

Club drugs are hardly risk-free, and the next decade or so will probably provide the public with more evidence of their dangers. A particularly risky and difficult-to-analyze aspect of the club drug phenomena is that most club drug users use several of these drugs as well as TOBACCO and alcohol. With such a variety of drugs being abused by individual users, toxic and other dangerous results are far more likely to occur and less predictable in terms of long-term consequences.

RICHARD G. HUNTER

COCA-COLA *See* Cola/Cola Drinks

COCA PASTE Coca paste is the first crude extraction product of coca leaves from the COCA PLANT; it is obtained in the process of extracting COCAINE from these leaves. The leaves are mashed with alkali and kerosene and then sulfuric acid (and sometimes also potassium permanganate). The result is an off-white or light-brown paste containing 40 to 70 percent cocaine, as well as other ALKALOIDS, benzoic acid, kerosene residue, and sulfuric acid (ElSohly, Brenneisen, & Jones, 1991). Peruvian and Bolivian paste is illegally exported to Ecuador or Colombia, where it is purified into cocaine hydrochloride and then illicitly shipped to markets throughout the world. Although cocaine is the major component of coca paste, the paste is chemically complex, reflecting additives used by the clandestine laboratories performing the extraction from the coca leaves.

Coca paste, also called cocaine paste or pasta, is smoked, primarily in Latin American countries, by mixing about 0.2 ounces (0.5 g) of it with TOBACCO (called "tabacazo") or with MARIJUANA (called "mixto") in a cigarette. When this dab of coca paste is smoked with tobacco, only 6 percent of the cocaine reaches the smoker—but since most the paste samples contain significant amounts of manganese as well as several gasoline residues, the inhaled condensate is an extremely toxic substance. Despite the low bioavailability of cocaine from coca paste when it is smoked, use of this illegal sub-

stance by the smoking route reached epidemic proportions in Latin America in the late 1970s. More recently, coca paste smoking has been reported in the NETHERLANDS, the Antilles, Panama, and the United States, although the level of use remains very low.

The effects of coca-paste smoking have been reported to be as toxic as those seen after intravenous or smoked cocaine (i.e., CRACK) in the United States. In fact, coca-paste smokers can achieve cocaine blood levels comparable to those seen in users injecting or smoking cocaine (Paly et al., 1980). Smoking the paste leads to an almost immediate euphoric response, and users smoke it repeatedly. As with smoking cocaine (FREEBASING), large quantities of the paste are taken repeatedly within a single smoking session, which is terminated only when the drug supply is depleted. Users report a dysphoric response (unease, illness) within about thirty minutes after smoking, so more paste is generally smoked at this time if available.

Substantial toxicity has been reported for chronic use of the coca-paste—tobacco combination, with users smoking it repeatedly, and progressing from stimulant-related effects and euphoria to HALLUCINATIONS and paranoid psychoses. In fact, studies carried out in Peru defined a mental disorder of coca-paste smoking, made up of four distinct phases—euphoria, dysphoria, hallucinosis, and paranoid psychosis (Jeri et al., 1980). Since substantial amounts of paste are smoked at one time, the paranoid psychosis seen after chronic stimulant use has also been reported for paste use. As with cocaine abusers, experienced users of coca paste usually turn to criminal activities to support their illicit drug use.

(SEE ALSO: *Bolivia, Drug Use in; Complications; Crime and Drugs; Pharmacokinetics; Psychomotor Stimulants*)

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COCA PLANT The coca plant is a cultivated shrub, generally found in the Andean Highlands and the northwestern areas of the Amazon in South America. The plant, however, can be grown in many parts of the world and in the early part of the twentieth century much of the cocaine used in medicine was obtained from plants grown in Asia. Of the more than 200 species of the genus *Erythroxylon*, only *E. coca* variety *ipadu*, *E. novogranatense*, and *E. novogranatense* variety *truxillense* contain significant amounts of COCAINE, ranging from 0.6 to 0.8 percent. In addition to cocaine, the leaves of the coca plant contain eleven other ALKALOIDS, although no others are extracted for their euphorogenic effects.

Coca plants have long histories of use for both their medicinal and stimulant effects. Coca leaves are believed to have been used for well over a millenium, since archeological evidence from Peruvian burial sites of the 6th century A.D. suggests coca use. In fact, ancient Indian legends describe its origin and supernatural powers. The Inca called the coca plant a "gift of the Sun God," and attributed to it many magical functions. The Inca and the other civilizations of the Andes used coca leaves for social ceremonies, religious rites, and medicinal purposes. Because of their energizing property, coca leaves were also used by soldiers during military campaigns or by messengers who traveled long distances in the mountains. Under the Spanish conquest of the sixteenth century, coca plants were systematically cultivated and the custom of chewing coca leaves or drinking coca tea was widely adopted as part of the Indian's daily life in South America. Use of coca leaves as both a medicinal and a psychoactive substance continues to be an integral part of the daily life of the Indians living in the Andean highlands. Substantial societal controls have existed concerning the use of these leaves, and minimal problematic behavior related to use of the coca leaves has been reported.

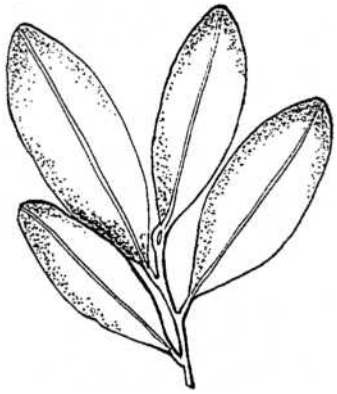

Figure 1
Coca Leaf

In the highland areas of Peru and Bolivia, and less frequently, in Ecuador and Colombia, the dried leaves are mixed with lime or ash (called "tocra") and both chewed and sucked. A wad containing 0.4 to 1 ounce (10 to 30 g) of leaf is formed, and daily consumption by coca-leaf chewers is between 1 and 2 ounces (30 and 60 g). The Indian populations in the Amazonean areas, however, crush the dried leaves, mix the powder with an alkaline substance, and chew it. Coca leaves are chewed today in much the same way that they were chewed hundreds of years ago.

Substantial cocaine plasma levels can be attained when coca leaves are chewed along with an alkaline substance, which increases the bioavailability of the drug by changing its pH. Volunteers allowed to chew either the leaf or the powdered form of coca mixed with an alkaline substance reported numbing in the mouth and a generally stimulating effect which lasted an average of approximately an hour after the coca chewing was begun (Holmstedt et al., 1979). This time-course corresponded to the ascending limb of the cocaine plasma-level curve, suggesting that the effect was cocaine-induced. Absorption of cocaine occurs from the buccal mucosa (inner cheek wall) as well as from the gastrointestinal tract after saliva-containing coca juice is swallowed. In fact, plasma concentrations in coca chewers are about what would be predicted if a dose of cocaine equivalent to that in the leaves was administered in a capsule (Paly et al., 1980).

(SEE ALSO: *Bolivia, Drug Use in; Coca Paste; Colombia As Drug Source*)

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MARIAN W. FISCHMAN

COCAETHYLENE: IMMUNOLOGIC, HEPATIC, AND CARDIAC EFFECTS Concomitant cocaine and ethanol use produce the compound cocaethylene. A 1995 study estimated that 60 to 80 percent of cocaine users consume ethanol simultaneously. Some users of cocaine mix it with ethanol together as they extend the euphoric sensation and lessen the dysphoria associated with a cessation of cocaine. Cocaethylene, a compound synthesized in vivo, was only identified in 1979. It also has been named in literature as ethylcocaine, ethylbenzoylecgonine, and benzoylecgonine ethyl ester. In 1990, an NIAAA Survey reported that 5.3 million Americans had used cocaine concurrently (during the same period of time) with alcohol, and 4.6 million simultaneously (on the same occasion) with ethanol.

Although the mechanism by which the combination of cocaine and ethanol may be particularly deleterious to the cardiovascular system is unknown, two hypotheses have been proposed:

(1) It may markedly increase the determinants of myocardial oxygen demand and simultaneously diminish supply, leading to a marked supply:demand imbalance; in human volunteers, the use of both drugs produces a greater increase in heart rate than either substance alone.
(2) The concomitant ingestion of cocaine and ethanol may lead to the production of a metabolite which induces marked coronary arterial vasoconstriction, leading to myocardial ischemia, infarction, and/or sudden death.

While formed when cocaine and ethanol were consumed simultaneously in humans, monkeys, and mice, formation of cocaethylene resulted through transesterification of cocaine by hepatic carboxylesterases in the liver. Further studies must be done in species that resemble humans to deter-

mine the pathways and significance of the cocaine and ethanol combination.

The toxicity that results from combined cocaine and ethanol use is not due to enhanced sensitivity to alcohol in cocaine abusers. In rats cocaethylene exposure during the brain growth spurt period causes teratogenic effects slowing brain growth. Cocaethylene is a neuroteratogen as indicated by altered concentrations of catecholamines and indoleamines in developing brains. There was a region-specific alteration in neurotransmitter levels in response to six days of cocaethylene exposure. It also appears that cocaethylene is more similar to ethanol than cocaine in terms of neuroteratogeneis. Measured cocaine and cocaethylene concentration in postmortem human cerebral cortex and that combined use of cocaine and ethanol increased the risk of death 18-fold.

In the first primate study the effects of intravenously administered cocaine on extracellular dopamine in the primate was compared to the effects of cocaethylene. There are numerous biochemical and pharmacological differences between primates, rodents and dogs that make it important to study primates if immediate extensions to clinical research studies in humans are to be made. Both cocaethylene and cocaine are equipotent and were found to increase extracellular dopamine in the caudate nucleus. Cocaethylene retains activity similar to cocaine including inhibition of dopamine transporter. In most case studies, the potentiality of cocaine and cocaethylene seem to point to equal potency in in vitro experiments. The organs of 60 percent of the addicts seeking medical attention in emergency rooms, or examined postmortem specimens, contain cocaethylene.

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Albert D. Arvallo
Ronald Ross Watson

COCAINE The abuse of cocaine has become a major public-health problem in the United States since the 1970s. During that period it emerged from relative obscurity, described by experts as a harmless recreational drug with minimal toxicity. By the mid-1980s, cocaine use had increased substantially and its ability to lead to drug taking at levels that caused severe medical and psychological problems was obvious. Cocaine (also known as "coke," "snow," "lady," "Crack" and "ready rock"), is an Alkaloid with both local anesthetic and Psychomotor Stimulant properties. It is generally taken in binge cycles, with periods of hours to days in which users take the drug repeatedly, alternating with periods of days to weeks when no cocaine is used. Many users are recalcitrant to treatment, and the introduction of substantial criminal penalties associated with its possession and sale have not yet been effective in reducing its prevalence of heavy use. In fact, although occasional use of cocaine diminished somewhat by the early 1990s, heavier use did not.

HISTORY

Cocaine is extracted from the Coca Plant (*Erythroxylon coca*), a shrub now found mainly in the Andean highlands and the northwestern parts of the Amazon in South America. The history of coca plant use by the cultures and civilizations who lived in these areas (including the Inca) goes back more than a thousand years, with evidence of use found archeologically in their burial sites. The Inca called the plant a "gift of the Sun god" and believed that the leaf had supernatural powers. They used the leaves much as the highland Indians of South America do today. A wad of leaves, along with some ash, is placed in the mouth and both chewed and sucked. The ash helps in the extraction of the cocaine from the coca leaf—and the cocaine is efficiently absorbed through the mucous membranes of the mouth.

Cocaine is usually distributed as a white crystalline powder, usually cocaine hydrocholride, that is often diluted with a variety of substances—sugars such as lactose, inositol and mannitol, and local anesthetics such as lidocaine. (Drug Enforcement Administration)

During the height of the Inca Empire (11th–15th centuries) coca leaves were reserved for the nobility and for religious ceremonies, since it was believed that coca was of divine origin. With the conquest of the Inca Empire by the Spanish in the 1500s, coca use was banned. The Conquistadors soon discovered, however, that their Indian slaves worked harder and required less food if they were allowed to chew coca. The Catholic church began to cultivate coca plants, and in many cases the Indians were paid in coca leaves.

Although glowing reports of the stimulant effects of coca reached Europe, coca use did not achieve popularity. This was no doubt related to the fact that coca plants could not be grown in Europe and the active ingredient in the coca leaves did not survive the long ocean voyage from South America. After the isolation of cocaine from coca leaves by the German chemist Albert Niemann in 1860 and the subsequent purification of the drug, it became more popular. It was aided in this regard by commercial endeavors in which cocaine was combined with wine (e.g., Vin de Coca), products for which there appeared many enthusiastic and uncritical endorsements by notables of the time.

Both interest in and use of cocaine spread to the United States, where extracts of coca leaves were added to many patent medicines. Physicians began prescribing it for a variety of ills including dyspepsia, gastrointestinal disorders, headache, neuralgia, toothache, and more—and use increased dramatically. By the beginning of the twentieth century, cocaine's harmful effects were noted and caused a reassessment of its utility. As part of a broader regulatory effort, the U.S. government began to control its manufacture and sale. In 1914, the HARRISON NARCOTIC ACT forbade use of cocaine in over-the-counter medications and required the registration of those involved in the importation, manufacture, and sale of either coca or opium products. This had the effect of substantially reducing cocaine use in the United States, which remained relatively low until the late 1960s, when it moved into the spotlight once again.

MEDICAL UTILITY

Cocaine is a drug with both anesthetic and stimulant properties. Its local anesthetic and vasoconstriction effects remain its major medical use. The local anesthetic effect was established by Carl Koller in the mid-1880s, in experiments on the eye, but because it has been found to cause sloughing of the cornea, it is no longer used in eye surgery. Because it is the only local anesthetic capable of causing intense vasoconstriction, cocaine is beneficial in surgeries where shrinking of the mucous membranes and the associated increased visualization and decreased bleeding are necessary. Therefore, it remains useful for topical administration in the upper respiratory tract. When used in clinically appropriate doses, and with medical safeguards in place, cocaine appears to be a useful and safe local anesthetic.

PHARMACOKINETICS

Cocaine can be taken by a number of routes of administration—oral, intranasal, intravenous, and smoked. Although the effects of cocaine are similar no matter what the route, route clearly contributes to the likelihood that the drug will be abused. The likelihood that cocaine will be taken for nonmedical purposes is assumed to be related to the rate of increase in cocaine brain level (as measured by blood levels) associated with those routes that provide the largest and most rapid changes in brain level being associated with greater self-

administration. The oral route of administration, not a route used by cocaine abusers, is characterized by relatively slow absorption and peak levels that do not appear until approximately an hour after ingestion. Cocaine, however, is quickly absorbed from the nasal mucosa when it is inhaled into the nose as a powder (cocaine hydrochloride). Because of its local anesthetic properties, cocaine numbs or "freezes" the mucous membranes, a quality used by those purchasing the drug on the street to test for purity. When cocaine is used intranasally ("snorting"), cocaine blood levels, as well as subjective and physiological effects, peak at about 20 to 30 minutes, and reports of a "rush" are minimal. Intranasal users report that they are ready to take a second dose of the drug within 30 to 40 minutes after the first dose. Although this route was the most common way for people to use cocaine in the mid-1980s, it is not as efficient in getting the drug to the brain as either smoking or intravenous injection, and it has declined in popularity.

When taken intravenously, venous blood levels peak virtually immediately and subjects report a substantial, dose-related rush. This route was, until the mid-1980s, traditionally the choice of the experienced user, since it provided a rapid increase in brain levels of cocaine with a parallel increase in subjective effects. Blood levels of cocaine dissipate in parallel with subjective effects, and subjects report that they are ready for another intravenous dose within about 30 to 40 minutes. Users of intravenous cocaine are also more likely to combine their cocaine with HEROIN (e.g., a "speedball") than are users by other routes.

In the mid-1980s, smoked cocaine began to achieve popularity. FREEBASE, or "crack," is cocaine base, which is not destroyed at temperatures required to volatilize it. As with intravenous cocaine, blood levels peak almost immediately and, as with intravenous cocaine, a substantial rush ensues after smoking it. Users can prepare their own freebase from the powdered form they purchase on the street, or they can purchase it in the form of crack, or "ready-rock." The development of a smokable form of cocaine provided a more socially acceptable route of drug administration (both NICOTINE and MARIJUANA cigarettes provided the model for smoking cocaine), resulting in a drug that was both easy to use and highly toxic, since the route allowed for frequent repeated dosing with a readily available and relatively inexpensive drug. The use of intravenous cocaine, in contrast, was limited to those able to acquire the paraphernalia and willing to put a needle in a vein. The toxicity of the smoked route of administration is in part related to the fact that a potent dose of cocaine is available to anyone who can afford it.

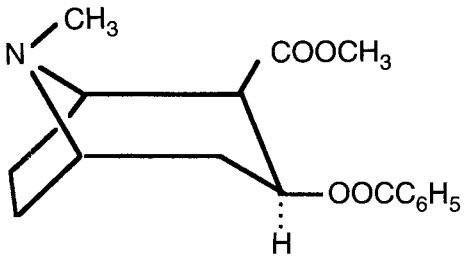

Figure 1
Chemical Structure of Cocaine

Cocaine is frequently taken in combination with other drugs such as alcohol, marijuana, and OPIATES. In fact, almost 75 percent of cocaine deaths reported in 1989 involved co-ingestion of other drugs. When taken in combination with alcohol, a metabolite—COCAETHYLENE—is formed, which appears to be only slightly less potent than cocaine in its behavioral effects. It is possible that some of the toxicity reported after relatively low doses of cocaine might well be due to the combination of cocaine and alcohol.

Cocaine is broken down rapidly by enzymes (esterases) in the blood and liver. The major metabolites of this action (all relatively inactive) are BENZOYLECGONINE, ecgonine, and ecgonine methyl ester, all of which are excreted in the urine. Cocaethylene is an additional metabolite when cocaine and alcohol are ingested in combination. People with deficient plasma cholinesterase activity—fetuses, infants, pregnant women, patients with liver disease, and the elderly—are all likely to be sensitive to cocaine and therefore at higher risk for adverse effects than are others.

PHARMACOLOGY

Research has been focused on the neurochemical and neuroanatomical substrates that mediate cocaine's reinforcing effects. Although a number of NEUROTRANSMITTER systems are involved, there is growing evidence that cocaine's effects on dopaminergic neurons in the mesolimbic and/or mesocortical neuronal systems of the brain are most closely associated with its reinforcing and other be-

havioral effects. The initial site of action in the brain for its reinforcing effects has been hypothesized to be the dopamine transporter of mesolimbocortical neurons. Cocaine action at the DOPAMINE transporter has the effect of inhibiting dopamine re-uptake, resulting in higher levels of dopamine at the synapse. These dopaminergic pathways may mediate the reinforcing effects of other stimulants and opiates as well. A substantial body of evidence suggests that dopamine plays a major role in mediating cocaine's reinforcing effects, although it is clear that cocaine affects not only the dopamine but also the SEROTONIN and noradrenaline systems.

TOXICITY

In addition to blocking the re-uptake of several neurotransmitters, cocaine use results in central nervous system stimulation and local anesthesia. This latter effect may be responsible for the neural and myocardial depression seen after taking large doses. Cocaine use has been implicated in a broad range of medical complications covering virtually every one of the body's organ systems. At low doses, cocaine causes increases in heart rate, blood pressure, respiration, and body temperature. There have been suggestions that cocaine's cardiovascular effects can interact with ongoing behavior, resulting in increased toxicity. Cocaine intoxication has been associated with cardiovascular toxicity, related to both its local anesthetic effects and its inhibition of neuronal uptake of catecholamines, including heart attacks, stroke, vasospasm, and cardiac arrhythmias.

Cocaine is generally taken in binges, repeatedly, for several hours or days, followed by a period in which none is taken. When taken repeatedly, chronic cocaine intoxication can cause a psychosis, characterized by paranoia, anxiety, a stereotyped repetitive behavior pattern, and vivid visual, auditory, and tactile hallucinations. Less severe behavioral reactions to repeated cocaine use include irritability, hypervigilance, paranoid thinking, hyperactivity, and eating and sleep disturbances. In addition, when a cocaine binge ceases, there appears to be a crash response, characterized by depression, fatigue, and eating and sleep disturbances. Initially, the crash is accompanied by little cocaine craving, but as time increases since the last dose of cocaine, compulsive drug seeking can occur in which users think of little else but the next dose.

BEHAVIORAL EFFECTS

Nonhuman Research Subjects. One of cocaine's characteristics, as a PSYCHOMOTOR STIMULANT, is its ability to elicit increases in the motor behavior of animals. Single low doses produce increases in exploration, locomotion, and grooming. With increasing doses, locomotor activity decreases and stereotyped behavior patterns emerge (continuous repetitious chains of behavior). When administered repeatedly, cocaine produces increased levels of locomotor activity, increases in stereotyped behavior, and increases in susceptibility to drug-induced seizures (i.e., "kindling"). This sensitization occurs in a number of different species and has been suggested as a model for psychosis or schizophrenia in humans. Although sensitization to cocaine's unconditioned behavioral effects generally occurs, such effects are related to dose, environmental context, and schedule of cocaine administration. For example, sensitization occurs more readily when dosing is intermittent rather than continuous and when dosing occurs in the same environment as testing.

Learned behaviors, typically generated in the laboratory using operant schedules of reinforcement in which animals make responses that have consequences (e.g., press a lever to get food), generally show a rate-dependent effect of cocaine. As with AMPHETAMINE, cocaine engenders increases in low rates of responding and decreases in high rates of responding. Environmental variables and behavioral context can modify this effect. For example, responding maintained by food delivery was decreased by doses of cocaine that either had no effect or increased comparable rates of responding maintained by shock avoidance. Cocaine's effects can also be modified by drug history. Although repeated administration can result in the development of sensitization to cocaine's effects on unlearned behaviors, repeated administration generally results in tolerance to cocaine's effects on schedule-controlled responding. This decrease in effect of the same dose after repeated dosing is influenced by behavioral as well as pharmacological factors.

Human Research Subjects. A major behavioral effect of cocaine in humans is its mood-

altering effect, generally believed related to its potential for abuse. Traditionally, subjective effects have provided the basis for classifying a substance as having abuse potential—and the cocaine-engendered profile of subjective effects is prototypic of stimulant drugs of abuse. Thus, cocaine produces dose-related reports of "high," "liking," and "euphoria"; increases in stimulant-related factors, such as increases on Vigor and Friendliness scale scores; ratings of "stimulated"; and decreases in various sedation scores. Subjective effects correlate well with single intravenous or smoked doses of cocaine, peaking soon after administration and dissipating in parallel with decreasing plasma concentrations. When cocaine is administered repeatedly, tolerance develops rapidly to many of its subjective effects and the same dose no longer exerts much of an effect. This means that the user must take increasingly larger amounts of cocaine to achieve the same effect. Tolerance to the cardiovascular effects of cocaine is less complete; the result here is a potential for drug-induced toxicity, since more and more drug is taken when the subjective effects are not present but the disruptions in cardiovascular function are still present.

Although users of stimulant drugs claim that their performance of many activities is improved by cocaine use, the data do not support their assertions. In general, cocaine has little effect on performance except under conditions in which performance has deteriorated from fatigue. Under those conditions, cocaine can bring it back to nonfatigue levels. This effect, however, is relatively short-lived, since cocaine has a half-life of less than one hour.

TREATMENT

Despite substantial efforts directed toward treatment of cocaine abuse, in the mid-1990s we are still unable to treat successfully many of the cocaine abusers who seek treatment. For many years the only approach to treating these people was psychological or behavioral. As of 1994, the most promising of these include behavioral therapy, relapse prevention, rehabilitation (e.g., vocational, educational, and social-skills training) and supportive psychotherapy. A major problem with these treatment approaches is related to their lack of selectivity. Rather than tailoring programs to an individual's background, drug-use history, psychiatric state, and socioeconomic level, individuals receive the treatment being delivered by the particular program they happen to attend. Treatment programs that focus on specific target populations will be far more successful than those which cover all who apply. For example, patients with relatively mild symptoms might do quite well in a behavioral intervention with some relapse-prevention instructions but those with more severe problems might require the addition of pharmacotherapy.

Pharmacological approaches to treating cocaine abusers have focused on potential neurophysiological changes related to chronic cocaine use. Thus, because dopamine appears to mediate cocaine's reinforcing effects, dopamine agonists such as AMANTADINE and bromocriptine have been tried. METHYLPHENIDATE, a stimulant, has been suggested as a possible substitution medication, and ANTIDEPRESSANTS such as desipramine have been studied because of their actions on the dopaminergic system. In addition, because cocaine blocks re-uptake of SEROTONIN at nerve terminals, serotonin-uptake blockers, such as fluoxetine, have also been tested. Although most of the potential medications have been shown to be successful in some patients under open label conditions, none have been clearly successful in double blind placebo-controlled clinical trials.

Clearly, no medication yet exists for the treatment of cocaine abuse. It may well be that different medications may be effective for the various target populations and that variations in dosages and durations of treatment might be required, depending on a variety of patient characteristics. In fact, several medications have been shown to be effective only for small and carefully delineated populations (e.g., lithium for cocaine abusers diagnosed with concurrent bipolar manic-depressive or cyclothymic disorders). An artificial enzyme has been developed that inactivates cocaine as soon as it enters the blood-stream by binding the cocaine and breaking it into two inactive metabolites, and this has the potential for destroying much of the cocaine before it reaches the brain. As of 1994, this technique is unavailable for human use. In addition, and most importantly, cocaine abuse (and drug abuse in general) is a behavioral problem, and it is unlikely that any medication will be effective unless it is combined with an appropriate behavioral intervention.

(SEE ALSO: *Cocaine, Treatment Strategies; Colombia As Drug Source; Epidemics of Drug Abuse; Epidemiology of Drug Abuse; National Household Survey on Drug Abuse; Treatment: Cocaine*)

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MARIAN W. FISCHMAN

COCAINE EPIDEMICS *See* Epidemics of Drug Abuse

CODEINE Codeine is a natural product found in the opium poppy (*Papaver somniferum*). An alkaloid of OPIUM, codeine can be separated from the other opium ALKALOIDS, purified, and used alone as an ANALGESIC (painkiller). It is however most often used along with mild nonopioid analgesics, such as aspirin, acetominophen, and ibuprofen. These combinations are particularly effective; the presence of the mild analgesics permits far lower codeine doses. Using lower doses of codeine has the advantage of reducing side effects, such as constipation. Codeine is one of the most widely used analgesics for mild to moderate pain.

Structurally, codeine is very similar to MORPHINE, differing only by the presence of a methoxy ($-OCH_3$) group at position 3, instead of morphine's hydroxy (-OH) group. The major advantage of codeine is its excellent activity when taken by mouth, unlike many opioid analgesics. Codeine itself has very low affinity for opioid receptors, yet it has significant analgesic potency. In the body, it is metabolized into morphine, and it is believed that the morphine generated from codeine is actually the active agent. Codeine has also been widely used as a cough suppressant. Codeine can be abused,

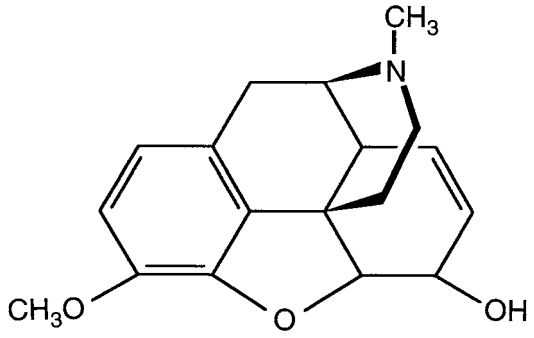

Figure 1
Codeine

and problems of abuse have often been linked to codeine-containing cough medicines, since they were once easily obtained over the counter. Chronic dosing with high codeine doses will produce TOLERANCE AND PHYSICAL DEPENDENCE, much like morphine.

(SEE ALSO: *Papaver somniferum*)

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GAVRIL W. PASTERNAK

CODEPENDENCE The term *codependence* replaced an earlier term, *coalcoholism*, in the early 1970s and achieved widespread acceptance among the general public during the 1980s. Both terms point to problematic beliefs and behaviors that family members of chemically dependent people tend to have in common, although the term codependence broadens the concept to cover a wider range of family dysfunctions than chemical dependence alone.

A rather large nonscientific literature has developed on the topic of codependence. Much of it is couched in terms of the need to deal with injuries to emotions sustained during childhood—that is, to heal the wounds of the "*inner child*," a term popularized by *John Bradshaw*.

Despite the current popularity of codependence, awareness that one person's alcoholism affects everyone in the family is not new. *The Big Book of*

Alcoholics Anonymous (1939; 1976) described the experience of family members of alcoholics in the following manner:

> We have had a long rendezvous with hurt pride, frustration, misunderstanding and fear. These are not pleasant companions. We have been driven to maudlin sympathy, to bitter resentment. Some of us veered from extreme to extreme, ever hoping that our loved one would be themselves once more.
>
> We have been unselfish and sacrificing. We have told innumerable lies to protect our pride and our husband's reputations. We have prayed, we have begged, we have been patient. We have struck out viciously. We have run away. We have been hysterical. We have been terror stricken. We have sought sympathy. We have had retaliatory love affairs with other men.
>
> Usually we did not leave. We stayed on and on [pp. 104–106].

In his book *I'll Quit Tomorrow* (1973), *Vernon Johnson* described the same experiences when he wrote that the *ism* of alcoholism is shared by other family members. In his words,

> While there may be only one alcoholic in a family, the whole family suffers from the alcoholism. For every harmfully dependent person, most often there are two, three, or even more people immediately around him who are just as surely victims of the disease. They too need real help and should be included in any thoroughgoing model of therapy. . . . With every drunk there is a sick dry who is almost a *mirror image*. [italics added]
>
> The people around the alcoholic person have predictable experiences that are psychologically damaging. As they meet failure after failure, their feelings of fear, frustration, shame, inadequacy, guilt, resentment, self-pity, and anger mount, and so do their defenses. They too use rationalization as a defense against these feelings because they are threatened with a growing sense of self-worthlessness. They too begin to project these masses of free-floating negative feelings about themselves upon the children, back on the spouse, on other family members, on employees, and everybody else at hand. Their defenses have begun to operate in the same way as the alcoholic's, although they are unconscious of this, and they are victimized by their own defenses rather than helped. Out of touch with reality, just like the alcoholic, they say, "*I* don't need help. It's his problem, not mine!" The chemically dependent and those around him all have impaired judgment; they differ only in the degree of impairment [p. 30].

DEFINITION

Although considerable debate still remains among professionals regarding the definition and meaning of codependence, most addiction specialists agree that the concept has successfully ushered huge numbers of people into recovery. Perhaps the best general definition of codependence is called the Scottsdale definition, after the conference location where several lecturers met to achieve consensus:

Co-dependence is a pattern of painful dependence on compulsive behaviors and on approval from others in an attempt to find safety, self-worth and identity.

CHARACTERISTICS

The following five characteristics form the common thread weaving through the lives of many, if not most, family members of alcoholics and other drug addicts:

1. *Codependents change who they are, and what they are feeling, to please others.* Codependents are chameleons who sacrifice their own identity in an effort to get others to love them. They do this for two reasons. First, they fear being abandoned if people know how they really feel or who they really are. Second, they have so little sense of who they are that they need to be in relationships in order to organize their lives and feel complete. Unless they are in a relationship, and can take their cues from another person, they feel desperately lonely and worthless. As a result, codependents are split between two worlds. One world is the facade they show other people—the false version of themselves. The other world is how chaotic, fearful, and empty their life feels underneath.
2. *Codependents feel responsible for meeting other peoples' needs, even at the expense of their own needs.* Codependents are so afraid of rejection that they will do anything to keep other people happy, including sacrificing their own needs to keep people from leaving them. They actually get more upset if others are disappointed or hurt than if their own problems go unsolved. This

habit of focusing more on others often leads to codependents' enabling a family member's drinking. *Enabling* means that the codependent protects the chemical dependent from the negative consequences of their drinking and other drug usage to keep the other person from having to feel any pain or embarrassment.

3. *Codependents have low self-esteem*. Most people who are chemically dependent feel ashamed of themselves and are inwardly very self-critical. So perhaps it is not strange that other family members also begin to feel bad about themselves. For codependents, low self-esteem comes from two main places:
   (a) It comes from having very little sense of self to esteem. By always pleasing others and taking their whole identity from others, codependents end up not knowing who they are apart from the relationships they are in. It's hard to respect people who are afraid to be themselves, even when it's you!
   (b) Low self-esteem also comes from believing that they truly are responsible for someone's alcohol/drug use. Once they believe this, they will always feel inadequate when they fail to control the chemical dependent's behavior. This mistaken sense of what should be under their control is at the very core of *both* codependence and chemical dependence.
4. *Codependents are driven by compulsions*. Codependents feel they do not have any real choices about what is happening to them. They typically feel *compelled* to keep the family together, to stop the drinking or other drug use, to save the family from shame, to work, to eat or diet, to take physical risks, to spend or gamble, to have affairs, to be religious, to keep the house clean, and on and on. The driven quality of compulsions accomplishes two things:
   (a) Compulsions create excitement and drama. As people battle their compulsions, the adrenaline begins to flow, and simple decisions, such as what to eat or how much to work, are turned into life and death struggles. This drama temporarily gives a feeling of purpose and vitality.
   (b) Compulsions also occupy a lot of time and block people from their deeper feelings. Codependents often get locked into compulsive behaviors to avoid more painful feelings of fear, sadness, anger, and abandonment caused by a family member's chemical dependence.
5. *Codependents have the same use of denial and distorted relationship to willpower that is typical of active alcoholics and other drug addicts*. Denial and an unwillingness to accept human limitations are the two most destructive parts of the *ism* of alcoholism described by Vernon Johnson. In their own way, codependent family members fall into the same distorted relationship to reality and willpower as the chemical dependent. Both deny reality and think they can control alcoholism (their own or another's) if they just use enough willpower. For example, if chemical dependents deny that they are abusing alcohol or other drugs and remain unaware of its impact on their lives and their relationships with family members, friends, and coworkers, then codependents show exactly the same denial. They often refuse to see that a family member is chemically dependent, or they refuse to acknowledge that their children are being hurt. Shame and the compulsion to keep things under control cause codependents to deny the problem. Denial is a universal human trait, but it is overused by every member of a chemically dependent family.

Codependents are driven by the firm belief that their coping strategies fail because of personal inadequacy. When they cannot control the drinking or other drug use of someone they love, they blame themselves for not trying hard enough—or for not trying the right way. When codependents take too much responsibility for another person's recovery, it keeps the chemical dependent from seeing that only they can be responsible for their own recovery.

PSYCHIATRIC PERSPECTIVE

In many ways, codependence is the mirror image of a chemical dependent's self-centeredness and grandiosity. Another term for such self-centeredness is *narcissism*. Codependence is the complement of narcissism, just as a glove complements the hand it is shaped to fit.

In the Greek's myth that gives us the prototype for self-centeredness, Narcissus had relationships only with people who shared his values and interests. He was unable to feel a sense of human connection with people who were separate from him, just as chemical dependents may break off relationships with people who do not support their denial.

The myth of Narcissus also gives us the prototype for *other-centeredness* in Echo—who is the perfect reflection of Narcissus. The two fit together and seemed to complete each other. Their relationship had intense chemistry.

In the eternal struggle within each individual between the need to be nurtured and the need to nurture others, Narcissus and Echo (and chemical dependents and codependents) strike a balance between two extreme positions. Rather than balancing the two needs within each of themselves, they allot the need to be validated and appreciated to Narcissus and the need to nurture and be in a relationship to Echo. Neither is capable of a truly mutual relationship—but, together, they create an intense experience of connectedness.

In healthy families, children remain comfortable with the competing, normal childhood needs to be unconditionally loved and validated as worthwhile (i.e., to be the center) and the opposite need to be completely dependent upon all powerful and good parents (i.e., to have others be the center). When parents are unable to tolerate not being the center of relationships, even with their children (which often happens with a chemically dependent parent), children often renounce their own need to be focused on. They become the opposite of narcissistic; they become codependent.

(SEE ALSO: *Adult Children of Alcoholics (ACOA); Al-Anon; Alateen; Families and Drug Use*)

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TIMMEN L. CERMAK

COERCED TREATMENT FOR SUBSTANCE OFFENDERS The logic for coerced treatment is that substance abusers have limited internal motivation and consequently need to be externally motivated to enter treatment in order to change their behaviors. Expected change includes reduced arrests, reduced crime, and no drug use. It is important to keep in mind that, from a criminal justice point of view, no drug use is expected, which is different from a public health harm reduction approach. Consequently, substance offenders who have limited internal motivation to change their behaviors are externally motivated to enter treatment using the authority of the criminal justice system. This authority includes probation, parole, diversion, and drug courts, which can include incentives for substance offenders like reduced sentences or decreased time under criminal justice supervision.

Coerced substance-abuse treatment has a traditional relationship with community treatment. The history of drug-abuse treatment in the United States can be traced to two U.S. Public Health Service farms at Lexington, Kentucky and Fort Worth, Texas, which were opened in the late 1930s. These facilities were established largely through the effort of James V. Bennett, former Director of the Federal Bureau of Prisons, when he recognized the need for treating drug abusers. Drug-abuse treatment at these farms, which were renamed hospitals, was designed primarily for federal prisoners, but volunteers without coercion could also receive treatment. However, after withdrawal from drugs, most volunteers did not stay, and with no community followup, there was a high relapse rate.

With these high relapse rates and using the California and New York civil commitment coerced treatment programs as models, the Narcotic Addict Rehabilitation Act (NARA PL 89-793) was passed by Congress in 1966 as a federal civil commitment program to reduce drug use. For NARA Titles I (treatment in lieu of prosecution) and III (treatment with no formal charges) court-ordered treatment was initially provided at the Lexington and Fort Worth hospitals after an evaluation period. Later, NARA inpatient treatment facilities were opened in several cities. These facilities served as the foundation for community-based drug abuse treatment. When NARA was phased out, the U.S. Public Health

Service facilities were transferred to the Federal Bureau of Prisons in the mid–1970s (Leukefeld & Tims, 1988).

Another milestone in coerced substance abuse treatment was establishment of the Treatment Alternatives to Street Crime (TASC) program, which is now called Treatment Accountability for Safer Communities. The Special Action Office created TASC in 1972 for Drug Abuse Prevention, an office with responsibilities similar to those of the current White House Office of National Drug Control Policy. TASC can be described as a diversion program and as case management that helps bridge community corrections and the drug-abuse treatment system. TASC provides identification, assessment, referral, case management and monitoring services for drug/alcohol dependent offenders accused or convicted of nonviolent crimes. TASC defuses some of the friction between community corrections and drug treatment services and is now operating in over 125 communities nationwide. Overall, TASC's effectiveness has been established in reducing drug abuse and keeping drug abusers in treatment for a longer period of time.

THE DRUG–CRIME RELATIONSHIP

Coerced treatment is considered within the context of the relationship between drugs and crime that has been well documented. For example, since the mid–1970s, both the National Institute on Drug Abuse and the National Institute of Justice have supported projects to understand the drug–crime connection, with findings that suggest that drug use enhances criminal careers. In fact, a survey of inmates in state and federal correctional facilities indicates that 83 percent of state prisoners reported previous drug use and 57 percent reported using a drug in the month before their offense (BJS, 1998). The Drug Use Forecasting (DUF) system, renamed ADAM (Arrestee Drug Abuse Monitoring Program) has consistently reported that 51 to 83 percent of male arrestees in major urban cities test positive for drugs (ADAM Annual Report, 1998). In fact, two-thirds of prisoners are drug abusers whereas over 60 percent of persons who come into contact with jails and lock-ups use a drug other than alcohol at the time of arrest (ONDCP, 1995).

CONTROL VERSUS TREATMENT

The relationship between treatment and control can cloud the overall perception of coerced treatment. Most community treatment providers perceive treatment and control as opposites with treatment on one side, as "the good guys," and control from the criminal justice system on the other side. In fact, many community treatment providers point to criminal justice authority as disruptive to the therapeutic relationship. However, this is largely refuted by the literature that indicates drug offenders under criminal justice authority generally remain in treatment longer and consequently have better treatment outcomes. In fact, criminal justice involved offenders remain in community treatment at least as long as others in treatment who are not criminal justice involved. There are other ways of thinking about treatment and control if the assumption is that interventions incorporate both treatment and control. For example, a therapeutic community/residential treatment facility is very high in treatment and control, whereas outpatient treatment is low in both treatment exposure and control unless a participant is involved in criminal justice supervision. Nevertheless, treatment and control are usually discussed as opposite processes, with this depending on ideology, perceived public interest, and political needs.

CONTROVERSIES

Coerced treatment and the use of court authority within the criminal justice system have not been without controversy, particularly since many community drug treatment providers believe that substance abusers should enter treatment voluntarily. As one early example of this controversy, Robert L. DuPont as Director of the National Institute on Drug Abuse, when addressing the Federal Bar Association in 1977, proposed setting up a trip wire, in the form of urine testing, that would identify heroin users who were on probation and parole. If an addicted probationer or parolee did not stop daily drug use, he or she would be referred for compulsory drug abuse treatment. And if treatment was refused or daily heroin use continued, the individual would be reincarcerated. Although the trip wire proposal was modified by other proponents, it never got underway because of the ensuing controversy. Controversy focused on three areas:

(1) the image problem created when a health agency proposed a mechanism for behavioral control using the criminal justice system,
(2) the violation of probationers' civil rights when tested, and
(3) the inadequacy of the urine testing technology.

In spite of the controversy, practitioners interested in the relationship between drugs and crime supported the concept because of the large number of crimes committed by substance abusers (Leukefeld, 1985).

COMMUNITY SUBSTANCE-ABUSE TREATMENT

Drug abusers in community treatment are involved with community corrections. They are frequently on diversion, probation, parole, or mandatory release. Early data from the Client Oriented Data Acquisition Process (CODAP) indicates that 17 percent of clients who entered drug-abuse treatment were on probation, parole, or mandatory release. By 1982, CODAP reported an increase in community corrections involvement for 27 percent of the males and 15 percent of the females. During the 1980s, Hubbard et al. (1989) found that over 30 percent of clients in residential and outpatient treatment were referred to treatment by the criminal justice system; this finding remains valid in the year 2000.

COERCED TREATMENT OUTCOMES

Drug treatment provided through the criminal justice system has had successes. As a result, coerced drug treatment, for example, has been separated into categories, including Civil Commitment (supervision of parolees with urine testing), Criminal Justice Authority (community corrections), urine testing, offender community treatment services (community drug abuse treatment) and treatment in prisons and jails. The research on drug treatment for drug offenders has grown. The interest in examining interventions comes from

(1) the decreased anti-rehabilitation atmosphere in the criminal justice system (Martinson, 1974);
(2) data which have shown promise including the Stay'n Out Program in New York (Wexler et al., 1992), the Cornerstone Program in Oregon (Field, 1985), and Key and Crest Programs in Delaware (Martin et al., 1999);
(3) the large number of chronic drug abusers who are incarcerated; and
(4) the need to understand interventions and retention for drug offenders and their related costs.

DRUG COURTS

The current interest in drug courts developed in response to the overlap between substance abuse and crime in order to provide treatment for defendants. The interest in drug courts increased recently with the expanded number of courts that grew to 275 jurisdictions in 1998 from the first drug court in Dade County, Florida, in 1989 (Belenko, 1998). The benefits of drug courts have been documented: reduced recidivism, decreased drug use, increased birth rates of drug-free babies, high program retention, increased relapse prevention, and cost efficient treatment. The drug court is a court-managed drug intervention and treatment program designed to provide a cost-effective alternative to traditional criminal case processing. Drug courts are treatment-oriented and target clients whose major problems stem from substance abuse. However, although there are standards that are required for each drug court program, each drug court program is different.

CHRONIC AND RELAPSING NATURE OF SUBSTANCE ABUSE

It is easy to forget that drug abuse can be chronic and relapsing. Without proper followup and treatment, substance abusers often return to drug use. It is no secret that recovery is a difficult process that is not completely understood, with or without coerced treatment. Intervention and treatment efforts need to focus on those factors that keep individuals drug free. These options can range from urine testing to methadone treatment. Nevertheless, many people believe that substance-abuse treatment does not work. They cite professional and/or personal experiences about individuals who immediately return to drug use during treatment and/or supervision. However, after discussion it becomes clear that the proper blend of treatment combined with followup supervision, relapse prevention, and self-help

groups like Alcoholics Anonymous was not used, and/or attendance was minimal.

Finally, there are no instant cures for substance abuse. Recovery for many can take a lifetime. Decreasing substance use during the course of an addict's life can combine the interventions of coerced treatment, community treatment, and possibly incarceration with twelve step groups. More emphasis needs to be placed on matching substance abusers with appropriate services as well as looking at ways to mix and match interventions. In addition, both external motivation—coerced treatment—and internal motivation that substance abusers bring to treatment at varying levels need to be better understood. Clearly, coercion can bring a substance offender to treatment, but it cannot be used to force a substance offender to participate in treatment.

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Revised by Carl Leukefeld

COFFEE Coffee is the world's most common source of caffeine, providing a little more than half of all caffeine consumed daily. In the United States, coffee is usually a beverage made by percolation or infusion from the roasted and ground or pounded seeds of the coffee tree (genus *Coffea*), a large evergreen shrub or small tree, which was native to Africa but now is grown widely in warm regions for commercial crops. Caffeine from coffee accounts for an estimated 125 milligrams of the 211 milligrams of U.S. caffeine consumed per capita per day. Recent estimates suggest that more than 50 percent of the adolescents and adults in the United States consume some type of coffee beverage. Coffee is one of the main natural commodities in international trade, ranking second only to petroleum in dollar value. Approximately fifty countries export coffee and virtually all of those countries rely on it as a major source of foreign exchange. An estimated 25 million people make their living in the production and distribution of coffee products.

In addition to caffeine, roasted coffee contains at least 610 other chemical substances, which may contribute to its smell, taste, and physiological effects. Nevertheless, coffee's primary psychoactive ingredient is caffeine. The amount of caffeine in an individual cup of coffee varies considerably, depending on the type and amount of coffee used, the form of the final coffee product (e.g., ground roasted or instant), and the method and length of brewing. On average, a 6-ounce (177 milliliters) cup of ground roasted coffee contains about 100 milligrams caffeine; the same amount of instant coffee typically contains about 70 milligrams caffeine. However, the caffeine content of any given 6-ounce cup of coffee can vary considerably and can reach as much as 210 milligrams. Drip coffee

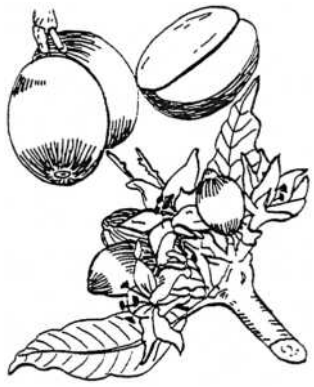

Figure 1
Coffee

typically contains more caffeine than percolated; decaffeinated coffee contains a small amount of caffeine, approximately 4 milligrams in a 6-ounce cup. Individual servings of caffeinated coffee contain amounts of caffeine that have been shown experimentally to produce a range of effects in humans including the alteration of mood and performance and the development of physical dependence with chronic daily use.

Coffee cultivation probably began around 600 A.D. in Ethiopia, but the drink was spread into the Middle East and Europe. Today, much of the world's coffee is grown in South and Central America, particularly Brazil and Colombia, and in several African countries. Coffee beverages derive primarily from the seeds of two species of *Coffea* plants, *Coffea arabica* and *Coffea canephora* var. *robusta*. Robusta coffees contain approximately twice as much caffeine as Arabicas. Arabica beans are used in the majority of the coffee consumed today, particularly in the higher quality coffees. Since processing for instant and decaffeinated coffee extracts flavor components from the bean, the stronger flavored beans, typically Robusta beans, are used for these coffee products. Caffeine extracted in the decaffeination process is sold for use in soft drinks and medications.

The coffee bean, covered with several layers of skin and pulp, occupies the center of the coffee berry. During the first part of coffee production, the outer layers of the coffee berry are removed, leaving a green coffee bean. The beans are then roasted, removing between 14 and 20 percent of their water and changing their color from green to various shades of brown; generally, the beans get darker as more water is extracted. The beans are then ground and ready for use. To produce instant coffee, roasted and ground coffee is percolated to produce an aqueous coffee extract. That extract is dehydrated by spray or freeze-drying to produce water-soluble coffee extract solids. Since this process removes flavor and aroma from the coffee, compounds are added to the extracts at the completion of the process to restore the lost characteristics.

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COGNITIVE-BEHAVIORAL THERAPY

Cognitive-behavioral treatments represent a group of approaches, grounded in social learning theories of substance abuse, that hold that lack of effective coping skills may be one factor underlying the development or perpetuation of substance use disorders. Cognitive behavioral treatments have been among the most well defined and rigorously studied of the psychosocial treatments for substance abuse and dependence, and have a comparatively high level of empirical support across the addictions. For example, in their review of cost and effectiveness data for treatments for alcohol use disorders, Holder and colleagues (1991) included social skills training, self-control training, stress management training, and the Community Reinforcement Approach (Azrin et al., 1976), all broad-spectrum CBT approaches, as having good empirical evidence of effectiveness. Recent meta-analyses (Irvin et al., 1999) and reviews of the effectiveness of treatments for substance abuse (APA Workgroup on Substance Use Disorders, 1996; DeRubeis & Crits-Christoph 1998) have identified this group of approaches as having among the highest level of empirical support for the treatment of substance use disorders.

OVERVIEW AND STRUCTURE OF CBT

Cognitive-behavioral treatments are typically highly structured in comparison to other approaches for substance use disorders. That is, these treatment approaches are typically comparatively brief (12-24 weeks) and organized closely around well-specified treatment goals. There is typically an articulated agenda for each session and discussion remains focused around issues directly related to substance use. Progress toward treatment goals is monitored closely and frequently, and the therapist takes an active stance throughout treatment.

Cognitive-behavioral approaches typically include a range of skills to foster or maintain abstinence and to prevent relapse. These typically include strategies for:

(1) reducing availability and exposure to the substance and related cues,
(2) fostering resolution to stop substance use through exploring positive and negative consequences of continued use,
(3) self-monitoring to identify high risk situations and to conduct functional analyses of substance use,
(4) recognition of conditioned craving and development of strategies for coping with craving,
(5) identification of seemingly irrelevant decisions which can culminate in high risk situations,
(6) preparation for emergencies and coping with a relapse to substance use,
(7) substance refusal skills, and
(8) identifying and confronting thoughts about the substance.

The techniques of teaching these coping responses include a combination of direct verbal instruction, modeling of appropriate skills through role play, and rehearsal of the skills within the therapy session (Marlatt & Gordon, 1985). Material discussed during sessions is typically supplemented with extra- session tasks (i.e., homework) intended to foster practice and mastery of coping skills.

Broad-spectrum cognitive-behavioral approaches such as that described by Monti and colleagues (1989), and adapted for use in Project MATCH (Kadden et al., 1992), expand to include interventions directed to other problems in the individual's life that are seen as functionally related to substance use. These may include general problem-solving skills, assertiveness training, strategies for coping with negative affect, awareness of anger and anger management, coping with criticism, increasing pleasant activities, enhancing social support networks, job seeking skills, and so on.

There are a variety of manuals available (Monti et al.,1989; Kadden et al. 1992, Carroll, 1998) which describe key CBT strategies and techniques, as well as guidelines for its implementation with a variety of types of substance users. The classic resource in this area remains the Marlatt and Gordon's (1985) landmark book on relapse prevention.

The goals of cognitive-behavioral treatments tend to be somewhat broader than those of 'strict' behavioral approaches, and the choice of treatment goals will dictate the specific interventions implemented. For example, in broad spectrum cognitive-behavioral treatments (e.g., Azrin et al., 1976; Monti et al., 1989), the patient and therapist may select a wide range of target behaviors in addition to a treatment goal of abstinence, including improved social skills or social functioning, reduced psychiatric symptoms, and reduced social isolation, entry into the work force. Cognitive behavioral therapy also differs from cognitive therapy through greater emphasis on building specific behavioral skills (e.g., coping with craving, avoiding high risk situations, understanding behavioral patterns) and somewhat lesser emphasis on targeting and challenging maladaptive cognitions in the earlier stages of abstinence.

STRENGTHS AND WEAKNESSES

Strengths of cognitive-behavioral approaches have been summarized by Rotgers (1996) and include:

(1) flexibility in meeting individual needs,
(2) acceptability to a wide range of substance-abusing individuals seen in clinical settings,
(3) solid grounding in established principles of behavior theory and behavior change,
(4) an emphasis on linking science to treatment,
(5) well-specified treatment goals and clear guidelines for assessing treatment progress,
(6) emphasis on building self-efficacy, and
(7) a comparatively strong level of empirical support.

These approaches are highly flexible, and can be used in a number of treatment modalities and settings, can be applied across different types of sub-

stance use with minor modifications, and are compatible with a wide range of other treatment approaches, including family therapy and pharmacotherapy. Another advantage is that these approaches have emphasized clear specification of treatment and a variety of manuals are available, thus allowing a high level of technology transfer. Disadvantages of this group of approaches include:

(1) research evaluating these approaches have tended not to emphasize the importance of isolating and evaluating the specific 'active ingredients' associated with behavior change,
(2) comparative underutilization of these approaches outside of academic treatment settings (Rotgers, 1996), and
(3) lack of emphasis on patient motivation and specific procedures for addressing the patient's readiness for change.

SUMMARY

Cognitive behavioral treatments have emerged in the last decade as a leading approach to the treatment of substance use disorders. Solidly grounded in well-established principles of behavior change, with strong empirical support, and applicable to a wide range of individuals with substance use disorders, these well-defined approaches should be a part of any clinician's treatment repertoire.

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COGNITIVE THERAPY OF ADDICTIONS *See* Treatment Types

COLA/COLA DRINKS Cola drinks are carbonated soft drinks, sodas, that contain some extract of the kola nut in their syrup. Kola nuts are the chestnut-sized and -colored seeds of the African kola tree (*Cola nitida* or *Cola acuminata*). For the softdrink industry, the trees are now grown on plantations throughout the tropics. Historically, kola nuts were valued highly among African societies for their stimulating properties. Kola nuts were

Figure 1
Kola

cracked into small pieces and chewed for the effect—which increased energy and elevated mood in extremes of heat, hunger, exhaustion, and the like. The European colonists in Africa learned of the effect and some chewed it. In the 1800s, Europeans brought kola nuts to various strenuous endeavors in Africa and in other regions, and they began to increase the areas under cultivation. Kola nuts were soon finely powdered and made into syrups for ease of use—with no loss of effect, it was claimed.

The active ingredient responsible for these stimulatory properties is CAFFEINE, a powerful brain stimulant, which is also present in other plants such as COFFEE, cocoa, TEA, maté, and others. Besides reversing drowsiness and fatigue, a heightened awareness of stimuli and surroundings may occur. Studies have shown that less energy may be expended by the musculature with equal or greater results—in animals as well as humans—but excess use causes TOLERANCE and dependence, often unrealized until deprivation results in severe headaches. Large doses can cause nervous irritation, shaking, sleep disturbances, insomnia, and aggravation of stomach ulcers or high blood pressure.

In the late 1800s, in the United States, cola drinks came onto the market with other carbonated or phosphated (fizzy) drinks. Coca-cola™, one of the first and most popular, contained extracts of both the COCA PLANT (cocaine) and the kola nut (caffeine)—but by the early 1900s, with the realization of COCAINE's dangers, this was removed and replaced by additional caffeine. Drinking cola is part of American culture, emulated and enjoyed worldwide, with many brands competing for a huge and growing consumer market. Colas are now available with sugar or artificial sweeteners, with or without caffeine, with or without caramel coloring (clear)—thus indicating that people seem to like the flavor regardless of the specific ingredients or the "effect."

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COLLEGE ON PROBLEMS OF DRUG DEPENDENCE (CPDD), INC. The College on Problems of Drug Dependence (Martin W. Adler, Ph.D., Executive Officer, CPDD, Department of Pharmacology, 3420 N. Broad Street, Philadelphia, PA 19140; 215-707-3242; http://views.vcu.edu/cpdd/) is the nation's oldest organization devoted to the problem of drug use and addiction. It is an incorporated, not-for-profit, scientific organization that acts independently of both the U.S. government and the pharmaceutical industry while fostering an exchange of knowledge and resources across the academic, medical, governmental, and business communities. The CPDD is known internationally as a World Health Organization Collaborating Center for research and training in the field of drug dependence. The CPDD also offers consulting services and, along with the National Institute on Drug Abuse, supports drug-dependence testing and research at several select U.S. universities.

Among the goals of the CPDD are the following:

(1) to support, promote, and carry out ABUSE-LIABILITY research and testing, both at the preclinical and clinical levels;
(2) to serve as advisor to both the public and private sectors, nationally and internationally;
(3) to sponsor an annual scientific meeting in fields related to drug abuse and chemical dependence.

The annual scientific meeting of the CPDD has become one of the few forums where scientists from diverse disciplines can discuss problems of drug

abuse and drug dependence at a rigorous academic and scientific level.

A primary goal of the CPDD is the publication of data on the physical-dependence potential and abuse liability of OPIOIDS, stimulants, and depressants, as well as the development of a new methodology for drug evaluation. These data provide an independent scientific evaluation of drugs that might have abuse liability. A number of scientists from various medical schools work collaboratively to assess these drugs. The data are collated in the Laboratory of Medicinal Chemistry, National Institute of Diabetes, Digestive and Kidney Diseases (NIDDK), National Institutes of Health (NIH), Bethesda, MD. They are discussed by the Drug Evaluation Committee of the CPDD before publication. Government agencies can use the data to help determine whether a medically useful drug should be scheduled under the CONTROLLED SUBSTANCES ACT to restrict access and thus reduce possible abuse.

The contemporary CPDD originated in 1913, as the Committee on Drug Addiction of the Bureau of Social Hygiene in New York City. In 1928, the Bureau of Social Hygiene provided funds to the Division of Medical Sciences, National Research Council (NRC), of the National Academy of Sciences (NAS), for the support of a chemical, pharmacological, and clinical investigation of narcotic drugs by the Committee on Drug Addiction, NRC, NAS. This research continued until World War II. From 1939 to 1947, the Committee on Drug Addiction served as an advisory group to the U.S. Public Health Service (Eddy, 1973).

The Committee on Drug Addiction was reestablished in 1947 as the Committee on Drug Addiction and Narcotics (CDAN), in the Division of Medical Sciences of the NRC, NAS. In 1965, CDAN's name was changed to the Committee on Problems of Drug Dependence (CPDD). The CPDD remained as an NRC, NAS committee until 1976, when it became an independent scientific organization, the Committee on Problems of Drug Dependence (CPDD), Inc. It was guided by a Board of Directors with the sponsorship of nine major scientific organizations (May & Jacobson, 1989). In 1991, the CPDD underwent its most recent reorganization and its name was modified to reflect its contemporary role. Now known as the College on Problems of Drug Dependence, Inc., the CPDD has become a scientific membership organization that enables its members to have a voice on issues relating to drug abuse. Sixteen institutions and professional and scientific societies are affiliated with or have liaison representation with the CPDD, including such diverse groups as the American Chemical Society, the American Medical Association, and the Food and Drug Administration.

The members of the CPDD are involved in all the aspects of the effects of drugs subject to abuse—encompassing the enormous range from social, economic, and political issues through basic research in molecular biology and the study of the interaction of these drugs with specific RECEPTORS in the central nervous system. Membership is divided into four categories: Fellows, Regular Members, Associate Members, and Student Members. In addition, corporations with an interest in the field may join as Corporate Members. The CPDD sponsors the publication of the monthly journal, *Drug and Alcohol Dependence*, an international journal covering the scientific, epidemiological, sociological, economic, and political aspects of substance abuse.

(SEE ALSO: *Drug Types; World Health Organization Expert Committee on Drug Dependence*)

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REVISED BY NANCY FAERBER

COLOMBIA AS DRUG SOURCE Smuggling and the commerce in contraband have been a way of life in Colombia for nearly 500 years. Approximately 1,000 miles (1609 km) of largely unpatrolled Pacific and Caribbean coastline and vast tracts of mostly uninhabitable territory—ranging from tropical jungles in the south, to rugged Andean mountain slopes in the east, to sparsely populated deserts in the north—have made Colombia a haven for smugglers of illegal COCAINE, MARIJUANA, and, most recently, HEROIN. Violence, cor-

ruption, inadequate control by the central government over much of its territory, and an ineffective judicial system have hampered Colombia's drug-control efforts. Consequently, Colombian cocaine is the single largest supply of that illicit drug to be smuggled into the United States.

During the 1980s, despite positive law enforcement and crop-control programs, Colombian laboratories processed large volumes of (COCA PLANT) (*Erythroxylon coca*) into cocaine; by the 1990s, their sophisticated trafficking infrastructure had diversified into heroin production and distribution, adding to the already large Asian and Mexican supply in the United States. To reduce the level of violence and achieve peaceful coexistence throughout the country, the Colombian government offered a type of amnesty, or plea bargain, to major drug traffickers willing to surrender and cease their trafficking operations. However, by the late 1990s, the government had shifted its approach, employing the military and law enforcement to attack narcotics cultivation, processing, and trafficking.

Colombia has powerful, often violent, trafficking organizations based in the major urban areas of Medellin and Cali, as well as the oldest continuous political insurgency in the Western Hemisphere, the Revolutionary Armed Forces of Colombia (FARC). Although the extradition treaty with the United States is no longer constitutional, the Colombian government has implemented the strongest drug law enforcement efforts of any Andean country. In the 1990s, Colombia began efforts to eradicate its burgeoning crop of opium poppy (*Papaver somniferum*), but met with little or no success. It also attempted to enhance the investigation of drug crimes through the development of a new code of criminal procedure; tougher chemical control; anti-money-laundering, asset-seizure, and evidence-sharing procedures.

Colombia was a signatory to the 1961 SINGLE CONVENTION ON NARCOTIC DRUGS and the 1971 Convention on Psychotropic Substances. It has signed but not yet ratified the 1988 U.N. Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances. Although money laundering is not illegal in Colombia, the government is actively tracking narcotics proceeds (e.g., exchanging tax information and writing tougher financial disclosure laws) to improve its drug-investigative capacity.

By 1991, cocaine cartels in Colombia had diversified into the more lucrative heroin trade by cultivating opium poppies on the slopes of the Andes. (Drug Enforcement Administration)

ROLE AS COCAINE SUPPLIER

Proximity to the U.S. marketplace, remote and vast tracts of unpatrollable land, powerful criminal organizations, indigenous entrepreneurial spirit, and a long tradition of violence and smuggling make Colombia an ideal source for illegal drugs. Coca production rose dramatically in the 1990s as the governments of Bolivia and Peru pursed aggressive eradication programs. The U.S. government estimated that Colombian coca cultivation doubled between 1995 and 1999, constituting over two-thirds of the production in South America. Cultivation takes place in the Llanos (plains) region, which encompasses almost half of eastern Colombia. Heavy growth also occurs in the Caqueta, Guaviare, Putumayo, and Vaupes departments (counties or provinces), with evidence of crop expansion in the Bolivar department and in south and southwest Colombia. In addition, new, more potent strains of coca have been developed that reach maturity more quickly and yield more product in the cocaine conversion process.

Colombia's importance to the U.S. government's international narcotics control strategy lies in its role as the world's leading processor and distributor of cocaine hydrochloride (HCl)—the cocaine salt (powder) that is sniffed or snorted—and cocaine base, or CRACK cocaine—the rock or crystal that has been converted from cocaine HCl. Colombian cocaine-trafficking organizations are sophisticated and well-organized industries; they derive their strength from longtime control of cocaine laboratories and the smuggling routes to North America. Colombian traffickers operate a large number of base and HCl labs—ranging from small simple operations to large sophisticated complexes.

HISTORICAL AND INSTITUTIONAL FACTORS

In the mid-to-late 1970s, the United States directed its international drug-control attention to eliminating the heroin and marijuana crossing our border at Mexico. As the U.S.-Mexican crackdown began to achieve positive results and the number of U.S. smokers of Mexican marijuana diminished, Colombian traffickers seized the opportunity to break into the lucrative U.S. drug market by smuggling large amounts of marijuana and small packages of cocaine. In the early 1980s, Florida became the destination of choice for smugglers because of its long coastlines, access to boats and planes, location in the Caribbean, and large Hispanic population; by 1986, Colombia supplied an estimated 80 percent of the cocaine HCl.

More than 150 Colombian drug groups organized loosely into autonomous business cartels in the two principal urban areas of Colombia—Medellin and Cali—represent one of the most important power centers. The government of Colombia and the roughly ten thousand members of the two antigovernment guerrilla groups called the Revolutionary Armed Forces of Colombia (FARC) and the National Liberation Army (ELN) are also political forces there (Lee, 1989).

In controlling all stages of cocaine production, from cultivation to sale, Colombian traffickers have developed sometimes competitive, sometimes symbiotic, relationships with each other and with insurgent groups. The guerrillas benefit from the illicit drug trade by providing protection for the coca fields, laboratories, and storage facilities; they carry out kidnapping and terrorism to support the traffickers' aims. In return, the political insurgents "tax" the profits of the drug trade, thereby earning hard currency and occasionally extracting payment in weapons.

Elected in 1982, Colombia's President Belisario Betancur appointed a strong counternarcotics minister of justice, Rodrigo Lara Bonilla. In 1984, Lara Bonilla ordered a police raid on a Medellin cartel laboratory complex in a remote area called Tranquilandia, which produced more than 3 tons of cocaine per month. The police seized some 15 tons (13.8 metric tons) of cocaine, several airplanes, a variety of arms, and chemicals essential to the processing of cocaine. One month later, the minister of justice was assassinated in the capital, Bogota. In retaliation, President Betancur signed the first extradition order for Carlos Lehder, the Medellin cartel's transportation czar. Lehder was extradited in 1987, convicted in a Florida federal court, and is serving a sentence of life plus 135 years in the United States.

Between 1984 and 1987, a total of fifteen drug traffickers were extradited to the United States. In 1984, 1988, and 1990, major traffickers met privately with senior Colombian politicians to discuss the possibility of a general amnesty and an end to extradition—in return for ceasing the violence, abandoning the cocaine business, and relinquishing the assets used in the industry (e.g., planes, laboratories, airstrips). Although the first three offers were rejected, in September 1990, the Gaviria administration gave the *Extraditables* the option of accepting reduced sentences and guarantees against extradition if they surrendered to the authorities, confessed their crimes, and offered up their assets.

Under President Vergilio Barco, 1986 to 1990, the violence throughout Colombia's countryside increased in response to the continuing extraditions. In the 1980s, the murders in Medellin, a city of two million inhabitants, increased ten-fold—from seven hundred in 1980 to seven thousand by 1991—due in part to a bounty of $300 offered by Medellin cartel boss Pablo Escobar in 1990 for every policeman killed. In August 1990, the populist politician Luis Carlos Galan, a presidential contender, was assassinated at a political rally because of his strong support for the extradition treaty and antidrug position. His murder mobilized the civilian population against the cartels and incensed Colombia's national police and military.

Through a series of raids on cartel laboratories, the government began an unparalleled crackdown, culminating in the shooting of Medellin strongman Rodrigo Gacha and the destruction of one of their largest cocaine-processing centers.

The Cali cartel, which was less violent and less obtrusive than its Medellin counterpart, quickly began to move into the position vacated by the Medellin leaders, many of whom were either imprisoned or killed. However, by 1996 the Colombian government had broken up the Cali cartel through the arrest or surrender of its leaders.

DRUG-REDUCTION EFFORTS

Among the principal Andean SOURCE COUNTRIES for coca, Colombia has become most committed to defeating the cocaine cartels, since they threaten to undermine its society. Colombians recognize and fear that the violence and corruption endemic to drug trafficking are harming their economy, political system, and society. By the late 1990s, the government sought international assistance to overcome the growing power of drug organizations.

Colombia's national police, the military, and the security forces successfully broke up the Medellin and Cali cartels. In 1989, with the assistance of U.S. technical and material support, they attacked and killed several mid-level Medellin traffickers, including Rodrigo Gacha. Colombia has also used the extradition to incarcerate or immobilize major traffickers. In late 1990, President Cesar Gaviria's offer of amnesty for major traffickers resulted in a decrease in violence and the surrender and imprisonment of five traffickers and one terrorist—including Pablo Escobar and the three Ochoa brothers (Jorge Luis, Juan David, and Fabio). The Medellin cartel lost its leader when Pablo Escobar was killed in a shoot-out in December 1993 after having escaped from prison. The Cali cartel met its demise in the mid–1990s.

As a signatory of the 1961, 1971, and 1988 U.N. International Narcotics Control Conventions, Colombia demonstrates its political commitment to immobilizing drug traffickers and eradicating coca, cannabis, and opium. In the early 1990s, the government created public-order courts and began to share evidence, reform its judiciary, and track substantial money flows—requiring banks to keep records on cash transactions of over 10,000 U.S. dollars.

Despite these promising efforts, the shift of coca production from Bolivia and Peru to Columbia in the 1990s placed civil society in jeopardy. In addition, the destruction of the Medellin and Cali cartels has led to the proliferation of smaller, decentralized drug-trafficking organizations. These traffickers are primarily located near Cali and on the Caribbean North Coast. In addition, these drug organizations have the support of well-armed and well-organized guerrillas and paramilitary groups.

President Andres Pastrana, elected in 1998, formed a counterdrug joint task force with elements from all the military services and the national police. Pastrana proposed a comprehensive, integrated strategy called "Plan Columbia" to address the country's drug-related, economic, and social troubles. At a cost of over $7 billion, the United States agreed to fund a large part of the plan, which includes a massive eradication program and the interdiction of air transportation. United States military forces provide technical assistance and the United States has also supplied helicopters and airplanes. In 2000, the U.S. Congress approved over $1 billion in funds toward continued eradication.

(SEE ALSO: *Crop-Control Policies; Drug Interdiction; Foreign Policy and Drugs; International Drug Supply Systems; Source Countries for Illicit Drugs*)

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COMMISSIONS ON DRUGS For hundreds of years, governments have been using commissions of inquiry to help them investigate pressing social problems and to formulate plans for organized social and governmental responses to these problems. One of the first government commissions on problems associated with drug use, appointed in 1893, was the Indian Hemp Drugs Commission—whose purpose was to investigate the extent to which the hemp plant was cultivated, the preparation of drugs from it, the trade in those drugs, the extent of their use, and the effects of their consumption on the social, physical, and moral conditions of the people. In addition, they were asked to investigate certain economic aspects of the use of hemp and also the potential political, social or religious results of the prohibition of hemp. After one year of investigation, the commission published a report summarizing its conclusion that the occasional use of hemp in moderate doses was not harmful but that excessive use did cause injury.

Commissions often can create a firm basis for long-lasting social policy. One of the best examples relating to drugs is provided by the Shanghai Commission and the Smoking Opium Act of 1909. Several laws directed at the traffic in narcotics had been introduced into the U.S. Congress before 1908, but only after President Theodore Roosevelt convened the Shanghai Opium Commission in 1909 was federal enactment of any legislation accomplished. The Shanghai meeting had been called to aid China in its attempt to eliminate opium addiction. Then the first U.S. federal legislation against American narcotic abuse, the Smoking Opium Exclusion Act of 1909, outlawed the importation into the United States of opium prepared for smoking.

Within Great Britain, the Rolleston Commission served the important function of establishing basic principles for long-lasting social policy. For more than sixty years, the recommendations of the Rolleston Commission, called the ROLLESTON REPORT, have guided British social and governmental policy toward the prevention and control of nonmedical usage of heroin and other opioid drugs.

In North America, there have been a series of important commissions directed toward nonmedical drug use. In 1939, President Franklin D. Roosevelt asked Mayor of New York Fiorello LaGuardia to chair a scientific commission to investigate the effects of marijuana and other drugs in communities, especially within urban areas of the United States. After looking into these issues for five years, the LaGuardia commission members published their final report entitled *The Marijuana Problem in the City of New York: Sociological, Medical, Psychological and Pharmacological Studies*. Based on interviews with hundreds of users, and after sociological studies and laboratory investigations, the commission concluded that marijuana was not an addictive drug as compared to morphine and that tolerance to marijuana was developed to only a limited degree. Although the LaGuardia report is a significant contribution to the marijuana literature, its conclusions must be qualified because of various weaknesses in the experimental methodologies available at that time.

In 1962, President John F. Kennedy appointed the President's Advisory Commission on Narcotics and Drug Abuse. This commission considered how best to begin reexamination of the problem of drug abuse in the United States, as well as what specific recommendations to make regarding the control of the problem of addiction by law enforcement. The commission's final report in 1963 suggested that psychological treatment might be useful against addiction. The report also marked a shift away from the trend to consider all NARCOTICS the same under law.

During the late 1960s and early 1970s, both the United States and Canada launched commissions of inquiry into a growing problem of nonmedical drug use among the young people of their countries. The Canadian LeDain Commission was appointed in 1969 with the goals of referencing the existing literature and data regarding the nonmedical use of drugs; reporting on the current state of medical knowledge of these drugs; studying the motivation underlying such nonmedical use of drugs; investigating social, economic, educational and philosophical factors related to such use; and finally, recommending ways the Canadian govern-

ment could act to reduce the problems associated with such use.

Shortly afterward, in the United States, the NATIONAL COMMISSION ON MARIHUANA AND DRUG ABUSE issued two important reports, the first entitled *Marihuana: A Signal of Misunderstanding* and the second called *Drug Use in America: Problem in Perspective*. For the most part, the commission's recommendations about MARIJUANA fell on deaf ears. A most important recommendation of the commission was realized in the form of a well-funded national program of periodic epidemiologic surveillance concerning nonmedical drug use and the consequences of such use, now apparent in the MONITORING THE FUTURE studies and in the NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE.

(SEE ALSO: *Marihuana Commission; Opioids and Opioid Control; Shanghai Opium Conference of 1909; U.S. Government: The Organization of U.S. Drug Policy*)

JENNIFER K. LIN

COMMITTEES OF CORRESPONDENCE This organization works toward a drug-free America. It is a nonprofit organization that does not accept government funding and is headed by Otto and Connie Moulton, 24 Adams Street, Danvers, Massachusetts 01923, (508) 774-2641. Drug Watch International and its subsidiary, the International Drug Strategy Institute, were founded to expand the information gathering and dissemination efforts of the Committees of Correspondence.

In 1977, Otto had been coaching little-league baseball and youth hockey and was uninformed on the youth drug culture. After four of his players changed in attitute and ability, he discovered the cause—MARIJUANA. The Moultons began learning about the health effects of marijuana, which was not an easy task. PRIDE and The American Council on Marijuana provided research reports and, armed with facts, the Moultons shared them in their local communities, alerting parents and students to marijuana's effects.

At the 1980 PRIDE conference, they joined with other groups to found a grass-roots PARENTS MOVEMENT called The National Federation of Parents for Drug-Free Youth. The Moultons also revived the Committees of Correspondence, an organization originally founded in 1772 by Samuel Adams—to exchange ideas on building colonial unity. The modern version was formed to build national unity by exchanging facts and ideas on drug prevention, with a newsletter and letter campaign to government favoring antidrug legislation.

The Moultons have served Massachusetts state government and the U.S. government as advisors in the 1980s and 1990s. The Committees of Correspondence maintains a large library on drug-culture history, with books, videotapes, and publications to provide information for global requests; it also provides the public, policymakers, and the media with current research data in an ongoing effort to counter drug advocacy.

OTTO MOULTON

COMMUNITY PARTNERSHIPS *See* Parents Movement; Prevention Movement

COMPLICATIONS This section has articles on some aspects of the physical and psychological complications of substance abuse. It contains an overview of *Medical and Behavioral Toxicity* and individual articles on the following: *Cardiovascular System; Cognition; Dermatological; Endocrine and Reproductive Systems; Immunologic; Liver (Alcohol); Liver Damage (Other Drugs); Mental Disorders; Neurological; Nutritional*; and those *Due to Route of Administration*. Each article is extensively cross-referenced and will refer the reader to other articles throughout the Encyclopedia that will either expand or simplify concepts introduced here, and to articles on the many other behavioral and nonmedical complications that arise as a result of alcohol and drug use.

Cardiovascular System (Alcohol and Cocaine) Since the 1960s, the effects of ALCOHOL (ethanol) on the heart and blood vessels have been extensively studied. Clearly, the toxic effects of both acute and chronic ingestion are independent of nutritional and cardiovascular risk factors. In 1964, a relationship was established between the duration and quantity of alcohol use and the degree of heart disease in patients without nutritional or liver disease. Alcoholism, once felt to be coinciden-

tal with heart muscle damage, is now among the most frequently identified causes, according to two recent studies that report a 32 percent and a 45 percent incidence, respectively.

Attention was also paid to COCAINE abuse as the popularity of this drug skyrocketed in the 1980s (an estimated 30 million Americans had used it and some 6 million were users in 1985). Here too came the recognition that acute and chronic-use users were associated with cardiopulmonary manifestations. A 1998 survey estimated that nearly twelve million Americans use cocaine and alcohol together, and researchers have found a unique product of the body when these two drugs are used in combination—COCAETHYLENE.

ALCOHOL

Effects of Acute Administration in Those With and Without Heart Disease. Even mildly intoxicating levels of alcohol affect the cardiovascular system. The magnitude of effects may depend on the chronicity of use. When six ounces of scotch were given to both alcoholics and nonalcoholics, neither of which groups was suffering from previously recognized cardiac problems, over a two-hour period, only the nonalcoholics demonstrated evidence of diminished heart muscle contractility (pumping less blood per contraction). This depressant effect is enhanced by increasing blood levels 75 milligrams/100 milliliters, but remains for only a few hours after ingestion ceases. However, the amount of blood the heart can pump per minute may actually increase in normal subjects acutely exposed to alcohol, because of an acceleration of the heart rate (increase of contractions per minute).

During the late-intoxication/early-withdrawal stage of acute alcohol consumption, blood pressure may be affected in the noncardiac alcoholic. Blood-pressure elevation is the rule. Blood levels of certain hormones, as well as urinary levels of certain breakdown products, correlate directly with blood-pressure response, which appears to vary with the degree of alcohol intake.

Arrythmias, abnormal heart rhythms, are also commonly described in patients without cardiac problems during acute alcohol intoxication. The so-called holiday heart syndrome represents an acute transient-rhythm disturbance in persons without otherwise detectable heart disease who are examined following heavy drinking. Atrial fibrillation—a very rapid and irregular but generally not life-threatening heart rhythm—was the most common heartbeat irregularity. Normal rhythm was restored in all cases, but recurrence of the syndrome is common.

Cardiac patients who have already had at least one prior episode of heart failure, and who have not compensated by clinical means, usually with symptoms of severe fatigue and shortness of breath, may exhibit even greater sensitivity to acute alcohol consumption. Such individuals given six ounces of scotch over two hours exhibited a substantial increase in measured internal heart pressures, suggesting poor heart function and reserve.

EFFECTS OF CHRONIC ALCOHOL USE

Subclinical Dysfunction. Results of studies on alcoholic subjects without evidence of heart disease suggest that a subclinical disease state may exist. In this situation, recognition of the possibility of disease exists, but no methodology has been developed to detect one in the clinic. In one study, those with at least a ten-year history of heavy alcohol consumption were compared to controls. These patients had biopsy-proven fatty liver disease (a common sequel to chronic alcohol use) without prior history of heart disease. The response of the heart's main pumping chamber—the left ventricle—to storehouses was assessed. Alcoholic hearts were found to have abnormally high internal pressures and could not appropriately compensate by increasing forward blood flow. An index of heart contractions in response to the stimulus was correspondingly low in the alcoholics compared to control. Doppler echocardiography, an ultrasound technique for assessing heart function, has also demonstrated an inability of the heart muscle to relax and its chambers to fill properly with blood. Therefore, the pumping chamber of the heart cannot fill or expel blood in a proper manner. Not surprisingly, autopsy specimens from these patients demonstrate fibrosis, development of abnormal and often harmful tissue, and scarring of the heart muscle. Consideration must also be given to cardiac status in cirrhotics, a group once thought relatively resistant to heart muscle failure. A group of thirty-seven patients with cirrhosis of the liver but no evidence of heart disease were studied. One subset with poor heart function at rest had an abnormal response to left ventricle stressors. The

other subset, which had abnormally vigorous function at rest, also failed to respond appropriately to stressors, suggesting cardiac dysfunction in this group of patients despite a lack of symptoms.

MECHANISMS OF ACTION

The exact mechanism by which alcohol exerts its effects on the heart muscle, at the cellular level, remains speculative. It is believed that altered movement of calcium ions within these cells may be of major importance.

The Picture of Clinical Heart Failure. A full-blown cardiomyopathic picture, a complete observance of disease of the middle layer of the heart, although relatively uncommon among alcoholics, is not unlike that seen with other causes of this syndrome. Most commonly, complaints of weakness and fatigue are present before a history of exertional and nocturnal shortness of breath. Engorgement of the veins of the neck, an enlarged, tender liver, and swelling of the legs and feet are other peripheral signs of heart failure frequently seen in these patients. Heart size is variable. The most significant cardiomegaly, enlargement of the heart, is seen in those patients who develop atrioventricular valve regurgitation, a leaking of blood backward from the large to the small heart chambers, as a result of muscle weakness. This abnormal flow creates heart murmurs and may give a clue to the severity of disease. Blood clots are also a common feature of this syndrome. They often form in the vein of the leg or along the walls of the heart chambers and can travel to the lungs and brain causing near instantaneous death or stroke.

Studies performed in the 1960s clearly documented these effects of alcohol on cardiac difficulties that are left untreated. In one, a patient was fed twelve to sixteen ounces of Scotch daily. Gradually, over a four-month period, he developed the signs and symptoms of heart failure. These reversed when the alcohol was discontinued. This sensitivity to alcohol has individual variability and, unfortunately, the factors contributing to this are unknown. There may be some difference in susceptibility between the sexes. In a study of matched subjects with alcoholic cirrhosis under age forty-five, measures of heart muscle performance were significantly worse in men than in women. Experience also shows that heart failure is rare in alcoholic women prior to menopause.

In either gender, heart failure is generally found in those who continuously (chronically) ingest intoxicating amounts for a minimum of ten years. Isolated studies do, however, suggest the potential for the beneficial effects of cessation of alcohol ingestion. One study of thirty-one alcoholics matched for degree of heart size and symptoms found that all twelve abstainers survived over several years, while all nineteen who continued drinking died. A study of sixty-four patients found a 9 percent mortality rate among abstainers over four years and a 57 percent mortality rate in persistent drinkers.

Coronary Artery Disease. Chest pain in patients with alcoholic hearts is not uncommon, although it had long been held that chronic alcoholics have less severe atherosclerotic coronary artery disease, characterized by a hardening of the arteries accompanied by cholesterol and fatty blockages, than nonalcoholics. Yet, it has been postulated that many heavy drinkers may have a higher incidence of heart attacks (myocardial infarction). Heart attacks have even been reported in a small number of patients without significant coronary disease—possibly related to the scarring of coronary arteries or the spontaneous formation of blood clots within them.

Alcohol may exert at least an indirect effect on coronary anatomy via interaction with fat metabolism. Three major components of fat metabolism, low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, and triglycerides all impact on coronary atherosclerosis or plaquing. Levels of LDL cholesterol (the so-called bad cholesterol responsible for atherosclerosis) appear to be lowered by heavy alcohol use. Moderate use (1–3 drinks per day) does not seem to affect levels of either triglycerides or LDL cholesterol.

HDL cholesterol, the so-called good cholesterol (believed to be protective against atherosclerosis), has shown a consistent relationship with even moderate alcohol consumption. Alcohol is believed to enhance the activity of the enzymes lipoprotein lipase and hepatic triglyceride lipase—thereby favoring HDL production. Autopsy studies have shown a lower prevalence of atherosclerosis, blockages, in alcoholics and cirrhotics. Whether this is due to malnutrition or direct effects on fat metabolism remains unclear.

A reduced degree of coronary disease has also been documented in light as compared to

nondrinkers. Two recent studies both support the hypothesis that light or moderate alcohol intake appears to reduce the risk of fatal and nonfatal heart attacks and the need for coronary angioplasty and bypass operations in both men and women. Neither study included many heavy drinkers, and multiple coronary-risk factors (including cigarette SMOKING) were accounted for. Although these studies supported the positive association between increased HDL cholesterol levels and alcohol intake, no consensus exists to promote alcohol use for the primary prevention of coronary artery disease, owing to its ease of addiction and other multiple harmful effects.

COCAINE

Cocaine abuse has dramatically increased in the United States since the 1980s. One consequence has been the recognition of cocaine's unpredictable medical side effects—the most dramatic and devastating of which are cardiovascular. These include acute impairment of blood supply to the heart muscle, and heart attacks, as well as hypertension, accelerated arteriosclerosis, ruptured aorta artery, inflammation of heart muscle, cardiomyopathy (heart disease), cardiac arrythmia, and sudden death.

Cocaine has two separate primary pharmacologic effects on the heart and vascular system. First, it causes an accumulation of CATECHOLAMINES—accomplished by increasing their release (which include epinephrine [also called adrenaline], NOREPINEPHRINE, and DOPAMINE) from both brain and spinal cord stores and by blocking their reuptake at nerve endings. The result is a more pronounced and long lasting stimulation of the sympathetic nervous system, heart muscle, and vascular smooth muscle, represented clinically by an increase in heart rate, blood pressure, heart contractions, vasoconstriction, when blood vessels constrict, and coronary vascular resistance, a resistance of the blood vessels to blood flow. Cocaine may also induce vasoconstriction via direct stimulation of calcium into smooth muscle cells.

Cocaine's second pharmacologic property is a local anesthetic effect on cardiac tissue. Cocaine can paralyze the movement of the ions (like sodium and potassium) required for the inherent electrical stimulation of heart-muscle function. Therefore, severe toxicity may result—with acute electromechanical dysfunction manifest as an abnormally slow heart rate or acute pump failure.

Several controlled trials have assessed the acute effects of cocaine on humans. Investigators have demonstrated a dose-related increase in heart rate and blood pressure. Others have induced a significant reduction in coronary blood flow in patients receiving intranasal (inhaled through the nose), cocaine at 2 milligrams per kilogram, with coronary angiography, a test using a chemical that can be tracked and seen in the body using an x-ray, revealing a diffuse reduction in vessel caliber (width). Coronary vasospasm, a sudden constriction of the blood vessel, has also been documented with intranasal administration. The effects of chronic use have been studied in animal models, with two experiments demonstrating that cocaine fed to rabbits on high cholesterol diets significantly increased aortic atherosclerosis, or blockages, as compared to rabbits fed similar diets minus the cocaine.

Since all the mechanisms discussed above either increase myocardial oxygen demand or decrease myocardial oxygen supply, heart attacks are natural consequences of cocaine toxicity. Many case reports have temporarily related cocaine use to myocardial infarction, frequently in patients without coronary artery narrowing. Thrombosis, the acute formation of an occlusive clot in a blood vessel, has been documented in nearly one-third of cases. This is consistent with evidence that cocaine causes an increase in the aggregation, collection and buildup, of platelets, blood elements that cling together to initiate blood clotting. Cocaine toxicity may also cause rupture of pre-existing small lipid-containing bulges on the arterial walls, as well as coronary spasm, both of which trigger thrombus formation.

Case reports have also suggested that cocaine has a primary depressant effect on cardiac muscle. Otherwise healthy patients have developed acute cardiac dilation and pump failure associated with acute or chronic cocaine use. With abstinence, such signs and symptoms resolved over days to weeks. Animal studies have confirmed this phenomenon. Autopsy studies on patients with cocaine in the bloodstream have shown inflammation and scarring of heart muscle cells in up to 20 percent, as compared with 4 percent of controls, suggesting a pathologic link.

Ultimately, most cocaine-related deaths are caused by cardiac arrhythmias. Abnormally fast

and abnormally slow heart rates have been reported with cocaine use. Low and moderate doses often trigger the fast and massive doses the slow, including the complete cessation of heart beats (termed *asystole*). The combination of alcohol and cocaine may be even more dangerous than cocaine alone. In the presence of alcohol (ethanol), in humans, cocaine is metabolized to the compound COCAETHYLENE. This chemical renders the combination of cocaine and alcohol more lethal than either alone, and a twenty-one times greater risk of sudden death exists in people with associated coronary artery disease.

Cerebrovascular Disease. The effects of alcohol and cocaine on vascular physiology do not bypass the brain. Epidemiologic studies indicate a passive association between the amount consumed and the risk of cerebral vascular accidents, generally presenting as intracranial hemorrhage, blood vessel ruptures in the head. In one study, heavy drinkers were twice as common among men and seven times as common among women who had sustained intracranial hemorrhage than in the general population. Furthermore, heavy drinkers were more likely to have been intoxicated in the twenty-four hours prior to their event. Young adults and women, generally unlikely candidates for intracranial hemorrhage, are not immune when subjected to acute, or heavy intoxication.

The mechanism for this remains unclear. Hypertension is a known risk factor for stroke in general and alcohol-induced hypertension may be a causative factor. The same may be postulated with cocaine use. As with alcohol-induced cardiomyopathy, individuals who reduce their alcohol intake have a significantly lower risk of developing hemorrhagic stroke than those who continue its use.

(SEE ALSO: *Alcohol; Complications*)

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Cognition PSYCHOACTIVE DRUGS of abuse, used for their perceived mind-altering effects, often have additional cognitive effects of which the drug user may not be aware. A cognitive effect is an impact on mental functions—including processes of learning, perceiving, imagining, remembering, feeling, thinking, reasoning, knowing, and judging.

Psychoactive drugs produce cognitive effects by causing chemical changes in the brain. These effects are mostly short-lived and correspond to the duration and intensity of the chemical changes in the brain. However, cognitive effects can persist after the drug has been eliminated from the body, and some can be irreversible. The common cognitive effects of some psychoactive drugs of abuse are summarized below.

ALCOHOL

Ethanol (also called ethyl alcohol) is the drinking ALCOHOL of BEERS AND BREWS, wine, distilled spirits, or medicinal compounds; it acts by depressing or reducing cognitions. Initially, alcohol reduces inhibitions, and this results in more spontaneity or impulsivity and a feeling of relaxation. As the amount of alcohol acting on the brain increases, the ability to perceive, remember, reason, and judge is progressively impaired. Further increases in the amount of alcohol can depress the brain and cognitions to the point of loss of consciousness. Due to cognitive impairment, the person may not perceive the impairment (e.g., "I'm not drunk") and take undue risks (e.g., DRUNK DRIVING, indiscretions).

Alcoholic blackouts are impairments of memory for events that occurred while one was conscious but under the influence of alcohol. Such black-outs are not limited to chronic alcoholics. Long-term use of alcohol can lead to subtle impairment of perceiving, responding, and remembering that may not be detectable without special psychometric tests. A particular form of impairment of memory,

called the amnestic syndrome, has been seen in alcoholics; they are unable to remember recent events although memories from long ago remain reasonably intact. By contrast, in alcoholic dementia, deficits in all cognitive functions are seen. Some deficits may persist for life even if the person stops drinking.

Paranoid states of unfounded suspicion or jealousy may manifest or be aggravated under the influence of alcohol. In alcoholic, HALLUCINATIONS people can have vivid but unreal perceptions while awake; these typically occur as a result of neurochemical changes in the brain when alcohol use is abruptly discontinued after periods of excessive drinking. Even after months have elapsed since their last drink, alcoholics can have cognitive deficits, especially in visual-spatial abilities, hand-eye coordination, abstract reasoning, and new learning.

TRANQUILIZERS, SEDATIVES, AND HYPNOTICS

These drugs are often collectively referred to as "downers." Persons taking them are at risk for the cognitive impairments discussed above, under "Alcohol." The ELDERLY are particularly at risk for confusion.

STIMULANTS

Stimulant drugs have effects that are the reverse of depressant drugs—they arouse the nervous system. They include such drugs as COCAINE, AMPHETAMINES (speed), and CAFFEINE. In low doses, perception is heightened, attention is increased, and thought processes are speeded up, resulting in a feeling of greater alertness. MEMORY, however, may be affected, resulting in impaired recall of material learned while under the influence of stimulants. Higher doses intensify the above effects, leading to restlessness and rapidity of thoughts, which reduce attention. Vulnerable persons may become paranoid or even psychotic. Higher effective doses of stimulants may occur via intravenous administration or smoking of cocaine, affecting the brain rapidly and resulting in an abrupt "rush" or "high." These effects are typically short-lived but are so intense (pleasurable) that individuals may repeat doses. Discontinuation of stimulants after a long period of use often leads to a temporary period of DEPRESSION. There is evidence that long-term and repeated doses of stimulants can severely damage the brain and affect concentration, mood, and reasoning

MARIJUANA

Cannabis sativa is often used for the subjective effects of relaxation and a decreased awareness of conflicts. It is also known to distort perception of time and to reduce responsiveness. Long-term use of *Cannabis* has been associated with apathy, under-achievement, and lack of motivation.

HALLUCINOGENS

Hallucinogenic drugs distort perceptions and cause hallucinations. They include LYSERGIC ACID DIETHYLAMIDE (LSD), PHENCYCLIDINE (PCP), MESCALINE, PSILOCYBIN mushrooms, and several newer drugs with hallucinatory and stimulant effects (called "designer drugs," e.g. ecstasy. Apart from profound effects on perceptions, responsiveness, learning, and judgment are affected. Some users experience flashbacks—spontaneous vivid recollections of experiences that occurred while under the influence of hallucinogens.

USE DURING PREGNANCY

Psychoactive drugs used during PREGNANCY affect the developing fetus. Prenatal exposure, particularly to alcohol but possibly to *Cannabis*, or stimulants has been associated with cognitive impairments detectable early in the child's life and eventually resulting in developmental problems and school, social, and occupational difficulties.

(SEE ALSO: *Imaging Techniques*)

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Dermatological With the exceptions of rashes and other skin conditions resulting from idiosyncratic or allergic reactions to drugs, most drug use complications involving skin damage result from the use of hypodermic needles or other means of drug injection that involve breaking the skin surface. There are three primary types of injection: (1) *subcutaneous*, also known as "skin-popping," wherein the needle is injected into or directly under the skin surface; (2) *intramuscular* (IM), wherein the needle is injected into muscle mass, often in the shoulder or buttock; and (3) *intravenous* (IV), direct injection into a blood vessel. Skin damage can result from repeated injection in the same area, failure to clean the injection site, nonsterile needles, and/or impurities or insoluble materials in the substance injected. Adulterants in the drugs, liquids used to dilute the drugs and contaminated injection paraphernalia, and the surface of the injection site all provide sources of viruses, bacteria, and fungi.

The most common skin damage from repeated injection is needle-track scars. These are usually caused by unsterile injection techniques or by the injection of fibrogenic particulate matter—material often used by dealers to add bulk and weight to the drug or buffers in tablets that have been ground up and liquified for injection. Carbon deposited on needles by users who try to sterilize by heating the needle tip with a match may produce a "tattoo" discoloration, accompanied by a mild inflammatory reaction under the skin at the point of entry. Such scars are found mostly on the arms, but they can occur anywhere on the user's body that has been used as an injection site, including thighs, ankles, neck, and penile veins.

Needle abscesses, characterized by redness; a stinging, itching sensation; and swelling at the site, often result from repeated injection without cleaning the injection site. Such skin flora as staphylococci and streptococci are driven beneath the skin surface to infect the site, often with pus formation. Forms of contact dermatitis can also result from allergic reactions, especially to fluids used to sterilize the skin at injection sites. Infections and allergic reactions increase as an individual's resistance decreases with a drug-compromised immune system.

Subcutaneous injection of SEDATIVE-HYPNOTIC drugs, such as BARBITURATES, can cause cellulitis—where the tissue becomes reddened, hot, painful, and swollen at the injection site. If not treated, the cellulitis may last for a long time. In extreme cases, the cellulitis may eventually cover most of the user's body as new needle sites are used to avoid painful areas. Superficial cellulitis, septic thrombophlebitis, and simple needle abscesses can usually be treated with local heat, incision, and drainage, followed by culture and sensitivity testing and appropriate antimicrobial therapy.

Repeated intravenous injection may produce anaerobic infections or abscesses that produce a foul-smelling discharge, sometimes gas formation, and a cellulitis that is characterized by a rapidly progressing stony or wooden-hard tenseness, often some distance from the original needle site. Although the mechanism of these infections is unclear, it is thought to involve a disruption of blood supply to the area from edema (fluid collection) resulting from the cellulitis. Treatment involves wide incision and pressure reduction in the affected area.

Kaposi's Sarcoma. Kaposi's Sarcoma is a malignancy arising in the skin usually in the cells lining the blood vessels (endothelium). The lesions have a nodular or plaquelike appearance, may be localized and indolent or disseminated, and involve aggressive spreading to mucous membranes and visceral organs, especially the gastrointestinal tract.

Prior to 1980 and the advent of the human immunodeficiency virus (HIV) and AIDS, Kaposi's sarcoma was considered a rare disease and primarily limited to elderly males of Mediterranean ethnic origin. Since that time, widespread dissemination of HIV and the epidemic of AIDS accompanying it has made Kaposi's sarcoma much more common. Substance abusers who administer their drugs parenterally (subcutaneously, intramuscularly, intravenously) are a higher risk group for Kaposi's sarcoma since many of these individuals inject drugs with unsterile needles that frequently are used in common with others. They therefore have an excellent opportunity of acquiring HIV from infected blood.

The aggressive form of Kaposi's sarcoma has occurred in at least one-third of patients with AIDS and has reached epidemic proportions in the United States and many African countries. In many AIDS patients, Kaposi sarcoma lesions may actually be the first notable manifestation of the disease. The lesions usually first appear on the upper part of the body, but rapidly spread to lymph nodes, the mucosa of the mouth and the gastrointestinal tract and other visceral organs.

Chemotherapy is the treatment of choice, either a single agent or a combination of agents. Interferon-a effectively slows the progression of lesions and cures others. The injection of vincristine into the lesions is also useful. The course of the disease is dictated by the level of immunosuppression that is present.

(SEE ALSO: *Allergies to Alcohol and Drugs; Complications: Route of Administration*)

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Endocrine and Reproductive Systems

Many fundamental challenges remain in understanding the impact of ALCOHOL and drugs on endocrine and reproductive function. This article presents what is currently known; before beginning, a few caveats deserve attention.

Many factors may influence the degree to which illegal drug or alcohol abuse may cause an abnormality of endocrine or reproductive function. These factors include (1) the amount and duration of consumption; (2) the route of illegal drug administration; (3) whether there is preexisting or concurrent damage to an endocrine/reproductive organ; (4) concurrent use of another drug; and (5) the genetic predilection for an endocrine disorder. Often, our knowledge about these factors and how they interact with one another is more limited than what is known about the range of endocrine and reproductive dysfunction associated with the chronic consumption of alcohol and the abuse of illicit drugs.

Knowledge is also limited because some endocrine or reproductive consequences may be manifested only by an abnormal result from a laboratory (biochemical) test. The absence of a physical sign or a clinical symptom may lead to the false impression that there is no endocrine/reproductive consequence. In addition, there are challenges in ascertaining whether the alcohol- or drug-abuse related endocrine/reproductive dysfunction is due to the drug itself or to the social context in which the drug is used. Finally, endocrine or reproductive disturbances may also occur from the consequences of WITHDRAWAL syndromes when the drugs or alcohol ingestion is stopped or reduced. To the extent to which these issues have been clarified, we will note them here.

HYPOTHALAMIC/PITUITARY

Most endocrine and reproductive function is influenced directly or indirectly by the BRAIN—specifically by the functional interactions of the brain's hypothalamus and pituitary with the target endocrine organs. The hypothalamus produces pituitary-regulating hormones; all are peptides except one (DOPAMINE). In response to each of these hypothalamic hormones, the pituitary releases a hormone, which influences the function of an endocrine or reproductive organ.

Alcohol. The anecdotal reports of changes in sexual function following alcohol consumption was the stimulus for much of the research targeting hypothalamic-pituitary relationships, since impairments here may often result in sexual dysfunction. Although acute alcohol use has been reported in public surveys to be associated with increased sexual drive and functioning, clinical and animal research have revealed major hormonal dysfunctions in chronic or heavy alcohol users.

Prolactin (PRL)—the pituitary hormone associated with preparation during pregnancy for breastmilk secretion—is increased with heavy alcohol use; however, chronic alcohol use inhibits the pituitary release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Both LH and FSH are important in regulating the sex hormones produced by the testes in males and the ovaries in females. Yet, when alcohol is administered acutely, there are no significant changes in PRL, LH, or FSH serum levels.

Heavy alcohol consumption is associated with an increase in pituitary-secreted adrenocorticotropic hormone (ACTH), partly explaining the

"pseudo-"Cushing's syndrome (moon faced appearance, central obesity, muscle weakness) and the increased melanocyte-stimulating hormone (MSH), which possibly leads to darkening skin pigmentation. Although there is no consistent effect of heavy alcohol use on the pituitary's release of thyroid-stimulating hormone (TSH) or growth hormone (GH), a rise in the blood alcohol level is associated with the inhibition of antidiuretic hormone (ADH) release from the posterior pituitary, resulting in increased urination.

Drugs. Complaints of derangements of libido (sex drive) and sexual functioning in OPIOID addicts (HEROIN) were among the first lines of clinical evidence to suggest the possible role of such narcotics in altering hypothalamic-pituitary functioning. Although most of what is known about drug-abuse related hypothalamic-pituitary abnormalities focuses on heroin use, the epidemic proportions of COCAINE abuse and dependency in the 1980s have brought renewed scientific interest to this area.

Studies have shown that opioid use has been associated with increased serum PRL without disturbances in serum GH or TSH levels; and cocaine use has been associated with both high and low PRL levels. The contradictory findings in the case of cocaine use might be attributable to the variations in patterns of cocaine use. Animal studies have shown that gonadotropin-releasing hormone (GnRH), released from the hypothalamus, did not stimulate PRL following acute cocaine administration, and it did not prevent acute cocaine-associated PRL suppression. Elevated levels of dopamine have been observed during acute cocaine administration, but chronic cocaine use may deplete dopamine.

In patients receiving METHADONE therapy for opioid addiction, some investigators have reported a normal rise in TSH released by the pituitary, in response to stimulation by the hypothalamic hormone called thyrotropin-releasing hormone (TRH). Others have observed a blunted TSH and PRL response following TRH administration in active heroin users.

Although normal basal LH secretion has been observed in cocaine abuse, opiate use is associated with decreased basal FSH and LH levels in males. In female heroin addicts, these low levels of the pituitary gonadotropins have clinical relevance, resulting in a consistently normal FSH response and a variable LH response following a GnRH challenge.

Some researchers have demonstrated normal functioning of the hypothalamic-pituitary-adrenal (HPA) axis in former heroin addicts who were maintained on methadone both long-term and only for a number of months. However, there is also evidence suggesting alteration of the normal biological rhythm of hormonal secretion.

SEX HORMONES

Diminished sexual drive and performance in opioid users have raised questions about the relationship between such narcotic drug use and disturbances in the levels of sex hormones. Although some reports show no significant differences in serum-testosterone levels between heroin addicts, METHADONE-MAINTAINED patients, and normal controls, other studies have not confirmed these results. Some researchers have reported plasma levels of testosterone to be consistently lower in active heroin addicts, in addicts who self-administer heroin in controlled research settings, and to be within normal range in long-term methadone-maintained patients. Additionally, some evidence shows that plasma testosterone levels that are depressed under circumstances of heroin administration followed by methadone maintenance and then withdrawal gradually returned to preheroin-use levels.

Opioid effects on the estrogens of both males and females may be responsible for the clinical observations of sexual dysfunction. In the male heroin addicts studied, the plasma estradiol concentrations were either low or within normal ranges; in the females, the plasma estrogens are low. A clear explanation of these observed derangements in plasma testosterone and estrogens is unknown. Female heroin addicts frequently experience cessation of or irregular menses. However, most regain normal menstrual funcion when stabilized on methadone and under these circumstances fertility seems unaffected. The anecdotal reports and, in limited cases, experimental evidence of the influence of MARIJUANA on sexual function and sex-hormone levels are also inconsistent and confusing.

The illicit drug-related disturbances discussed above suggests that the narcotic-related depressions in sex-hormone production of the ovaries and testes may occur because they reduce the pitu-

itary's stimulation of these sex organs. Still, this has not been a consistent finding.

REPRODUCTION AND PREGNANCY

Impotence, atrophy of the testes, infertility, and decreased libido are not uncommon complaints in male alcoholics. These observations are thought to be secondary to the direct effects of alcohol on testicular tissue, to an alcohol-associated decrease in sperm motility, and to an alcohol-related decrease in Vitamin A and zinc. Both Vitamin A and zinc are important in maintaining testicular tissue growth. In young females, alcohol abuse is associated with amenorrhea and anovulation; in chronic users, with early menopause. There is evidence that vaginal blood flow decreases as the alcohol serum level increases.

Despite these clinical observations, when rigorously investigated, there were no consistent changes in estradiol, progesterone, or testosterone. Consequently, it is difficult to determine whether these observations were due to alcohol-related liver disease, malnutrition, or the direct toxic effects of chronic alcohol use.

During PREGNANCY, alcoholism is associated with increased risk of spontaneous abortion and the development of FETAL ALCOHOL SYNDROME (FAS). FAS is comprised of a characteristic pattern of skin/facial abnormalities with growth and development impairments, which are believed to be related to alcohol's suppression of the sex hormone progesterone. While the features of FAS may vary, fetal abnormalities associated with alcohol can be divided into the following four categories: (1) growth deficiency; (2) central nervous system dysfunction; (3) head and facial abnormalities, and (4) other major and minor malformations.

ADRENALS

Our understanding of the relationship between opioid drug use and the functioning of the adrenal gland is based on incomplete and often contradicting information. Some scientists have published reports of normal plasma cortisol levels (from the adrenals) during heroin use and withdrawal, under research conditions of heroin self-administration, and during methadone-maintenance treatment. In methadonetreated patients, ACTH produced by the pituitary stimulates the adrenal gland to produce cortisol. In another study, there was a decreased plasma-cortisol response to intravenous cosyntropin (an ACTH-like substance) stimulation in methadone-treated patients. There are also reports of low normal or subnormal plasma-cortisol levels in heroin users and disturbances in the daytime cortisol secretion from the adrenal gland in methadone-maintained patients.

The variable findings from the several studies may be attributed to differences in the types of drugs used, in the state of stress-associated drug withdrawal, in patterns of drug use, in study design, or to a combination of these and other as yet unknown factors. There is also the well-known problem of ACTH measurement, often resulting in falsely low values.

CARBOHYDRATE METABOLISM

The opioids are virtually the only class of *illegal* drugs for which there is information about the pharmacologic effects on serum-glucose levels. We have long-standing reports of opiate-associated hyperglycemia, but the mechanisms explaining these empirical observations are incompletely understood. In association with chronic opioid use, there are reports of both low levels of serum glucose and high levels of insulin. The conflicting results of some investigations may be due, in part, to differences in study design (e.g., nutritional state of the research subjects, amount of the glucose used in clinical studies, or the time(s) administered).

To briefly review the regulation of glucose control: The pancreas, an endocrine organ located in the upper abdomen, plays a central role by secreting glucagon to raise serum-glucose levels and by secreting insulin to lower serum-glucose levels. After the discovery of endogenous opioid peptides in the human pancreas, subsequent research provided information that one such endogenous opioid, beta-endorphin, stimulates the secretion of glucagon and a biphasic rise of insulin. This may, in part, explain the observations of both elevated and reduced serum-glucose levels in heroin users. Whatever the nature of the exact mechanism, glucose metabolism is deranged in both heroin and methadone use by some direct or indirect parameter of serum-glucose regulation.

The alcohol-related aberrations of carbohydrate metabolism are also quite complex. Some investigators have demonstrated that acutely adminis-

tered alcohol may result in a reversible and mild resistance to the glucose-lowering effects of insulin, perhaps explaining the observations of a rise in glucose following alcohol use. In fasting individuals, alcohol administration can lead to severe depressions of serum glucose, primarily by reducing the liver's production of glucose. Serum-glucose levels are also lower in chronic alcohol users with concurrent alcohol-related liver disease. Nevertheless, serum levels are elevated in alcoholics with concurrent alcohol-related destruction of the pancreas. Even without other concurrent diseases, alcohol consumption may result in either no changes or in minimal to mild elevations or reductions in serum glucose.

THE THYROID

Located in the anterior aspect of the neck, the thyroid gland secretes thyroxine (T_4) and other hormones whose principal purpose is to regulate the metabolism of other tissues in the body. The production of T_4 by the thyroid is under the control of the TSH produced by the pituitary. Therefore alterations in thyroid function can be the result of problems directly involving the gland or disruptions in the TSH-mediated control of the thyroid gland.

Despite the frequency, duration, or amount of use, it appears that there are no clinical signs or symptoms of thyroid dysfunction in chronic heroin or alcohol users. Disturbances in biochemical indices (laboratory tests) of thyroid function are, however, not uncommon in opiate or alcohol use. The total T_4 is decreased while the amount of biologically available T_4 (free T_4) and other indices of thyroid function are normal in heavy alcohol users.

In active heroin users or during heroin withdrawal, total T_4 levels are increased in association with normal, subnormal, or high levels of other parameters of thyroid function. In methadone-maintained persons, there are reports of normal and slight-to-significant increases in total T_4 in conjunction with increased levels of thyroxine-binding globulin (TBG), a protein that binds thyroid hormones in blood. Interestingly, successful methadone maintenance is associated with a correction of these biochemical disturbances.

There are a number of possible explanations for the biochemical derangements observed during opiate use. To maintain an adequate range of biologically active T_4, the total T_4 is increased whenever there is an increase in TBG. Perhaps the increase in total T_4 is the result of a direct opiate-induced elevation of TBG. It is also possible that the altered liver function seen in chronic heroin and alcohol users may be responsible TBG abnormalities, leading to disturbances in T_4 levels. Finally, it is possible that opiate-related or alcohol-related disturbances may be due to a combination of the above mechanisms as well as to some other still undefined processes.

BONE METABOLISM

The observations of increased fractures sustained by alcoholics has prompted investigations about the role alcohol may play in disturbances of the structure and mechanical properties of bone. Some studies have shown reduced bone mass in alcoholics, while others have reported decreases in compact and trabecular bone mass—a condition called osteoporosis. Some of these disturbances in new bone formation may be mediated by alcohol's impairment of calcium and Vitamin D metabolism, both of which are crucial to bone metabolism. Nevertheless, there does remain considerable doubt as to whether the bone complications are due to alcohol itself or due to alcohol-related liver disease, to malnutrition, or to a potential host of other factors. Chronic liver disease unrelated to alcohol has also been a cause of osteoporosis and other bone diseases.

CONCLUSION

The endocrine and reproductive consequences of illicit drug and alcohol abuse are extensive and profound. Both drug and alcohol abuse result in clinically significant multiglandular derangements. Although knowledge about the dimensions of such disturbances to endocrine and reproductive function slowly increases, explanations of the scientific mechanisms accounting for these observations remain to be elucidated. Given the role of alcohol and illicit drugs in society, however, the spectrum of related endocrine and reproductive complications can be expected to expand and, thereby, increase in public-health significance.

(SEE ALSO: *Complications: Liver Damage*)

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Immunologic This article describes the basic and clinical immunologic aspects of alcohol and drug abuse.

ALCOHOL

The physiological characteristics of ALCOHOL (ethanol) allow it to interfere intensively with the functions of immune cells. Alcohol is completely miscible with water and, to some degree, is fat-soluble. It crosses membranes by diffusion across a concentration gradient. Historically, alcohol has been associated with lower host resistance and increased infectious disease. For example, ALCOHOLISM has been closely associated with lung abscesses, bacteremia, peritonitis, and tuberculosis. Although these infections might be a result of malnutrition or poor living conditions, prolonged consumption of alcohol also results in alterations of immune responses, ultimately manifested by increasing susceptibility to infectious agents. Overwhelming evidence shows that alcohol abuse broadly suppresses the various immune responses, seriously impairing the body's normal host defense not only to invading microbes but also to its defenses against CANCER cells.

These disruptions are the combined result of direct toxic effects on the immune system and indirect effects such as malnutrition, oxidative stress, endocrine changes, and the complications of liver disease. The alcoholic's predisposition to extracellular and intracellular infection indicates the effects of alcohol consumption at the local, humoral, and cellular levels, inhibiting immune response and host defense. Recent evidence suggests that aberrant regulation of the neuroimmune-endocrine networks may be a major risk factor for the development of alcohol-induced immunosuppression, leading to the collapse of host defense. Bidirectional communication can occur between the immune and neuroendocrine systems. Accordingly, stimulated lymphoid cells send signals mediated by cytokines and other immune products to inform the central nervous system about the activity of the immune system. Neuroendocrine molecules, in turn, may complete a feedback loop by modulating the immune response via the pituitary-endocrine axis as well as the autonomic neural output. Thus, effective feedback communications between the endocrine and the immune system may be crucial to the host's defense responses.

Clinical and experimental studies indicate a relationship between excessive alcohol use and compromised immune responses. Human studies have shown that chronic alcohol ingestion is associated with abnormalities of both humoral and cellular immunity. These abnormalities include a depression of serum bacteriocidal activity, alterations of immunoglobulin production, leukopenia, defects in chemotaxis, decreased antigen trapping and processing, and decreased T-cell mitogenesis. The clear association between alcoholism and infections such as tuberculosis and listeriosis indicates defective functioning of cell-mediated immunity. A study has linked alcohol abuse and deficient T-cell responsiveness. Skin-test reactivity using purified protein derivative and dinitrochlorobenzene has also demonstrated poor responses in alcoholics with liver disease. Natural killer (or NK) cell activity is impaired in acute alcohol intoxication and in chronic alcoholic liver disease; NK cells are programmed to recognize and destroy abnormal cells, such as virus-infected or tumor cells. Some researchers have speculated that decreased NK cell activity may be intimately involved in the increased incidence of tumors in alcoholics.

Giving alcohol to animals also has a profound effect on decreasing the weight of their peripheral lymphoid organs as measured by a decreased number of thymocytes and splenocytes. In mice, alcohol use produces thymic and splenic atrophy and alterations in the circulating lymphocytes and lymphocyte subpopulations, as well as alterations in cellular and humoral immunity and impaired cytokine production. Also impaired by dietary alcohol are

antibody-dependent cellular cytotoxicity, lymphocyte proliferation, B-lymphocyte functions, and cytokine production by lymphoid cells (the lymph and lymph nodes). Thus, alcohol-induced immunosuppression may render alcoholics more susceptible to tumorigenesis and infection.

Alcoholics are susceptible to infections by bacteria such as *Listeria monocytogenes*, *Vibrio vulnificus*, *Pasteurella multocida*, and *Aeromonas hydrophilia*. The severity of these infections has raised the possibility of a neutrophil (white blood cell) abnormality in these patients. The proper functioning of neutrophils is critical for host defense against microorganisms. Neutrophils are the chief phagocytic leukocyte of the blood; they are short-lived cells having a life span of approximately four days. Their production is a tightly regulated process centered in the bone marrow. Chronic alcoholics have often been noted to be leukopenic (abnormally low in leukocytes). The toxic effect of alcohol is now believed to be caused by the depression of the T-cell-derived colony-stimulating factor rather than to direct suppression of myeloid (bone marrow) precursors secondary to bone marrow toxicity.

Neutrophils must recognize the invading pathogens, engulf them, and destroy them using a number of killing mechanisms, which include adherence, chemotaxis, locomotion, phagocytosis, and intracellular killing. Several functions of neutrophils are affected by alcohol *in vitro*, including impairment of chemotaxis, decreased migration of neutrophils within vessels, altered adherence to nylon fibers *in vitro*, impaired phagocytosis, and decreased intracellular killing of bacteria. In human with advanced cirrhosis from chronic (prolonged and excessive) ingestion of ethanol and impaired phagocytic capacity, decreased metabolic activity was observed in the liver's reticuloendothelial system; there also were impairments of neutrophil chemotaxis, bacterial phagocytosis and killing, and alterations of neutrophil-antigen expression. Neutrophil dysfunction is therefore responsible for aggravating the susceptibility to secondary infections seen in alcoholics.

The balance of cellular and humoral immune response to antigens is controlled by communication between immunocompetent cells. They are regulated to a great extent by soluble mediators (termed *cytokines*) produced mostly by T-helper cells and macrophages. Cytokines are biologically active polypeptide intercellular messengers that regulate growth, mobility, and differentiation of leukocytes. Thus, cytokines have extremely important roles in the communicating network that links inducer and effector cells to immune and inflammatory cells.

Since any perturbation in the tightly controlled cytokine regulatory system can result in immune alterations modifying host resistance to infectious disease and cancer, the influence of alcohol consumption on cytokine secretion has been investigated considerably. Several studies have indicated a correlation between circulating levels of macrophage-derived cytokines and disease progression during chronic alcohol consumption. Increased plasma concentrations of tumor necrosis factor have been observed in cases of alcoholic liver disease and, interestingly, relate significantly to decreased long-term survival; plasma Interleukin-1 is also significantly increased in these patients (relative to healthy controls) but does not correlate with increased mortality.

In a model of alcohol-fed mice, we found that, compared to controls, production of all cytokines was suppressed by chronic alcohol consumption, suggesting general immunosuppression. The elevated levels of cytokines in some animals with murine (mice and rats) AIDS were, however, increased further by alcohol ingestion as compared to controls that indicated alcohol-induced aggravation of some AIDS symptoms. Similarly, those cytokines suppressed by murine AIDS were further suppressed by alcohol. Thus, alcohol exacerbated their immune dysfunction. Several pathways may be involved in mediating the interaction between the endocrine system and the immune system. Recent findings indicate that pituitary peptide hormones can directly influence immune response. In addition, when a neurotransmitter is released in lymphoid tissues, it may locally modify the functional properties of lymphocytes and release of cytokines.

In human studies regarding alcohol, all parameters, such as hormone levels and immune responses to monitor changes of immune response and neurotransmitter, are usually detected in serum. Since the serum levels of these parameters cannot accurately reflect the real situation in the lymphoid organs or tissues, some results from them, therefore, could be misleading. No animal model for alcohol studies can mimic the complications of alcoholic liver disease often observed in human alcoholics.

Furthermore, the facts that different hormonal status in individual animals, even within same strain of animal, and difficulty in defining hormonal status in animals indicate that some results from animal studies could also be misleading. Therefore, the research on the mechanism of alcohol's effects on the neurological system at cellular and systemic levels and the interaction between endocrine and immune system should continue if we are to understand the complex changes caused by the direct and indirect effects of alcohol consumption.

COCAINE

COCAINE acts directly on lymphoid cells (the lymph and lymph nodes) and indirectly modulates the immune response by affecting the level of neuroendocrine hormones. The first studies about the impact of cocaine use on the immune response were initiated because epidemiological data demonstrated a high prevalence of ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) in polydrug users. Depending on the different administration routes, the plasma levels of cocaine in humans appear to be in the range of 0.1 to 1 micrograms per milliliter (μg/ml). Such concentrations last only for thirty to sixty minutes at these levels and then decline because of cocaine's short biological half-life of about 1 hour. The direct effects of cocaine and its metabolites on immune cells should occur only during a short time, except in heavy cocaine users who use the drug several times a day every day. Besides the direct effects on immune cells, cocaine could indirectly affect the immune response via its impact on the neuroendocrine system—and both have been shown.

Short-term exposure of mice to cocaine by daily intraperitoneal injection for fourteen days reduced body, spleen, and thymus weight in the animal. Cocaine increased the responsiveness of lymphocytes to mitogens (cell proliferation initiators) and the delayed hypersensitivity responsiveness, but it suppressed the antibody response. All the animal studies, however, suggest that the immune system requires continuous exposure to cocaine to demonstrates its suppressing or stimulating effects. After a single dose of cocaine (0.6 mg/kg), nonhabitual cocaine users showed a significant stimulation of natural killer cell activity, which is vital to defend against cancers. The levels of natural killer cells were also increased, but the levels of T-helper and suppressor cytotoxic cells, B cells, and monocytes were not elevated.

Cocaine causes neuroendocrine-mediated effects on the immune response. It stimulates the brain's hypothalamus to increase secretion, producing potentiated secretion of beta-endorphin. As a result of cocaine administration, beta-endorphin binds to opioid receptors on monocytes and lymphocytes and exerts multiple stimulating and suppressing effects on these cells, including secretion of immunoregulatory cytokines. The net outcome of the reactions related to the immune response of the host is difficult to assess because other determinants of these interactions (such as the psychological and social situation of the cocaine user) are also possible.

There are other mechanisms that might be operating to mediate cocaine-induced immunomodulation, including nutritional deficiencies and their impact on lymphoid cells. As early as 1870, the French physician Charles Gazeau suggested that coca leaves might be used to suppress the appetite. With food deprivation, which is common under conditions of habitual drug use, the self-administration of cocaine by rats increased. Although data indicate a poor nutritional status for cocaine users, no study has yet assessed the nutritional status of drug users as it contributes to a compromised immune competence. Cocaine clearly modifies hormones with immunoregulatory properties via neurological effects. In addition, malnutrition could be a factor on cocaine use, resulting in altered disease and tumor resistance. Intravenous use of drugs, including cocaine, is associated with the transmission of HUMAN IMMUNODEFICIENCY VIRUS (HIV), and ultimately the development of AIDS. Immunomodulation by cocaine after HIV infection could accelerate disease development as well as overall resistance to a variety of pathogens found frequently in intravenous drug users.

TOBACCO

Although it is now well known that the use of TOBACCO is a major health hazard, millions of Americans still continue to smoke and the popularity of smokeless tobacco is on the rise. Tobacco use is the chief cause of lung cancer in smokers and is strongly linked with the oral cancers of those who use chewing tobacco or SNUFF.

The pulmonary alveolar macrophage (PAM) is the cellular component of the immune system comprising the first line defense of the lung, offering protection against inhaled particles, including irritants and microbial invaders. Because PAM has exposure to both the bloodstream and the atmosphere, it is uniquely suited to perform its protective functions, which include clearance, immune modulation, and modulation of surrounding tissue. There is general agreement that the number of PAMs in smokers' lungs is increased two to twenty times above that found in the lungs of nonsmokers. It also appears that there is a difference in the morphology and certain aspects of the function of alveolar macrophages between the two groups. In general, PAMs from smokers are larger, contain more lysosomes and lysosomal enzymes, and are more metabolically active than those nonsmokers, suggesting that they may be in a chronically stimulated, more active state. This might lead to the inference that there would be greater phagocytic capacity in the lungs of smokers, resulting in increased clearance of foreign matter. However, the responsiveness of smokers' macrophages to foreign bodies or bacteria was equal to or less than that of nonsmokers, leading researchers to conclude that chronic stimulation of PAMs by cigarette smoke may be harmful rather than beneficial to the immuno-competence of the lung.

There is some disagreement as to whether smoking affects the phagocytic and bactericidal activity of PAMs. The question of whether tobacco smoke alters the tumoricidal ability of PAMs has not yet been answered. Thus, the relationship between cigarette smoking, neutrophil accumulation in the lung, and lung destruction continues to be researched. It is known that particles from cigarette smoke are present in the PAMs of smokers, and researchers have found that the PAMs of cigarette smokers released a potent chemotactic factor for neutrophils, whereas those of nonsmokers did not. Therefore, cigarette smokers had an increased number of neutrophils in the lavage fluid and in lung biopsy tissue as compared to nonsmokers. Neutrophils store and release elastase, a substance implicated in the development of certain lung diseases. Smokers' lungs are exposed to a large chronic burden of elastase from neutrophils, which may predispose them to lung destruction.

A number of animal and human studies comparing peripheral blood samples of smokers and nonsmokers have indicated that smokers have altered immunoglobulin levels. Elevated levels of immunoglobulin E (IgE) were present in a high proportion of the smoke-exposed animals but in none of the controls. Studies on human subjects have also revealed that IgE levels were higher in smokers than in nonsmokers. A study of coal workers indicated that both mining and nonmining smokers had depressed serum IgA and IgM levels as compared with similar groups of nonsmokers. A disturbing finding in relationship to increased immunoglobulin levels in smokers is the effect that maternal smoking may have on the fetus. In newborn infants of mothers who smoked during pregnancy, IgE was elevated three-fold. Tobacco smoke affects fetal immunoglobulin synthesis, stresses the fetal immune system, and can predispose the infant to subsequent sensitization. Thus, 34 percent of the reported asthma in childhood may be caused by maternal smoking.

Natural killer cells, thought to serve important antitumor and antiviral functions in the body, have been found to be decreased in smokers. Studies of the white blood cells called basophils in the peripheral blood indicated that there are alterations linked to tobacco smoking as well.

When considering the effect that tobacco use has on immunocompetence, other confounding variables must also be accounted for, including genetic factors, preexisting disease, and nutritional status. Smoking has been observed to cause deficiencies of vitamin C, beta-carotene (vitamin A), and other nutrients that have important functions in protecting immunity.

Tobacco smoking causes deleterious effects on the pulmonary and systemic immune systems of experimental animals and in humans. Aspects of both cell-mediated and humoral immunity are affected. It is often difficult to compare studies directly because of the variability in smoking behaviors and the differences among tobacco products. Although it is expected that heavy smoking causes the most amount of immune system damage, that does not mean light to moderate smoking is safe. Thus, if some alterations due to smoking are reversible, it is not yet known whether long-term smoking may cause the impairment of the immune system to become permanent. Further, simultaneous exposure to other air contaminants or air pollution may exert damaging synergistic effects on local or systemic immune defenses.

MORPHINE AND OTHER OPIOIDS

Several studies have drawn a parallelism between MORPHINE abuse and immune inhibition. *In vitro* studies have shown that polymorphonuclear cells and monocytes form in patients subjected to morphine treatment but that they were severely depressed in their phagocytic and killing properties as well as in their ability to generate superoxide. OPIOID addiction also caused alterations in the frequencies of T-cells and null lymphocytes in human peripheral blood.

There is convincing evidence of the presence of opioid receptors on various types of human immune cells. The presence of opioid receptors on immune cells may allow for modulation of specific immune functions in the presence of exogenous opiates. Various administration schedules for opioids were shown to potentiate infections by *Klebsiella pneumoniae* and *Candida albicans*. The increased susceptibility was partly due to a decrease in reticuloendothelial-system activity as well as a reduction in the number of phagocytes, not by a direct cytotoxic effect of the opioid.

Chronic administration of morphine has also inhibited a primary antibody response of mice. These effects were worsened by naloxone (a nonaddictive analog of morphine), indicating that morphine inhibits the immune system in a specific manner—via its interaction with opioid receptors. Other studies in animals have shown that morphine can affect NK cell activity, perhaps yielding reduced resistance to tumors.

Such changes, which can also include morphine suppression of spleen and body weight, show evidence of significant changes in immune functions.

MARIJUANA

Several approaches have been used to study the effects of MARIJUANA or its active component, TETRAHYDROCANNABINOL (THC), on human immune systems. These include using cells isolated from chronic marijuana smokers, from volunteers who have been only exposed to marijuana smoke, or from nonexposed donors but exposing their cells to THC in the laboratory. A survey of chronic marijuana smokers showed that the response of their cells was depressed to stimulation with mitogens (substances that cause cell division).

Several studies have shown that neither marijuana smoking nor THC is immunosuppressive. Nevertheless, other immune alterations have been associated with marijuana or THC, including significantly reduced serum IgG levels in chronic smokers; inhibition of natural killer cell activity; inhibition of phagocytic activity; elevation of serum IgD levels; and reduced T-cell numbers. THC also inhibited DNA-, RNA-, and protein synthesis in stimulated human lymphocytes.

Studies performed in animals have produced more consistent findings than those in humans. In most cases, THC is associated with immunosuppression of various immune parameters. The greater consistency observed in animal studies probably reflects the influence of genetic factors, consistent dosage levels, and controlled diets and other conditions.

Animal studies have thus provided strong evidence of the immunosuppressive effects of THC. Such effects were clearly demonstrated when animals exposed to THC were more susceptible to infections than were others. THC has also exacerbated viral infection, as has been shown in mice and guinea pigs, and it has reduced resistance to bacterial pathogens.

Obvious differences in the susceptibility of humans versus animals to the effects of marijuana and THC will be resolved when other, more regulated research studies are carried out in immunosuppression and decreased disease resistance.

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RONALD R. WATSON

Liver (Alcohol) For all the attention being directed toward HEROIN and COCAINE, the favorite mood-altering drug in most human societies is ALCOHOL. Alcohol, in different quantities for different people, is also a toxic drug—its overconsumption taxes the body's economy, produces pathological changes in liver and other tissues, and can cause disease and death. In urban areas of the United States, just one of the complications—namely scarring or cirrhosis of the liver—is the fourth to fifth most frequent cause of death for people between the ages of twenty-five and sixty-five. In recent years, changes in liver and other tissues have been directly associated with specific steps in the metabolism of alcohol (also called ethanol or ethyl alcohol), giving some hope that rational methods can be developed for prevention and treatment.

PATHOLOGY OF ALCOHOL ABUSE

Alcohol abuse affects all organs of the body (Lieber, 1992a). It atrophies many tissues, including the brain and the endocrine glands. Indeed, altered hepatic (liver) metabolism plays a key role in a variety of endocrinological imbalances (such as gonadal dysfunctions and reproductive problems). Alcohol also exerts toxic effects on the bone marrow and alters hematological status (e.g., macrocytic anemias), and it scars the heart and other muscles. This article focuses mainly on the liver and gastrointestinal tract, since this is where alcohol penetrates into the body and has its most vicious effects; this focus will also allow exemplification of the insights and possible benefits that can be derived from the application of newly acquired knowledge in biochemistry, pathology, and molecular biology.

Liver disease, one of the most devastating complications of alcoholism, was formerly attributed exclusively to the malnutrition associated with ALCOHOLISM. Indeed, nutritional deficiencies are common in the alcoholic for various reasons, some socioeconomic, but also because alcohol is a unique compound. Alcohol is a drug, a psychoactive drug, but unlike other drugs, which have negligible energy value, alcohol has a high energy content—each gram of alcohol contributes 7.1 kilocalories, which means that a cocktail or a glass of wine will provide 100 to 150 kilocalories. Thus, alcoholic beverages are similar to food in energy terms, but, unlike food, they are virtually devoid of vitamins, proteins, and other nutrients; they act as a provider of empty calories.

As shown in Figure 1, because of its large energy load, alcohol decreases the appetite for food and displaces other nutrients in the diet, thereby promoting primary malnutrition (Lieber, 1991a). Nutrition is also impaired because alcohol affects the gastrointestinal tract. Alcohol-induced intestinal lesions, including pancreatitis, are associated with maldigestion and malabsorption, causing secondary malnutrition. Moreover, malnutrition itself will create functional impairment of the gut. Finally, alcohol (ethanol or its metabolite acetaldehyde) also adversely affects nutritional status by altering the hepatic activation or degradation of essential nutrients.

Indeed, in experimental animals, malnutrition may produce a variety of liver alterations, including fatty liver and fibrosis; however, the extent to which malnutrition contributes to the development of liver disease in the alcoholic remains unclear. Furthermore, studies conducted in the past three decades have shown that either the initial liver lesion—the fatty liver—or the ultimate stage of cirrhosis can be produced by excess alcohol, even in the absence of dietary deficiencies (Lieber & DeCarli, 1991), because ethanol (via its metabolism and/or its metabolite acetaldehyde) exerts direct hepatotoxic effects. Thus, malnutrition plays a per-

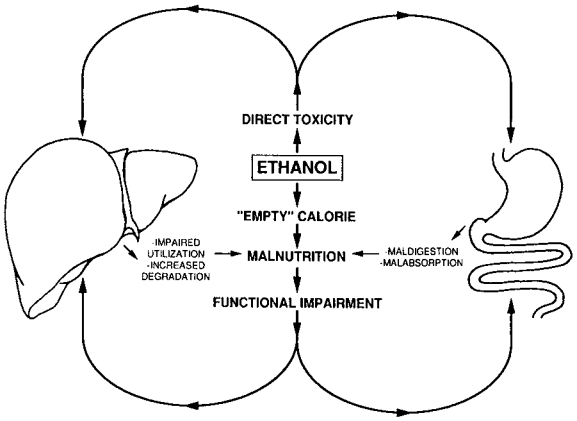

Figure 1
Interaction of Direct Toxicity of Ethanol on Liver and Gut with Malnutrition Secondary to Dietary Deficiencies, Maldigestion and Malabsorption.
SOURCE: *Lieber, C. S. (1991a). Alcohol, liver, and nutrition.* Journal of the American College of Nutrition, 10 *602–632.*

missive, but not an obligatory, role in alcohol-related somatic pathology.

METABOLISM OF ETHANOL AND SOME INTERACTIONS

Ethanol is readily absorbed from the gastrointestinal tract. Only 2 percent to 10 percent of that absorbed is eliminated through the kidneys and lungs; the rest is oxidized in the body, principally in the liver. Except for the stomach, extrahepatic (outside the liver) metabolism of ethanol is small. This relative organ specificity probably explains why, despite the existence of intracellular mechanisms to maintain homeostasis (equilibrium), ethanol disposal produces striking metabolic imbalances in the liver (Lieber, 1991b). These effects are aggravated by the lack of a feedback mechanism to adjust the rate of ethanol oxidation to the metabolic state of the hepatocyte (liver cell) and the inability of ethanol, unlike other major sources of calories, to be stored in the liver or to be metabolized or stored to a significant degree in peripheral tissues. As summarized here, the displacement by ethanol of the liver's normal substrates and the metabolic disturbance produced by the oxidation of ethanol and its products explain many of the hepatic and metabolic complications of alcoholism.

A major pathway for ethanol disposition involves alcohol dehydrogenase (ADH), an enzyme of the cell sap (cytosol) that catalyzes the conversion of ethanol to acetaldehyde. Liver ADH exists in multiple molecular forms that arise from the association in various permutations of different types of subunits. Extrahepatic tissues contain isozymes of ADH with a much lower affinity for ethanol than the hepatic isozymes; as a consequence, at the levels of ethanol achieved in the blood, these extrahepatic enzymes are inactive, and therefore, extrahepatic metabolism of ethanol is negligible, with the exception of gastric metabolism. Because of the extraordinary high gastric ethanol concentration after alcohol consumption, even the gastric ADH with low affinity for ethanol becomes active, and significant gastric ethanol metabolism ensues. This decreases the bioavailability of ethanol and represents a kind of protective barrier against systemic effects, at least when ethanol is consumed in small social-drinking amounts. This gastric barrier disappears after gastrectomy (Caballeria et al., 1989) and may be lost, in part, in the alcoholic, because of a decrease in gastric ADH (Di Padova el al., 1987).

Similar effects may also result from gastric ADH inhibition by some commonly used drugs. For example, aspirin, or some H_2-blockers such as those used in treatment of ulcers (Di Padova et al., 1992) were found to inhibit gastric ADH activity and to result in increased blood levels of ethanol when alcohol was consumed in amounts equivalent to social drinking. Women also have a lower gastric ADH activity than do men (Frezza et al., 1990); as a consequence, for a given intake, women's blood ethanol levels are higher, an increase that is compounded by their body composition (more fat, less water than men) and, on average, a lower body weight. Their higher blood ethanol levels, in turn, may therefore contribute to women's greater susceptibility to alcohol.

Alcohol dehydrogenase converts ethanol to acetaldehyde and hydrogen. Hydrogen is a form of fuel that can be burned (oxidized). Normally, the liver burns fat to produce the energy required for its own functioning but, when alcohol is present, its hydrogen displaces fat as the preferred fuel. When the liver stops burning fat and instead burns the hydrogen from the ethanol, however, fat accumulates, and a fatty liver develops, which is the first stage of alcoholic liver disease (Lieber, 1992a). Once a fatty liver has developed, fat accumulation does not increase indefinitely, even though alcohol consumption may be continued (Salaspuro et al., 1981). Fat deposition is offset, at least in part, by lipoprotein secretion, resulting in hyperlipemia—elevated amounts of fat in blood. Hyperlipemia of a moderate degree is commonly associated with early stages of alcoholic liver injury, but it wanes with the progression of liver disease (Lieber & Pignon, 1989). In some individuals, marked hyperlipemia may develop, sometimes associated with Zieve's syndrome—hemolytic anemia, fatty liver, and jaundice. This represents the potentiation, by alcohol, of an underlying abnormality in the metabolism of either lipids (essential hyperlipemia) or carbohydrates (prediabetes, pancreatitis). In addition, the degree of hyperlipemia is also influenced by the duration of alcohol intake. The capacity for a hyperlipemic response develops progressively and is accompanied by an increased activity of enzymes of the endoplasmic reticulum (within the living cells) engaged in lipoprotein production. This hyperlipemia involves all lipoprotein classes, includ-

ing high-density lipoproteins (HDL), which have been said to be involved in the protection against atherosclerosis and in the lesser incidence of coronary complications in moderate drinkers (compared to total abstainers). However, factors other than alcohol may also contribute to this apparent protection. The ability of the liver to respond with hyperlipemia reflects the integrity of the hepatocytes; its capacity decreases with the development of more severe liver injury.

Elucidation of the hepatic redox (contraction of oxidation reduction) associated with ethanol oxidation via the alcohol dehydrogenase pathway has also furthered our understanding of associated disorders in carbohydrate, purine, and protein metabolism—including hypoglycemia (low blood sugar), hyperlactacidemia (excessive levels of lactic acid in the blood) and acidosis, as well as hyperuricemia (elevated uric acid levels in blood) (Lieber, 1992a).

In addition to the enzyme ADH, alcohol is also oxidized in the liver by the enzyme system referred to as the microsomal ethanol-oxidizing system (MEOS), which involves a specific cytochrome P-450 (P450IIE1) (Lieber, 1987). Contrary to ADH, this pathway is inducible by chronic alcohol consumption. In rat livers (Lieber et al., 1988) and in human liver biopsies of heavy drinkers (Tsutsumi et al., 1989), a five to tenfold increase of this alcohol-inducible form was found. This induction represents one of the most striking biochemical differences between heavy drinkers and normals and provides an explanation for the metabolic tolerance to ethanol—a more rapid metabolism—that develops after alcohol abuse. The induction spills over to microsomal systems that metabolize other substrates, resulting in cross-tolerance to other drugs—not only sedatives and tranquilizers but also many commonly used medications such as anticoagulants and hypoglycemic agents. Thus, heavy drinkers require an increased dosage of many commonly used medications, at least at the initial stage, prior to the development of severe liver disease, which, when it develops, it will offset the enzyme induction, at which time drug dosage may have to be decreased. What complicates treatment of heavy drinkers even further is the fact that the microsomal enzymes (especially P450IIE1) also activate many xenobiotic agents (substances from outside the body) to highly toxic compounds. This explains the increased vulnerability of heavy drinkers to the hepatotoxicity of industrial solvents, anesthetics, analgesics (painkillers), and chemical carcinogens. The latter contribute to the increased incidence of various cancers in the alcoholic.

Alcohol has a major impact on gastrointestinal cancers, with a significant increase in the incidence of neoplasms of the oropharynx, the esophagus, the stomach, the liver, and the colon (Garro & Lieber, 1990). There is also activation to toxic metabolites of commonly used drugs and even over-the-counter analgesics (acetaminophen or paracetomal) (Sato et al., 1981) and vitamins, such as Vitamin A. In the heavy drinker, there are both increased breakdown and hepatic depletion of Vitamin A (Leo & Lieber, 1982), with adverse consequences. In addition, alcohol potentiates the toxicity of Vitamin A (Leo et al., 1982), which complicates supplementation with the vitamin in the presence of alcohol abuse. Alcohol abuse also promotes the microsomal breakdown of testosterone and its conversion to estrogens, which, together with testicular toxicity and decreased testosterone production, results in hypoandrogenism—the loss of masculinity (Lieber, 1992a).

In addition to environmental factors, there are individual differences in rates of ethanol metabolism that appear to be genetically controlled, and the possible role of heredity in the development of alcoholism in humans has been emphasized. The induction of the MEOS pathway also leads to increased conversion of alcohol to acetaldehyde, a highly reactive and thus potentially toxic compound.

TOXICITY OF ACETALDEHYDE

Acetaldehyde causes injury through the formation of adducts with proteins, resulting in antibody formation, inactivation of many key enzymes, decreased deoxyribonucleic acid (DNA) repair, and alterations in cell structures such as microtubules, mitochondria, and plasma membranes (Lieber, 1988, 1992a). Acetaldehyde also promotes synthesis of hepatic collagen—the key protein of scar tissue; furthermore, it causes glutathione depletion, thereby exacerbating the toxicity mediated by free radicals, which results in lipid peroxidation and other tissue damage (Lieber, 1991b). Because of the far-reaching toxicity of this metabolite of ethanol, some of the liver cells die; this attracts inflammatory cells, which results in the more severe stage

of alcoholic hepatitis, one of the precursors to the ultimate scarring or cirrhosis.

Once there is cirrhosis, a number of complications ensue, including obstruction of blood flow—with portal hypertension (elevated pressure in the veins leading from the intestine to the liver) and internal, life-threatening bleeding of distended veins, so-called varices. There is also a buildup of water in the abdominal cavity, so-called ascites (Lieber, 1992a).

Acetaldehyde is particularly elevated if drinking occurs in pregnancy; it crosses the placenta (Karl et al., 1988) and has been incriminated in the pathogenesis of the FETAL ALCOHOL SYNDROME (FAS), the most common preventable cause of cogenital abnormalities.

The bulk of acetaldehyde is oxidized to acetate by an acetaldehyde dehydrogenase of the liver mitochondria. Lack of the active form of the enzyme in some Asians explains their high blood acetaldehyde and flushing reaction after alcohol. DISULFIRAM (Antabuse—a drug used in recovering alcoholics) is an inhibitor of acetaldehyde dehydrogenase. It raises the acetaldehyde levels after drinking and thereby causes flushing and several adverse effects that can be utilized to sustain abstinence in patients motivated to take the compound.

TREATMENT AND CONCLUSION

Alcoholics suffer commonly from malnutrition. Therefore, nutritional deficiencies, when present, should be corrected—although such efforts were found to be ineffective in fully preventing liver disease in view of the intrinsic toxicity of ethanol (Lieber, 1991b; Lieber & DeCarli, 1991).

Although progress is being made at offsetting the direct toxicity of ethanol through chemical means (Lieber, 1992b), at present, the single fully effective way of preventing somatic alcoholic injury remains control of the toxic agent—ethanol—through control of consumption. Full abstinence is required in those who are genetically (or otherwise) prone to develop craving or to exhibit dependence, or those who are predisposed to develop the major somatic complication with chronic use of alcohol.

For the others, moderation is recommended. What is considered "moderate" or "excessive" has been the subject of debate. One view is that on the average, moderate drinking represents no more than one drink a day in women and no more than two drinks a day in men—with a drink being 12 ounces of regular beer, 5 ounces of wine, or 1.5 ounces of distilled spirits (80 proof) (Dietary Guidelines, 1990). It is important, however, that "excess" be defined individually, taking into account not only gender, but also heredity and personal idiosyncrasies.

(SEE ALSO: *Addiction: Concepts and Definitions; Alcohol: Complications; Cancer, Drugs, and Alcohol; Complications: Liver Damage; Social Costs of Alcohol and Drug Abuse*)

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REVISED BY RALPH MYERSON

Liver Damage (Other Drugs) The liver is the largest organ of the human body, normally weighing about 3.3 pounds (1.5 kg). It occupies the right upper quadrant of the abdominal cavity just below the diaphragm. As befitting its anatomical prominence, its function is essential to maintain life. If we surgically removed the entire liver from any animal (including humans), it will fall into a coma shortly and die. The absence of a certain critical mass of functioning liver tissue is incompatible with life. While the human liver has a remarkable resilience and regenerative capacity after injury or illness, this is true only up to a certain point. If illness pushes the liver beyond the "point of no return," the person dies.

The liver has a multitude of complex functions and is justly called the "laboratory" of the human body. It secretes a digestive juice into the intestine, called bile; it produces a number of essential proteins, clotting factors, and fatty substances; it stores and conserves energy-producing sugars; it detoxifies both internally produced and external toxins and drugs that would otherwise be poisonous to the human organism—just to name some of its important functions.

What can seriously jeopardize this very important organ and consequently the well-being and survival of the individual? For one, there are diseases—both congenital and acquired—over which we have little or no control, such as some genetically determined and developmental abnormalities, circulatory liver problems, certain tumors, and infections.

A very large part of hepatology (the technical term to describe the study and treatment of liver diseases) is, however, devoted to liver problems created by a peculiar human behavior—the abuse of ALCOHOL and drugs. Whereas discussions as to whether ALCOHOLISM and DRUG ABUSE are truly self-inflicted problems elicit a variety of opinions, the liver disease that results from substance abuse in a given individual could have been avoided if the substance-abusing behavior had not occurred. Beyond the psychosocial consequences of substance abuse, diseases of the liver (and brain) represent the major COMPLICATIONS of alcohol and drugs.

The morbidity (disease incidence) and mortality (death incidence) from alcoholic and drug-induced liver injury are very high. In the scientific literature, it is well established that the mortality from alcoholic liver disease is correlated with the per capita alcohol consumption; in fact, the prevalence of alcoholism in a given society has been calculated from liver mortality statistics. While alcohol is a direct liver toxin, most of the other commonly abused psychoactive substances are generally not known to affect the liver directly to a great extent; their major contribution to liver morbidity and mortality is via exposing people to viral hepatitis—a potentially fatal disease.

ALCOHOLIC LIVER DISEASES

Another article in this encyclopedia discusses the relationship between alcohol and the liver in great detail. In any article dealing with the effects of drugs on the liver, however, alcohol must be addressed.

The gamut of alcoholic liver diseases and their interrelationship is illustrated in Figure 1.

Alcoholic Fatty Liver. Fat accumulation in the liver is an almost universal response to excessive alcohol consumption. It occurs in the majority of heavy drinkers. How and why fat accumulates in liver cells is complicated and not completely understood; but we know for sure that it happens. If you examine a piece of biopsied liver tissue from an alcoholic under the microscope, you see that many liver cells are loaded with big bubbles consisting of fat, almost totally occupying the cell. In most cases, this fatty change does not matter too much as far as the patient's health is concerned. It is an almost invariable response to too much alcohol consumption and an early warning. The person who has nothing worse than an alcoholic fatty liver may not feel sick at all, and only if a biopsy is done can the fatty liver be diagnosed. The doctor may feel an enlarged liver by palpation, which may be a bit tender. The laboratory test may show a slight elevation in the blood of some liver enzymes, best known by their initials: SGOT (or AST) and SGPT (or ALT). These enzymes are elevated because some of them tend to leak out of the fatty liver cells into the blood.

If a person stops drinking, the fat disappears from the liver cells, the swelling subsides and the AST and ALT levels become normal. The two-way arrow in the diagram of Figure 1 indicates that fatty liver is reversible with abstinence, and the condition may fluctuate back and forth between normal and fatty liver with abstinence and drinking, respectively. Thus, this, per se is not likely a serious situation; it is an early warning that "your liver does not like alcohol" and that possibly worse things might yet come. There was a time when fatty liver was regarded as the precursor of the end-stage liver disease called cirrhosis (indicated by the broken arrow and question mark on Figure 1), but in the 1990s, most physicians do not believe that this direct connection exists.

Alcoholic Hepatitis. This is a potentially more serious form of alcoholic liver disease. A certain proportion of alcoholics, in addition to accumulating fats in their livers when drinking, will develop inflammation (hepatitis means liver inflammation)-consisting of an accumulation of white blood cells, the death (necrosis) of some of the liver cells, and the presence of some very char-

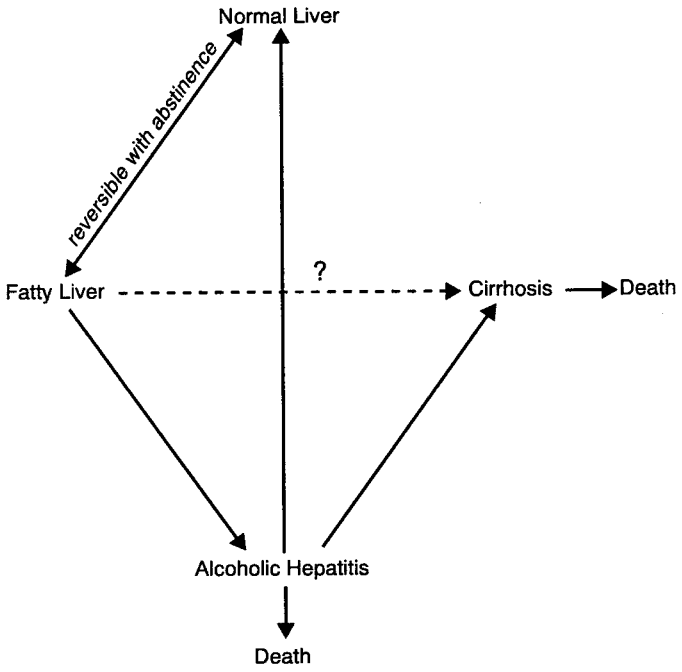

Figure 1
Interrelationships between Various Forms of Alcoholic Liver Disease

acteristic material (called Mallory bodies). Again, all this can be seen under the microscope in a biopsied piece of tissue.

The clinical picture of alcoholic hepatitis can be very variable. At one extreme is the person who feels perfectly well and only the biopsy could tell that something is wrong. At the other extreme is the patient with a swollen and painful liver, yellow jaundice (a yellowing of the entire body from bile pigment leaking into the blood), fever, and disturbed consciousness—who dies. Between these extremes are people with varying degrees of seriousness of the illness; for example, with or without some jaundice, with or without pain and fever, etc. The blood's white cell count is usually elevated. The bilirubin (bile pigment) level may be elevated in patients who are yellow (a pale to deep mustard). The liver enzymes are higher than normal in the blood, because they leak out of the inflamed liver cells. However, these values are not as high as in viral hepatitis and, characteristically, in alcoholic hepatitis AST (SGOT) is higher than ALT (SGPT), which helps to distinguish alcoholic hepatitis from viral hepatitis (difficult to do at times). In viral hepatitis not only are the absolute enzyme values higher, but the ratio is reversed: ALT is higher than AST.

Thus, the outcome of alcoholic hepatitis can be death (worst scenario) or recovery (best scenario)-as shown on Figure 1. Repeated episodes of drinking and alcoholic hepatitis, however, even if the patient does not die in a given episode, can lead to the endstage of alcoholic liver disease: cirrhosis.

Alcoholic Cirrhosis. In terms of histology (tissue damage) this indeed is an end-stage disease: a cirrhotic liver *cannot* become normal; in Figure 1, there is no arrow between cirrhosis and normal liver. Clinically, cirrhosis is a serious disease, potentially fatal, but not inevitably so. Alcoholism is not its only cause, but it is by far the most common.

Under the microscope a cirrhotic liver shows a disorganized architecture: the dead (necrotic) liver cells have been replaced by scar tissue. The liver tries to repair itself: In a somewhat haphazard fashion it attempts to produce new liver tissue in the form of nodules, which are separated from each other by scar. These newly formed liver nodules may indeed sustain liver function and thus life for a time, but at a price: the liver's blood circulation is mechanically compressed. Thus, the pressure increases in the blood vessels leading to the liver. Some of these overloaded blood vessels, especially those on the border of the stomach and esophagus (called esophageal varices), can rupture any time, causing a major hemorrhage.

Those who develop the cirrhotic stage of alcoholic liver disease present their symptoms in various ways. Some of them look quite normal and only the biopsy will reveal the presence of cirrhosis. Others are jaundiced, the yellow color coming from bile pigment leaking out of the damaged liver into the blood, thus staining the skin and the whites of the eyes. Still others have large fluid accumulations in their extremities (edema) or in their abdominal cavity (ascites); the latter may make these patients—men or women—look like they are nine months pregnant. Some may vomit blood, because of the hemorrhaging. In most advanced cases, there is just not enough functioning liver tissue left; the liver no longer can perform its "laboratory" function, and the person slips into a coma and may die.

When cirrhotic patients are examined by doctors, their livers do not feel smooth on palpation, but bumpy from all those nodules that formed. At first the liver may be swollen and enlarged, but at the later stages it shrinks. The ultrasound picture suggests a patchy, disorganized architecture of the liver. The spleen may enlarge from the increased pressure in the blood vessels. The liver enzymes (AST and ALT) may be moderately elevated as in other forms of alcoholic liver disease, but this has no prognostic importance. More ominous signs pointing toward severely compromised liver functions are the following: a decrease in blood level of albumin (an important protein manufactured by the liver), deficiency in blood-clotting factors that are also made in the liver, and the presence of anaemia (low hemoglobin and red blood cell count).

What Can Kill a Cirrhotic Patient? Ascites (fluid accumulation in the abdomen) is very uncomfortable and unsightly but, by itself, usually does not kill—unless it gets spontaneously infected, which is always a threat. Generally, cirrhosis compromises the immune system, rendering cirrhotic alcoholics susceptible to all sorts of potentially overwhelming infections. Portal hypertension is a serious complication of the cirrhotic fibrosis. The obstruction to portal vein flow through the liver results in the development of other vein channels to accommodate the return of blood from the abdominal organs which comprise the blood in the portal vein. The

result is the development of varices (enlarged, engorged veins) in the stomach and esophagus. These enlarged, thin-walled veins are prone to rupture leading to one of the most serious complications of cirrhosis of the liver—bleeding varices. This constitutes an emergency and calls for immediate intervention in the form of measures to control the bleeding. A variety of therapies are available, all of which have all been employed with a varying degree of success that depends on the severity of the hemorrhage and the skill and experience of the physician. Once the bleeding has been controlled, the patient should be considered for an appropriate permanent venous shunt procedure whereby venous blood bypasses the liver. Finally, total decompensation of liver-cell function may cause coma and death.

The good news is that even when there is irreversible cirrhosis at the tissue level, death may not be inevitable. Survival depends mainly on two factors: luck and alcohol abstinence. Abstaining alcoholics with cirrhosis can stabilize and survive on what's left of their liver tissue without necessarily and relentlessly progressing to one of the fatal outcomes. A famous Yale University study many years ago showed clearly the correlation between abstinence and survival in cirrhosis.

Who Gets Which Alcoholic Liver Disease? There are still no certain answers to this question. Fatty liver is an almost universally predictable response to heavy alcohol consumption, but this by itself is seldom a serious problem. A smaller number of people develop alcoholic hepatitis and still fewer (variously estimated in different populations between 5 to 25% of alcoholics) end up with cirrhosis. Considering the large number of alcoholics in our society, the minority who develop cirrhosis still represents huge numbers; cirrhosis *is* one of the leading causes of all deaths.

Still, why do some alcoholics develop alcoholic hepatitis and cirrhosis, while others who drink equally heavily do not? The amount of alcohol consumption and the length of time of heavy drinking is certainly one risk factor. Gender may be another: Women's livers generally are more vulnerable to the effects of alcohol than those of men, given equal alcohol exposures. Finally, there may be a genetically determined (but still unclarified) individual susceptibility, which may explain why some people never get cirrhosis, why some do after many years of alcoholism, and why still others get cirrhosis at a young age or after a relatively short drinking career.

Prognosis and Treatment. The issues of prognosis and treatment cannot be separated from each other. The cornerstone of treatment is complete abstinence from alcohol; achieving abstinence can arrest the progression of liver disease, even in established cirrhosis. Continued drinking leads to deterioration and death.

One therapeutic issue relating to alcoholism itself, should be addressed here because it is relevant to liver disease. The drug DISULFIRAM (Antabuse) is sometimes prescribed to reinforce abstinence: its unpleasant, sometimes severe interaction with alcohol is used as a deterrent against drinking. Since disulfiram (as so many other drugs) has been occasionally reported to produce liver toxicity of its own, the presence of alcoholic liver disease is sometimes regarded as a relative contraindication against the prescription of disulfiram. The liver toxicity caused by alcohol far outweighs any risk that may be caused by disulfiram.

Are there any other treatment techniques available beyond abstinence that can help the recovery from alcoholic liver damage? In the late 1980s, a Toronto research group reported the beneficial effect of propylthiouracil (PTU). This is a drug normally used for the treatment of thyroid disease, but by reducing oxygen demand in the body (including in the liver), it might help to repair the damage caused by alcohol. The early results were promising but it is still not a widely accepted treatment. Other drugs, such as corticosteroids (to decrease inflammation) or colchicine (to decrease scar formation) have dubious value.

There are relatively effective treatments available for some of the complications of alcoholic liver disease so that the patient may survive and thus begin his or her abstinence program. The fluid accumulation in the extremities (edema) or in the abdomen (ascites) can he helped by diet modifications (salt restriction), water removing drugs (diuretics), albumin infusion, or tapping the abdomen. Infections can be treated with antibiotics. The brain syndrome of liver failure (so-called hepatic encephalopathy or, in severe cases, hepatic coma) can improve with dietary means (protein restriction) or some drugs (e.g., neomycin, lactulose). The potentially or actually bleeding esophageal varicose veins can be obliterated by certain injections through a gastroscope (so-called sclerotherapy),

and the bleeding risk can be lessened by beta-blocking drugs or some surgical procedures to decrease pressure.

Finally, in the 1990s we have the possibility of liver transplantation. If all else fails, a successful liver transplant cures alcoholic liver disease. Apart from the general problems of donor matching and supply, some people have raised objections on ethical grounds to offering transplantation for alcoholic (i.e., "self-inflicted") liver disease. This is not an acceptable objection and goes against medical ethics. Well-motivated recovering alcoholics are entitled, as much as anybody else, to a life-saying procedure. In fact, studies have shown that the very dramatic and heroic nature of this operation may be an extremely powerful motivator for future abstinence by liver recipients. Numerous successful transplants have been carried out on alcoholics.

DRUGS AND THE LIVER

There are many drugs in medicinal use that can have direct liver toxicity. Peculiarly, most of the psychoactive drugs that people tend to abuse are not known to be particularly harmful to the liver. Occasional liver damage has been reported with SOLVENT sniffing and COCAINE use, but this is not a common problem. Narcotics (opioids), anti-anxiety, and other sedative drugs (such as BARBITURATES), MARIJUANA, and HALLUCINOGENS do not usually cause liver injury.

There are, however, several relevant secondary issues concerning drug abuse and the liver. For one, a damaged liver (for example from alcohol or hepatitis) results in poor tolerance of SEDATIVES, because good liver function is necessary to eliminate sedatives properly; impaired liver function can therefore result in exaggerated sedative effect. Conversely, some sedatives, notably BARBITURATES (which were often abused in the past and sometimes still are), actually stimulate ("induce") certain liver enzymes, which can result in increased elimination (i.e., decreased effect) of another therapeutically necessary drug. For example, a barbiturate user (or abuser) may have poor effect from a clotting preventative (anti-coagulant) drug that is necessary in heart disease or after a stroke. Some drugs do the opposite—they inhibit liver enzymes. For example, the anti-ulcer drug cimetidine (Tagamet), which per se has no PSYCHOACTIVE effect, can cause such enzyme inhibition; if a person at the same time also happens to use or abuse a sedative, the sedative can have an exaggerated effect.

Generally speaking, the normal liver transforms or inactivates drugs (detoxification) to less active or harmless forms. A notable and important exception is acetaminophen, one of the most commonly used medications against PAIN and fever (e.g., the various Tylenol preparations). The liver can transform acetaminophen into a toxic metabolic derivative that might cause a potentially lethal, acute liver injury. Generally, this does not happen at ordinary therapeutic acetaminophen dose levels. In the case of an acetaminophen overdose, however, such severe liver toxicity can occur that a person will die within days. Most of such overdoses are, of course, suicidal attempts.

Acetaminophen itself does not have any psychoactive (mind-altering) properties; thus people do not abuse it for such reasons. Many marketed acetaminophen preparations, however, are combined with CODEINE (a NARCOTIC). People seeking narcotic "highs" from such preparations might ingest them in large enough quantities to subject themselves to potentially severe liver injury. It is the codeine they are after, but it is the bystander acetaminophen that may kill them. There is an antidote against acetaminophen poisoning (called acetylcysteine), but it is effective only if it is given within a few hours (less than a day) after the ingestion of the drug. The person who is overdosing with a suicidal intent is more likely to be discovered and brought to quick medical attention than an unintentionally overdosing drug abuser. An additional issue in the acetaminophen story is that there is strong evidence of increased risk when alcohol and acetaminophen are combined. In alcoholics, relatively low, even therapeutic, doses of acetaminophen can cause severe and potentially fatal liver damage.

Apart from acetaminophen, direct liver toxicity is not a major feature of substance abuse except in the case of alcohol. The liver disease that is *very* commonly associated with drug use is viral hepatitis (liver inflammation) which is not caused by the drugs themselves but by infection with a virus. It is then transmitted from person to person through contaminated needles and syringes. The problem of viral hepatitis, then, is largely that of injecting drug users (IDUs).

VIRAL HEPATITIS IN DRUG ABUSERS

In the mid-1990s, at least five types of disease-causing hepatitis viruses have been identified, and they are designated by the letters of the alphabet, A–E. Table I summarizes some of their important characteristics. Of the five, hepatitis A and E are not particularly associated with injecting drug abuse; but the other three very much are and they will be discussed in some detail in that context.

Hepatitis B. This virus (which used to be called "serum hepatitis") is endemic to some parts of the world, such as Southeast Asia, where as much as 10 percent of the population may be infected. In the Western world, IDUs represent the greatest reservoir for hepatitis B virus. It is transmitted through a direct blood-borne route, such as (1) contaminated needles and syringes (which drug addicts notoriously did not sterilize in the past); (2) from an infected mother across the placenta and through the umbilical cord of a developing FETUS; (3) from blood contaminating accidental needle-stick injuries in health-care workers; and (4) from any blood to blood contact occurring during sexual intercourse. At one time blood transfusions were a common source of infection, but since the 1970s we have had a reliable test to screen out infected donors.

The symptoms of hepatitis B infection vary. In its severest form, it can cause general unwellness, fever, jaundice, coma, and death. The majority of patients, even with marked jaundice and fever, do not die. Many infected people do not even have an overt illness; they may not feel sick at all or may just have transient "flu-like" symptoms. There may be a tender enlargement of the liver. If such people are tested in the laboratory, they have elevated enzymes, such as AST (also known as SGOT) and ALT (also known as SGPT), which are usually much higher than the values found in alcoholic liver disease (in contrast to alcohol, viral hepatitis tends to cause more elevation in ALT than in AST). The bilirubin (bile pigment) level will be high if the person has yellow jaundice.

There are now quite good serological tests for hepatitis B. A virus particle (hepatitis B's antigen) can be identified in infected people. Those who recover from the illness and clear the virus out of their body will develop a protective antibody that will prevent their reinfection. The antibody can be detected in a laboratory test.

The majority of people who get infected with hepatitis B do recover and acquire protective antibodies. A sizable minority of those who survive, however, perhaps 10 percent, will continue to carry the virus (remain "antigen positive"); and some of these will have a chronic liver inflammation that can end up in cirrhosis. The cirrhosis caused by hepatitis B is essentially similar to alcoholic cirrhosis, with the same consequences and potential complications described above. Moreover, hepatitis B has the potential to cause liver cancer in some of those who develop cirrhosis. Not only is hepatitis B in such chronically infected individuals a threat to their own survival, but it is also a source of infection to others, particularly to their needle-sharing partners, to their sexual partners, and to their developing fetuses and newborn babies.

Hepatitis C. Until about 1990, this was called "non-A-non-B hepatitis," because we knew that there were viral hepatitis cases that were caused by neither of the two identifiable viruses, A or B. An antibody test can now identify this virus, which is called hepatitis C. The antibody detected is not a protective antibody, but it is similar to the AIDS (HIV) antibody in that it indicates the presence of the virus. A lot of the viral hepatitis caused by blood transfusions in the past was due to hepatitis C infection; the antibody test can eliminate this source of transmission, since it is used to screen the donor blood supply.

Injecting drug users, however, remain a major reservoir and source for the spread of this virus. Hepatitis C is transmitted similarly to hepatitis B—and, for that matter, to HIV—primarily through direct blood to blood contact (by contaminated injection PARAPHERNALIA) and to a lesser extent, but still possibly, via sex and from mother to fetus. The primary infection goes very often unnoticed. The laboratory tests, in addition to hepatitis C antibodies, will show elevated ALT and AST levels. Since this is a newly identified virus, the natural history of hepatitis C is not yet clear. A fair amount of evidence suggests that chronic hepatitis, eventual cirrhosis, and liver cancer may be an even greater risk with hepatitis C than it is with hepatitis B. Some studies in the current medical literature indicate that 50 to 80 percent of intravenous drug addicts may be positive for hepatitis C, so we are not talking about a trivial problem here.

Hepatitis D. This is a very peculiar virus, which was originally called "delta agent" and later

renamed hepatitis D. It is an incomplete virus that can exist only in the presence of hepatitis B. When the two organisms combine, the outcome is a particularly nasty, potentially lethal hepatitis, both in terms of acute mortality and chronic consequences. Discovered in Italy about 1990, in North America hepatitis D is known to be primarily harbored by the injection drug-using population.

Prevention and Treatment of Viral Hepatitis. Obviously the best prevention for injection drug users would be to stop injecting drugs. Other, often more realistic prophylactic measures—which are now all familiar from the HIV scene—are the use of sterile (or at least bleached) needles and syringes, needle-exchange programs, and condoms for sexual activities.

Immediately after a known or suspected exposure to hepatitis B, the injection of an antibody preparation (known as "hepatitis B immune globulin") can prevent illness. A more permanent prophylaxis in high-risk populations is provided by the hepatitis B vaccine, which gives long-term immunity in previously uninfected individuals. IDUs certainly represent one of these high risk populations, although the widespread use of the hepatitis B vaccine in this group raises some obvious ethical and logistic dilemmas. At the present time, there is no passive or active immunization available for hepatitis C.

The acute phase of any form of viral hepatitis cannot be treated effectively. Chronic hepatitis B and C infection may respond, to a certain extent, to some antiviral drugs known as interferons, which are currently widely studied. Finally, as mentioned under alcoholic liver disease, the most radical form of therapy in the end-stages is liver transplantation.

(SEE ALSO: *Complications: Liver (Alcohol); Needle and Syringe Exchanges; Social Costs of Alcohol and Drug Use; Vulnerability As Cause of Substance Abuse*)

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Medical and Behavioral Toxicity Overview Alcohol and other drugs of abuse have caused and continue to cause considerable adverse health effects to both the individual and to society. Both legal and illegal drugs (substances) of abuse are taken to modify mood, feeling, thinking, and perception. As with most drugs (medications), both acute and chronic toxicities occur. In general, the term *acute* refers to the short period of time when the drug is present in the body, exerting its main effects. The term *chronic* refers to a longer term, usually years.

Acute toxicity results in the impairment of behavior leading to other complications (e.g., trauma) and, in the case of some drugs, high doses can decrease breathing (respiratory depression) or change the rhythm of the heart, leading to accidental or intentional death. Chronic use can result in organ damage, which may lead to chronic illness or death (as with alcoholic cirrhosis of the liver). Persistent use of many classes of drugs also leads to TOLERANCE (an increased amount is required to produce the same effects) and physiologic (physical) dependence, so that a WITHDRAWAL syndrome is associated with sudden cessation of drug use. Drug users who employ hypodermic needles and syringes (injecting drug users [IDUs]) are at risk for blood-borne diseases associated with the use of unsterile equipment, such as hepatitis and human immunodeficiency virus (HIV 1 and 2—the viruses responsible for AIDS; see ACQUIRED IMMUNODEFICIENCY SYNDROME).

This article focuses on ALCOHOL as the representative drug, but other drugs of abuse will be referred to where appropriate. In North America, diagnosis of alcohol and other psychoactive substance abuse/dependence is usually made according to the DIAGNOSTIC AND STATISTICAL MANUAL (DSM) of the American Psychiatric Association (APA). The fourth edition, called DSM-IV, defines psychoactive substance *dependence* as at least

three of the following (occurring in the same 12-month period):

1. tolerance, as defined by either of the following:
   a. need for markedly increased amounts of the substance to achieve intoxication or desired effect
   b. markedly diminished effect with continued use of the same amount of the substance
2. withdrawal, as manifested by either of the following:
   a. the characteristic withdrawal syndrome for the substance
   b. the same (or closely related) substance is taken to relieve or avoid withdrawal symptoms
3. the substance is often taken in larger amounts or over a longer period than was intended
4. a persistent desire for or unsuccessful efforts in cutting down or controlling substance use
5. a great deal of time is spent in activities necessary in obtaining the substance (e.g., visiting multiple doctors or driving long distances), using the substance (e.g., chain-smoking), or recovering from its effects
6. important social, occupational, or recreational activities are given up or reduced because of substance use
7. continued substance use despite knowledge of having had a persistent or recurrent physical or psychological problem that was likely to have been caused or exacerbated by the substance (e.g., recurrent cocaine use despite recognition of cocaine-induced depression; continued drinking despite recognition that an ulcer was made worse by alcohol consumption)

The diagnosis of alcohol and other substance *abuse* (as opposed to dependence) relies on:

A. A maladaptive pattern of substance use leading to clinically significant impairment or distress as manifested by one or more of the following occurring at any time during the same twelve-month period:
   1. recurrent substance use resulting in a failure to fullfill major obligations at work, school, or home (e.g., repeated absences or poor work performance related to substance use; substance-related absences, suspensions, or expulsions from school; neglect of children or household)
   2. recurrent use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by substance use)
   3. recurrent substance-related legal problems (e.g., arrests for substance-related disorderly conduct)
   4. continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with family members about consequences of intoxication; physical fights)

B. Has never met criteria for Substance Dependence for this class of substance.

These criteria continue to evolve and are likely to be somewhat changed in the future. Clearly the lack of one of the above diagnoses does not preclude a given person from being at risk for complications of alcohol or drug use (e.g., trauma as a result of intoxication).

THE ACUTE EFFECTS OF ALCOHOL

At the level of the cell, very high doses of alcohol (ethanol) seem to act by disrupting fat (lipid) structure in the central nervous system (anesthetic effect). Lower doses are thought to interact with various proteins and NEUROTRANSMITTERS (which act as RECEPTORS), such as GLUTAMATE, GABA (GAMMA-AMINO BUTYRIC ACID), cyclic AMP (adenosine mono-phosphate), and G proteins. Other actions may involve ion (calcium) channels. The reinforcing (rewarding) effects of alcohol may be mediated via DOPAMINE (a neurotransmitter) in specific brain regions; dopamine acts as an intermediary compound in the reinforcement process. The reinforcement of responses to other drugs of abuse, such as COCAINE, are also thought to be mediated via dopamine.

For most persons at least some of the acute effects of alcohol are well known on the basis of personal experience. Low doses cause blood vessels

to dilate. The skin becomes flushed and warm. There is relaxation and mild sedation. Persons become talkative with loss of inhibitory control of emotions. Small doses (one to two drinks) do not impair complex intellectual ability; however, as the dose increases (two or more drinks or as the blood alcohol concentration approaches and exceeds the legal limit) impairment at multiple levels of the nervous system occurs. All types of motor performance are eventually affected, including maintenance of posture, control of speech, and eye movements. These movements become slower and more inaccurate. There is a decrease in mental functioning, such that there is impairment in attention and concentration, and a diminishing ability to make mental associations. There is a decreased ability to attend to incoming sensory information. Night and color vision are impaired. Judgment and discrimination and the ability to think and reason clearly are adversely affected. Even higher doses result in a stuporous condition associated with sleeping, vomiting, and little appreciation of surroundings. This is followed by coma and sometimes death from decreases in the functioning of the brain centers that control respiration.

DOES ALCOHOL IN MODERATION HAVE A BENEFICIAL EFFECT?

The impact of alcohol has been further enhanced recently by an impressive amount of evidence from epidemiological and clinical case-control and cohort studies over the last two decades demonstrating an inverse relationship between moderate alcohol consumption and coronary heart disease. Individuals find themselves caught in a dilemma between the oft-preached dangers of drinking and its recently acclaimed benefits.

Early investigators, impressed by France's relatively low incidence of coronary heart disease despite an intake of saturated fats at least three times that of the United States (the so-called "French paradox"), focused their studies on the potential cardioprotective properties of red wine. Based on other studies, however, the present consensus is that all alcoholic beverages—wine, beer, and liquor, in moderation, are associated with a lower coronary artery disease risk (Rimm et al., 1996). In dose-range studies, a J or U shaped curve has been demonstrated whereby the equivalent of two alcoholic drinks per day is associated with a decreased incidence of coronary heart disease compared with no drinks, while higher doses result in an increased risk of infarction as well as the well-known problems produced by alcohol excess.

Inasmuch as most American households already are exposed to alcohol (Thun et al., 1997). advice as to the benefits of moderation may be offered without reserve. In the case of abstainers, however, the risks of initiating alcohol appear to outweigh its potential benefits. This is especially applicable in families that include adolescents.

ACUTE EFFECTS OF OTHER DRUGS OF ABUSE

Other drugs of abuse can be classified into stimulants, depressants, OPIOIDS, and drugs that alter perception (including HALLUCINOGENS). The effects of any drug depends on the dose taken at any one time, the previous drug experience of the user, the circumstances in which the drug is taken, and the manner (route of administration) in which the drug is taken.

Stimulants such as cocaine and AMPHETAMINE produce euphoria, increased confidence, increased sensory awareness, increased ANXIETY and suspiciousness, decreased appetite, and a decreased need for sleep. Physiological effects include increases in heart rate, blood pressure, and pupil size, and decreases in skin temperature.

Depressants such as the minor tranquillizers (including the BENZODIAZEPINES, BARBITURATES, and other SEDATIVE-HYPNOTICS) produce acute effects of a similar nature to alcohol—also a depressant. Actual effects vary according to drug, so that benzodiazepines (such as diazepam/Valium) produce less drunkenness compared to alcohol or barbiturates.

The term opioid refers to both drugs derived from OPIUM (opiates) and other synthetic drugs with similar actions—those acting on the same receptor system. The term *NARCOTIC* is usually synonymous with opioid, but it can technically also include other drugs included in the HARRISON NARCOTIC ACT (e.g., cocaine). Opioids produce euphoria, sedation (to which rapid tolerance develops), itching, increased talkativeness, increased or decreased activity, a sensation of stomach turning, nausea, and vomiting. There are minor changes in blood pressure and the pupils become constricted (made smaller).

Drugs that alter perception include those above as well as MARIJUANA, PHENCYCLIDINE (PCP), and LYSERGIC ACID DIETHYLAMIDE (LSD). In general, most drugs of abuse can cause hallucinations under some circumstances. The drugs which more specifically affect perception (hallucinogens) produce a combination of depersonalization, altered time perception, body-image distortion, perceptual distortions (usually visual), and sometimes feelings of insight. Physiological effects such as changes in heart rate and blood pressure may also occur.

HARMFUL EFFECTS

Accidents. Alcohol is a significant factor in accident-related deaths. The main causes are motor-vehicle ACCIDENTS, falls, drownings, and fires and burns. Approximately 50 percent of motor-vehicle fatalities (driver, pedestrian, or cyclist) in the United States are alcohol-related, with the incidence having fallen a little in recent years. These alcohol-related accidents are more common at nights and on weekends. Falls are the most frequent cause of nonfatal accidents and the second most frequent cause of fatal accidents. According to various surveys of fatal falls, those that are alcohol-related range from 17 to 53 percent; for nonfatal falls, from 21 to 77 percent (Hingson & Howland, 1987). The higher the blood alcohol content (BAC), the higher is the risk for falls. The third leading cause of accidental death in the United States is drowning. About half (47–65%) of adult deaths by drowning are alcohol-related (Eighth Special Report to U.S. Congress, 1993). Fires and burns are the fourth leading cause of accidental death in the United States. Studies on burn victims show that alcohol intoxication is common. Cigarette smoking while drinking is an additional cause of fires and burn injuries. Estimates of the rates of intoxication range from 37 to 64 percent.

Users of other drugs of abuse (e.g., cocaine and opioids) also have higher rates of accidents in comparison to the nondrug-abusing population. The combination of cocaine and alcohol has been reported to be commonly associated with motor-vehicle deaths. Between 1984 and 1987 in New York City, 18 percent of motor-vehicle deaths showed evidence of cocaine use at autopsy. Cigarette smokers have higher rates of accidents than do non-smokers. Drugs that alter perception, such as PCP, are also associated with accidents mostly related to an impaired sense of judgment.

Crime. Associations between criminal activity and alcohol use have been established; however, methodological inadequacies of studies in this area preclude a clear causal relationship between alcohol use and crime. The strongest association between crime and alcohol use occurs in young males. Other forms of drug abuse (e.g., HEROIN and cocaine) have much higher associations with criminality. For example the majority of persons enrolled in METHADONE programs have extensive criminal careers. Those involved in drug dealing are at a high risk of being both a perpetrator or a victim of homicide.

Family Violence. Several studies indicate an association between alcohol use/abuse and spousal abuse; however, the nature of this interaction is not well understood. Intoxication is associated with an increase in negative behavior for episodic drinkers while less negative behavior is seen in steady drinkers, suggesting that drinking may be a short-term solution to problems for regular drinkers. Clearly, alcohol use is associated with physical VIOLENCE in some families, and there also appears to be a link between alcohol and child abuse. Female caregivers with a diagnosis of alcohol abuse, alcohol dependence, recurrent depression, or ANTISOCIAL PERSONALITY are more likely to report physical abuse of their children than those without these diagnoses (Bland & Orn, 1986).

Suicide. From 20 to 36 percent of SUICIDE victims have a history of alcohol abuse or had been drinking prior to death. Alcohol use is linked more to impulsive than to premeditated suicides, and to the use of firearms, rather than to other modes of killing. Death from OVERDOSE of illicit drugs is common; most of these are thought to be accidental but some are intentional.

Trauma or Severe Injuries. A history of trauma has been found to be a marker for (sign of) alcohol abuse. Emergency room trauma victims often have high rates of intoxication. Furthermore, heavy alcohol use both interferes with recovery from serious injuries and increases rates of mortality for a given injury. Users of illicit drugs have a higher age-adjusted rate of mortality than do nonusers. Many of these deaths result from trauma.

Fetal Alcohol Syndrome (FAS). Since the 1970s, alcohol has become firmly established as a teratogen (an agent that produces defects in the

developing fetus). It is considered the most common known cause of mental retardation. FAS defects range from specific structural bodily changes to growth retardation and subtle cognitive-behavioral abnormalities. The diagnostic criteria for FETAL ALCOHOL SYNDROME are the following: prenatal (before birth) and postnatal (after birth) growth retardation; characteristic craniofacial defects; central nervous system dysfunction; organ system malformations. When only some of these criteria are met, the diagnosis is termed *fetal alcohol effects* (Eighth Special Report to U.S. Congress, 1993). The abnormalities in physical appearance seem to decrease with age whereas the cognitive deficiencies tend to persist. There is no clear dose-response relationship between alcohol use and abnormalities. The safe amount of drinking during pregnancy (if it exists at all) is unknown. The peak level of blood (or brain) alcohol attained and the timing in relation to gestation (and particular organ development) are probably more important than the total amount of alcohol consumed during pregnancy. Genetic and maternal variables also seem to be important. Native-American and African-American children seem to be at high risk. While the public is generally aware of the relationship between alcohol consumption and fetal abnormality, surveys reveal that there is a need for greater public education in this area.

Smoking is associated with low birthweight. Cocaine use in PREGNANCY has been associated with complications (e.g., placental separation and in utero bleeding), and it appears to be associated with congenital abnormalities. Heroin use in pregnancy is associated with premature delivery and low birthweight; often there is a withdrawal syndrome in the baby at birth. Methadone (a long-acting opioid) usually reduces rates of prematurity and low birthweight but still causes as much or more opioid withdrawal in the newborn.

Cancer. There is clear epidemiologic evidence for an increased risk of certain types of CANCER in association with alcohol consumption. These include cancer of the esophagus, oropharynx, and liver. Other cancers possibly associated with alcohol consumption include cancer of the breast, stomach, prostate, and colon (Geokas, 1986). Alcohol plays a synergistic (additive) role with smoking TOBACCO in the development of cancer, particularly with respect to the head, neck and esophagus. There are several possible mechanisms through which alcohol enhances the onset of cancer. Alcohol appears to modify the immune response to cancers, facilitate delivery of carcinogens (substances which enhance cancer onset), and impair protective responses. Overall, alcohol is considered to act as a co-carcinogen; for example, it increases the likelihood of certain smoking-induced cancers.

Smoking is, of course, well established as a cause of lung as well as other cancers. Smoking is responsible for 85 percent of lung cancers and has been associated with cancers of the mouth, pharynx, larynx, esophagus, stomach, pancreas, uterine cervix, kidney, ureter, and bladder (Bartecchi et al., 1994). Chewing tobacco (SMOKELESS TOBACCO) is associated with mouth cancer. The chewing of BETEL NUTS with lime is common in Asia and results in absorption of arecoline (a mild stimulant). This practice also causes cancer of the mouth. It has been suggested that MARIJUANA smoking also causes lung cancer, since high tar levels are present in the smoked products.

ALCOHOL USE AND ABUSE AMONG ADOLESCENTS

Alcohol use among adolescents is a serious world-wide problem. Surveys indicate that up to 54 percent of eighth graders, and up to 84 percent of twelfth graders report having consumed alcohol (O'Malley et al, 1998). There is little doubt that parents' attitudes and habits concerning drinking are important influences on adolescent drinking (Ary et al, 1993). However, there is also evidence that adolescents who abuse alcohol often have co-existing psychopathology such as sociopathy, and bouts of depression and anxiety (Clark and Bukstein, 1998).

Another significant reason for concern about alcohol ingestion by adolescents is the close association of alcohol abuse with the use of other drugs. There is considerable evidence that alcohol use tends to precede use of illicit drugs, and some researchers argue that, based on long-term studies, alcohol serves as a "gate-way" to the use of illicit substances. As early as the eighth grade, alcohol users were found to have a significantly higher prevalence of cigarette smoking, and use of marijuana and cocaine than non-users of alcohol. This difference persists through grade 12 and thereafter (Kandel and Yamaguchi, 1993).

THE EFFECTS OF ALCOHOL ON BODILY SYSTEMS

Neurologic. Acute alcohol consumption causes impairment as described above. Alcohol potentiates the action of many drugs that produce acute effects on the brain. High blood-alcohol levels can result in "blackouts." This condition is due to acute loss of memory associated with intoxication, although the person usually behaves in apparently normal fashion during this period. Blackouts are also seen with the taking of other central nervous system depressants, such as the barbiturates and the benzodiazepines.

The main adverse consequences of chronic alcohol consumption with respect to the nervous system are the following: brain damage (manifested by dementia and alcohol amnestic syndrome); complications of the withdrawal syndrome (seizures, HALLUCINATIONS); and peripheral neuropathy. Chronic alcohol consumption results in tolerance, followed by an increased long-term consumption that likely leads to tissue damage. PHYSICAL DEPENDENCE may also develop (i.e., a withdrawal syndrome occurs on sudden cessation of drinking). The brain damage, when severe, is usually classified as one of two main disorders. The first is a type of global (general) dementia. It is estimated that 20 percent of admissions to state mental hospitals suffer from alcohol-induced dementia (Freund & Ballinger, 1988). The second is an alcohol-induced amnestic (memory-loss) syndrome, more commonly known as Wernicke-Korsakoff syndrome. This is related to thiamine (Vitamin B_1) deficiency. The Wernicke component refers to the acute neurologic signs, which consist of ocular (eye) problems such as a sixth cranial nerve palsy (disturbed lateral gaze), and ataxia (impaired balance); the Korsakoff component refers to the memory impairment, which tends to be selective for short-term memory and is usually not as amenable to treatment once it has become manifest.

Milder forms of these disorders are also detectable with neuropsychologic testing or brain IMAGING TECHNIQUES (CAT scans; MRI). Studies of detoxified alcoholics (without other evidence of organic brain damage) reveal that 50 to 70 percent have impairments in neuropsychologic assessment (Eckardt & Martin, 1986). In most of these cases there is reversibility with abstinence from alcohol. Severe liver disease (e.g., advanced cirrhosis or acute hepatitis) may also contribute to this neurologic impairment. Computerized tomography (CT) scans reveal that many alcoholics have cerebral atrophy—this consists of decreased brain weight, an increase in spaces (sulci) between various regions of the brain, and an increase in size of ventricles (spaces filled with cerebrospinal fluid). In a minority of cases, these structural changes are reversible with abstinence. Seizures are associated with heavy alcohol consumption and usually occur in association with alcohol withdrawal. Abstinence from alcohol is usually the only treatment needed for this type of seizure. The hallucinations that are mostly associated with alcohol withdrawal are usually treated with drugs—benzodiazepines and phenothiazines.

Peripheral neuropathy is seen in association with chronic alcoholism. Peripheral neuropathy usually refers to toxic damage to peripheral nerves. Concurrent nutritional deficiencies often contribute to this damage. This neuropathy results in changes in sensation and occasionally motor function, usually in the legs. Sometimes this can occur acutely with intoxication. For example, the abnormal posture in association with a drunken stupor can result in radial nerve ("Saturday night") palsy. Alcoholics are also at increased risk of subdural hematomas (blood clots due to ruptured intracranial veins secondary to trauma) and of stroke.

The neurologic complications associated with the acute use of other drugs of abuse include seizures (convulsions) and strokes in association with cocaine. High doses of some opioids, such as propoxyphene (Darvon) or MEPERIDINE (Demerol) can also cause seizures. Substances which can cause delirium (reversible disorientation) include *Cannabis* (MARIJUANA), phencyclidine (PCP), lysergic acid diethylamide (LSD), and atropine. Sudden cessation of use of central nervous system depressants (benzodiazepines, barbiturates, and alcohol) can result in seizures and hallucinations. Chronic use of other substances of abuse can also result in neurologic complications. Tobacco use is associated with increased rates of stroke (but it appears to be associated with lower rates of Parkinson's disease—a progressive disorder affecting control of movement). Solvent abuse (inhaling) can cause damage to the cerebellum (the part of the brain controlling movement) and to peripheral nerves. A form of "synthetic heroin," MPTP (1-methyl-4-phenyl-1,2,5,6-tetrahydropyridine),

an analogue of meperidine (Demerol), has been demonstrated to cause a severe form of Parkinson's disease.

Psychiatric. Alcohol-related diagnoses are common among psychiatric patients. For example, a recent study (Moore et al., 1989) showed that 30 percent of those admitted to a psychiatric unit had a concurrent alcohol-related diagnosis. Alcohol alone may produce symptoms and signs that mimic psychiatric disorders. Examples include depression, anxiety disorder, psychosis, and antisocial personality disorder. Alternatively, an alcohol-related disorder may co-exist with one of these or may aggravate the psychiatric disorder.

Alcohol as a central nervous system (CNS) depressant tends to *cause* low mood states (hypophoria) with chronic use. It does not commonly cause long-lasting significant clinical depression, but it may aggravate it. If alcohol is the primary cause of a low mood state, then abstinenee from alcohol, as the sole treatment, rapidly improves the disorder. Hallucinations may occur during alcohol withdrawal, mimicking a psychotic disorder. Similarly, the anxiety associated with alcohol withdrawal may mimic an anxiety disorder. Anxiety and hallucinations may also be seen during withdrawal from sedative-hypnotics. Behavior associated with alcoholism may lead to an erroneous diagnosis of antisocial personality disorder.

When alcohol is used for self-medication in some psychiatric conditions, such as anxiety disorders, it tends only to be of short-term help and leads to more long-term problems. Other drugs of abuse, such as the stimulants cocaine and amphetamine, also produce anxiety and occasionally may produce a psychotic state associated with acute intoxication. This usually disappears rapidly as the drug effects wear off. Withdrawal following chronic use of stimulants may be associated with depression, excessive fatigue, and somnolence (a "crash"). Tobacco smoking also appears to be somewhat associated with depression. (Individuals with a history of depression are more likely to smoke, and may develop depression when they try to stop.) The nature of this relationship is unclear, but patients with psychiatric diagnoses have higher rates of smoking than the general population. Hallucinogens (such as LSD and PCP) may also cause an acute psychotic disorder which typically disappears as drug effects wear off; however, in some cases there may be longer lasting effects. Antisocial personality disorder is a common diagnosis in those who abuse drugs.

Endocrine and Reproductive. Alcohol produces both acute and chronic effects on virtually all endocrine organs (glands). Acutely, alcohol raises plasma catecholamines, which are chemicals released from nerve endings that are responsible for certain emotional reactions—"fear, flight, and fight". Epinephrine (adrenaline) is released from the inside of the adrenal gland (medulla) and norepinephrine (noradrenaline) from sympathetic NEURONS (nerve cells) and the adrenal glands. Alcohol also causes release of cortisol from the outside (cortex) of the adrenal gland both acutely and chronically. Cortisol is a hormone (chemical messenger) responsible for multiple effects on the body, including changes in the immune response, glucose regulation, fat breakdown, blood pressure, and mood. Alcohol-induced cortisol excess can mimic Cushing's disease (a condition associated with excess cortisol production, often caused by a tumor on the adrenals) and is known as pseudo-Cushing's disease. Alcohol affects the hypothalamus (an area of the brain), where it modifies chemical-releasing factors, which in turn control release of hormones from the pituitary (a gland in the brain linked to the hypothalamus by a special blood supply), which in turn affect endocrine organs throughout the body. Acutely, alcohol also inhibits the release antidiuretic hormone (ADH) from the posterior pituitary, which results in an increase in urine production.

The best documented chronic endocrine effect of alcohol is male hypogonadism. This is a condition resulting from low sex-hormone function. Signs of this are small testes and decreased body hair. Symptoms include loss of libido and impotence. Hypogonadism can occur as a result of alcohol lowering testosterone levels. Alcohol acts both directly on the testes and indirectly via the hypothalamus. Alcoholic liver disease may also produce feminization in men, as a result of impaired metabolism (breakdown) of female sex hormones such as estrogen. Signs of such feminization in men include gynecomastia (enlarged breasts) and female fat distribution. In women who drink alcohol excessively, there is a high prevalence of gynecologic disorders (missed periods and problems in functioning of ovaries) and a possibly earlier onset of menopause than in nondrinkers. In women, also,

alcohol is metabolized at different rates according to the particular phase of the menstrual cycle.

Abnormalities of both growth hormone (impaired release) and prolactin (increased release) have been described in association with acute alcohol ingestion. Thyroid function (which controls rate of body metabolism) can be indirectly affected as a result of alcoholic liver disease. This results in impaired conversion of T4 (one version of thyroid hormone) to T3 (a more active form of thyroid hormone). Furthermore, in alcoholism there are abnormalities in the proteins to which thyroid hormone binds. This results in making thyroid function tests difficult to interpret. Overall thyroid function is usually normal despite mild abnormalities in the tests.

Other drugs, particularly the opioids, also have multiple effects on the endocrine system. Opioids produce a degree of hypogonadism as a result of lowered testosterone in males and disturbed menstrual function in females. This results from opioid inhibition of gonadotropin releasing hormone (GRH) in the hypothalamus, which in turn inhibits release of LH (lutenizing hormone) and FSH (follicle stimulating hormone) from the pituitary. Opioids also inhibit corticotropin releasing factor (CRF), which results in decreased adrenocorticotrophic hormone (ACTH) and decreased cortisol. Nicotine causes release of epinephrine and norepinephrine, which in turn increase blood pressure and heart rate. Nicotine also enhances the release of ADH from the hypothalamus, which decreases urine output (i.e., counteracts alcohol's effects).

Cardiovascular. Alcohol has direct effects on both cardiac muscle and cardiac electrophysiology (electrical functioning). These effects are also dependent on prior history of alcohol use (i.e., whether there have been underlying changes due to chronic use) and whether there is any evidence of underlying heart disease. Acutely, alcohol is a myocardial depressant (decreases muscle function) and, chronically, it may cause a degeneration of cardiac muscle (known as cardiomyopathy), which can lead to heart failure (condition due to excess body fluids because of inadequate pumping function of the heart). Abstinence from alcohol leads to improvement in function in some cases.

Both acute alcohol intoxication and acute withdrawal can lead to cardiac arrhythmias (abnormal heart beats). The most frequent association is with atrial fibrillation (frequent uneven and uncoordinated contraction of the atria). This is usually not life-threatening and mostly disappears without specific treatment. High levels of alcohol consumption are associated with increased rates of coronary (blood vessels which supply heart muscle) heart disease, while low levels of consumption (in comparison to complete abstinence) may be associated with a mild protective effect (the so-called U-curve relationship). However, low levels of consumption are not recommended as a preventive measure against coronary heart disease. Cigarette smoking is a much greater risk factor for coronary heart disease than is alcoholism. It should be noted however, that 80 to 90 percent of alcoholics are also cigarette smokers.

Multiple epidemiologic studies have established a relationship between alcohol and high blood pressure (hypertension). Somewhere between 5 and 24 percent of hypertension is considered to be alcohol related (Klatsky, 1987). The relationship seems to hold most strongly for white males over the age of 55 consuming at least 3 standard drinks per day on a chronic basis. Many cases resolve with abstinence. Acute alcohol withdrawal has also been associated with hypertension, but this usually lasts for only a few days.

The acute use of cocaine (a stimulant) results in increases in heart rate and blood pressure and causes narrowing of peripheral and coronary artery blood vessels. Repeated use of cocaine has been associated with abnormal heart beats, myocardial infarction (heart attack), and possibly myocardial fibrosis (an increase of scar tissue within the heart).

Acute tobacco use also results in constriction (narrowing) of blood vessels and an increase in heart rate because of the nicotine. Chronic tobacco use is the most important of the preventable causes of coronary heart disease. The coronary arteries supply the heart muscle. Long-term tobacco use results in an increase in atherosclerosis (build up of fat and other products inside the walls of blood vessels) in most of the arteries throughout the body and increases coagulation (clotting). This has important effects on the following blood vessels: the coronary (causes angina [chest pain] and infarction [heart attack]; the aorta (causes aneurysms, the ballooning effect on the arterial wall, which can be fatal); the carotid (cause of strokes); the femoral (causes intermittent claudication, pain on walking); and the kidney (cause of kidney failure and some hypertension). Acute use of opioids have mi-

nor effects on blood pressure. There are not thought to be important chronic adverse effects of opioids on the cardiovascular system. Marijuana acutely causes increases in heart rate and blood flow.

Respiratory System. Acutely, alcohol does not usually interfere with lung function; however a decrease in cough reflexes, a predisposition to reflux of stomach fluids, and the impairment of bacterial clearance in the respiratory tract occur after intoxication. For some persons with asthma, alcoholic beverages can induce bronchospasm (airway narrowing). This is thought to be related to nonalcoholic components in the beverage. Acute alcohol consumption also has a direct depressant effect on the respiratory center located in the brainstem. Accordingly, an overdose (intentional or unintentional) can result in death from respiratory failure (decreased ability to breathe). Alcohol also contributes to respiratory depression when taken with other central nervous system depressants such as barbiturates and benzodiazepines (minor tranquillizers). Acute alcohol intake increases sleep apnea (period of time not breathing) in those who suffer from this disorder.

Chronic alcohol consumption is associated with several pulmonary infectious diseases (in addition to risks associated with tobacco smoking). These include pneumonia, lung abscess, and tuberculosis. Aspiration pneumonia occurs in association with high levels of alcohol intoxication; it is thought to be caused by the inhalation of bacteria caused by the impairment of the usual reflexes, such as coughing. Pancreatitis and alcoholic cirrhosis are associated with pulmonary effusions (build-up of fluid on the lung).

Among the other drugs, cigarette smoking causes emphysema, chronic bronchitis, and lung cancer. The smoking of marijuana on a frequent long-term basis may also increase the likelihood of these disorders. Acute use of opiate drugs intravenously may cause pulmonary edema (accumulation of fluid in the lungs). which can be life-threatening. Chronic use of intravenous drugs may cause pulmonary fibrosis (increased scar tissue). This is probably related to impurities, such as talc, associated with the cutting of the drug (diluting the dose with fillers) prior to its sale and eventual injection.

Gastrointestinal Tract and Pancreas. Acutely, alcohol alters motor function of the esophagus. Chronic use of alcohol increases gastroesophageal reflux. Alcohol alone does not appear to cause peptic ulcers (cigarettes do), but alcohol interferes with healing. Alcohol disrupts the mucosal barrier in the stomach and causes gastritis (inflammation of the stomach) which can lead to hemorrhage, especially when combined with aspirin. Alcohol also interferes with the cellular junctions within the small intestine, which can result in the disturbance of fluid and nutrient absorption, producing, diarrhea and malabsorption. Any resulting nutritional deficiencies can further aggravate this process.

Heavy alcohol use interferes significantly with pancreatic structure and function. Alcohol abuse and gallstone disease are the major causes of pancreatitis, and alcoholism alone is responsible for most cases of chronic pancreatitis. Alcohol changes cellular membranes, resulting in changes in transport mechanisms and the permeability of vital ions and nutrients essential for normal cellular function. Acetaldehyde, which is a breakdown product of alcohol (and also present in cigarette smoke), is toxic to cells and has been proposed as a causative agent in the development of this disorder (Geokas, 1984). Acute pancreatitis is life-threatening; patients have abdominal pain, nausea, and vomiting. Increased levels of pancreatic enzymes, such as amylase and lipase, accompany this disorder. Treatment is usually by conservative measures, such as replacement of fluids and pain relief. Chronic pancreatitis can be without symptoms; can become evident with chronic abdominal pain and evidence of malabsorption (weight loss, fatty stools, nutritional deficiencies); or, uncommonly, with diabetes mellitus as a result of the destruction of the endocrine as well as the exocrine function of the pancreas.

Liver. Alcoholic liver disease is a major cause of morbidity and mortality in the United States; in 1986, cirrhosis of the liver was the ninth leading cause of death. Alcohol causes three progressive pathological (abnormal) changes in the liver—fatty liver, alcoholic hepatitis, and cirrhosis. These changes are useful in a prognostic sense but can only be diagnosed with a liver biopsy, which is not always feasible or practical. More than one pathological condition may exist at any one time in a given patient. Fatty liver is the most benign of the three conditions, and is usually completely reversible with abstinence from alcohol; it occurs at a lower threshold of drinking compared to alcoholic

hepatitis and cirrhosis. Alcoholic hepatitis ranges in severity from no symptoms at all to severe liver failure with a fatal outcome; it can be followed by complete recovery, chronic hepatitis, or cirrhosis. Treatment is primarily supportive. Similarly, the symptoms and signs of cirrhosis range from none at all to coma and death. Cirrhosis consists of irreversible changes in liver structure resulting from an increase in scar tissue. A consequence of this is an abnormal flow of blood through the liver (shunts), which can result in the adverse health consequences of bleeding and presentation of toxic substances to the brain. This, in turn, may result in effects ranging from impaired thinking to coma and death. Abstinence from alcohol can prevent progression of cirrhosis and reduces mortality and morbidity (illness) from this condition. Medications may also help to reduce mortality from alcoholic liver disease. These include propothiouracil (an antithyroid drug) and prednisone (a steroid). The former reduces the oxygen requirements for areas of the liver that are poorly perfused. The latter reduces inflammation. Women appear to be at higher risk for liver damage than are men.

Opioid use alone has not been associated with liver disease, but some opioids such as morphine can cause spasm of the bile duct, which results in acute abdominal pain. Tobacco use is associated with a more rapid metabolism (breakdown) of certain drugs in the liver. This means that sometimes higher or more frequent dosing of medicatons is required for smokers. This effect is thought to relate to the tars in tobacco rather than to the nicotine. High doses of cocaine have been associated with acute liver failure.

Acute and chronic hepatitis (types B, C, and D) is common in users of intravenous drugs. It is not usually the drug itself that causes hepatitis (inflammation of the liver) but rather the introduction of disease-producing organisms associated with the sharing of needles. Viruses and bacteria introduced by injecting drugs cause other problems, such as HIV infection and AIDS, endocarditis (infection of heart valves), cellulitis (skin infection), and abscesses.

Immune System. Alcohol affects the immune system both directly and indirectly. It is often difficult to discern the direct effects of alcohol from concurrent conditions, such as malnutrition and liver disease. Alcohol affects host defense factors in a general way; it also seems to predispose those who drink heavily to specific types of infection. With respect to host factors, alcohol alone can reduce both the number and function of white blood cells (both polymorphonuclear leucocytes and lymphocytes). This both predisposes toward infection while it interferes with the ability to counteract infection. Mechanical factors are also of importance. For example intoxication with alcohol and a depressed level of consciousness (and depressed cough reflex) predisposes toward aspiration pneumonia. Specific infections that alcoholics are at higher risk for, compared to the population at large, include pneumococcal pneumonia (the most common form of pneumonia), other lung infections (Hemophilus influenzae, Klebsiella), abscesses (anaerobic infections,), and pulmonary tuberculosis.

Alcoholics with liver disease are at increased risk of spontaneous bacterial peritonitis (inflammation of the lining of the abdominal cavity). Other infections possibly associated with alcoholism include bacterial endocarditis (infection of the heart valves), bacterial meningitis, pancreatitic abscess, and diphtheria. HIV infected drug abusers are at increased risk of tuberculosis as well as a multitude of other infections. As mentioned above, injecting drug users are also susceptible to a variety of infections associated with use of unsterile equipment.

Changes in immune function have been reported to occur in users of other drugs of abuse, including heroin, cocaine, and marijuana. The precise relationship of the immune function change to the drug of abuse is not yet understood. Lifestyle factors such as poor nutrition are also likely to contribute to this.

Nutrition. In heavy alcohol consumers, malnutrition as a result of poor dietary habits is common. In women, heavy alcohol consumption is associated with lower than usual body weight to a degree similar to that also associated with tobacco smoking. There is less of a weight-lowering effect in men. Specific nutritional disorders associated with alcoholism include anemia (due to iron or folate deficiency); thiamine (Vitamin B_1) deficiency—causing beri-beri or Wernicke's encephalopathy or neuropathy; malabsorption; and defective immune and hormonal responses. Alcohol also interferes with the absorption of vitamins (such as pyridoxine and Vitamin A), minerals (such as zinc), and other nutrients (such as glucose and amino acids) (Mezey, 1985).

Metabolic. Alcohol is metabolized (broken down) in the liver to acetaldehyde and hydrogen, and then to carbon dioxide and water. Acetaldehyde is toxic to many different cellular functions. Alcohol affects carbohydrate, lipid (fat), and protein metabolism. Alcohol can cause low blood glucose (hypoglycemia) due to inhibition of glycogen (liver stores of carbohydrate) metabolism. Alcohol also raises blood sugar and acids (alcoholic ketacidosis). By interfering with the elimination of uric acid, alcohol may precipitate acute attacks of gout. Increased urinary excretion of magnesium can result in muscle weakness. Alcohol causes disturbances in blood lipids, with mostly an increase in triglycerides and high density lipoprotein (HDL) cholesterol.

Acute alcohol consumption can decrease, whereas chronic consumption can increase, the metabolism of certain drugs. Tobacco smoking also increases the metabolism of some drugs, such as theophylline and caffeine. This results from the increased activity of various liver enzymes as discussed above.

Hematologic (Blood) System. The effects of alcohol on the hematologic system can either be direct, or it can be indirect (as a result of liver disease or nutritional deficiencies). Uncommonly acute alcohol consumption (a very large dose in a short span of time) has direct effects on the bone marrow, resulting in decreased production of red cells, white cells, and platelets.

The most frequent effect seen in alcoholics following chronic consumption is an increase in the size of the red blood cells (macrocytosis). This is mainly due to direct toxic effects on the red cell membrane rather than to folate (a vitamin found in green vegetables) deficiency, which also causes macrocytosis. A folate deficiency, however, is sometimes seen in alcoholics caused mainly by impaired intake and absorption of folate. Iron deficiency anemia is also seen because of impaired intake of iron and because of frequent bleeding (due to a variety of factors, such as coagulation defects, gastritis, and the impaired healing of peptic ulcer). Iron-overload syndromes are also diagnosed in alcoholics and are due to a multiplicity of causes. Chronic alcohol consumption can also lead to hemolytic (excess breakdown of red blood cells) anemia, which is mainly seen in association with liver disease. Platelet production and function can be suppressed by alcohol, resulting in prolonged bleeding times.

Other drugs also exert hematologic effects. Experimental addiction to opioids results in a reversible anemia and a reversible increase in erythrocyte sedimentation rate (a nonspecific indicator of disease process). Smoking allows carbon monoxide to enter the body and bind to hemoglobin (carboxyhemoglobinemia), which consequently causes an increase in red cell production (erythrocytosis). The hematocrit value and the plasma fibronogen (a clotting factor) rise and increase blood viscosity; platelets (sticky constituents of blood important in wound healing) aggregate more in smokers. These thickening factors, together with damage to the insides of blood vessels, increase the probability of both stroke and heart attack (myocardial infarction) in smokers. White cells are also at increased levels in smokers (leucocytosis).

Skeletal Muscle. Chronic alcohol consumption can result in muscle cell necrosis (death). Two main patterns are seen: (1) An acute alcoholic myopathy (disturbance of muscle function) occurs in the setting of binge drinking, sometimes associated with stupor and immobilization. This results in severe muscle pain, swelling elevated creatine kinase (a muscle enzyme), and myoglobinuria (muscle protein in the urine which can cause kidney failure). (2) This pattern consists of a more slowly evolving syndrome of proximal muscle (those closest to the trunk) weakness and atrophy (decreased size). Milder degrees of muscle injury are quite common and consist of elevated levels of the muscle enzyme creatine kinase.

Cocaine use can also cause muscle damage (rhabdomyolysis), resulting in abnormalities of creatine kinase. Most drugs of abuse (especially depressants) may indirectly cause muscle damage as a result of prolonged abnormal posture, for example, sleeping in an intoxicated state on a hard surface.

Kidney. Alcohol abuse causes a variety of electrolyte and acid-base (blood chemistry) disorders, which include decreases in the levels of phosphate, magnesium, calcium, and potassium. These abnormalities relate to disorders within the functioning kidney tubules (involved in secretion and reabsorption of minerals). The abnormalities usually disappear with abstinence from alcohol.

Chronic use of other abused substances is also associated with kidney (renal) damage and failure.

Long-term consumption of pain-relieving medicines (daily use over many years) has been associated with kidney failure (analgesic nephropathy). This is especially associated with the combination products—those that include two or more of CODEINE, CAFFEINE, aspirin, and phenacetin. The rewarding effects that perpetuate this form of drug use most probably relate to the codeine (an opioid) and the caffeine (a stimulant). Heroin use has been associated with a form of kidney failure known as heroin nephropathy. Its precise relationship to heroin use is unclear. Secondary effects on the kidneys from drug and alcohol abuse also occur (for example, from effects of trauma or muscle damage as described above).

(SEE ALSO: *Crime and Alcohol; Crime and Drugs; Family Violence and Substance Abuse; Inhalants; Social Costs of Alcohol and Drug Use; Substance Abuse and AIDS; Tobacco: Complications*)

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Mental Disorders Psychiatric disorders have long been recognized as being associated with psychoactive-substance-use disorders (commonly referred to as drug or alcohol abuse). The term *dual diagnosis* is frequently used to describe people with substance-use disorders combined with other psychiatric disorders. The term COMORBIDITY is also used to describe the situation in which an individual has two or more distinct disorders. Anxiety disorders and mood disorders are generally the disorders thought to occur in individuals with substance abuse, but other psychiatric disorders also demonstrate high rates of psychoactive-substance use also, including eating disorders (particularly BULIMIA), posttraumatic stress disorder (PTSD), personality disorders, somatization disorder, and SCHIZOPHRENIA. Children with ATTENTION DEFICIT

hyperactivity disorder (ADHD) may also be at increased risk of substance abuse as adults. Various relationships may exist between drug and alcohol use and the development of these psychiatric disturbances. In understanding the relationships between substance abuse and psychiatric disorders, the concepts of *primary* and *secondary* are of critical importance. The primary-secondary dichotomy is based on the time sequence in which each disorder developed. When a disorder is referred to as primary, this would indicate that it presented first. The rationale for using the primary-secondary concept involves improved prediction of familial clustering of the psychiatric disorder, implications for treatment, and improved outcome prediction.

In addition to the primary-secondary distinction, the approach to the individual with both a substance use and a nonsubstance use psychiatric disorder should incorporate a similar but slightly more encompassing approach. The drug or alcohol use in such individuals may be involved as a form of self-medication for the psychiatric disturbance; it may itself induce psychiatric symptoms in an otherwise unaffected individual; or the individual may be affected by both disorders (substance abuse and other psychiatric disorders) through separate routes of VULNERABILITY.

As can be anticipated from this introduction to dual diagnosis issues, the relationship of psychiatric disorders and substance abuse is complex. Despite this complexity, the extent of such problems underscores the need for attention to this area. Studies have shown higher prevalence rates of substance abuse in individuals with psychiatric disorders than in the general population, and conversely patients seeking care for substance abuse have shown high rates of other psychiatric disorders. Large epidemiologic studies of community samples in the United States reveal that greater than 50 percent of substance abusers have at least one other mental illness (Regier, 1990). Data from this same study indicate that approximately one third of those identified as having a mental disorder also have a substance-abuse disorder.

ALCOHOLISM AND MOOD DISORDERS

Depression and Alcoholism. The rate of depression in individuals with alcoholism and rate of alcoholism in individuals affected with mood disorders (depression and mania) varies greatly according to different studies. The reason for the lack of agreement regarding such rates involves problems shared by all combinations of dual diagnosis. Two such problems include the means of assessment (both of substance abuse and psychiatric symptoms), and the timing of the assessment of psychiatric symptoms (i.e., in relationship to the last occurrence of substance use).

The effect that the means of assessment has upon psychiatric comorbidity is well illustrated by depression. Different rates of depression in alcoholics are seen if one uses standard clinical interviews, structured research interviews or self-report measures. Such methodological differences have led to widely differing conclusions regarding both comorbidity rates and comorbid influence. However, recent estimates from a number of sources suggests that 40 percent of all alcoholics in the U.S. are also battling depression (Larson, 1998).

The critical importance of the timing of the psychiatric assessment and its relationship to comorbidity is demonstrated by studies from the Alcohol Research Center in San Diego (Brown, 1988; Schuckit, 1990). Symptoms of depression in 191 alcoholics were recorded within 48 hours of admission for alcohol detoxification and again after 4 weeks of abstinence (Brown, 1988). Significant levels of depression were noted in 42 percent of alcoholics in the first assessment, but in only 6 percent in the follow-up evaluation irrespective of any specific antidepressant therapy.

These studies demonstrate that for a number of alcoholics, psychiatric symptoms are directly induced by the intake of alcohol, and that these symptoms should be regarded as secondary to the alcoholism. This is important for two reasons. First, if the psychiatric symptoms are secondary to the alcoholism, treatment of the psychiatric symptoms alone will not treat the main disorder (alcoholism). Second, risk for relapse of the alcoholism is high.

Another complication of alcoholism in regard to depression is poor treatment response to standard ANTIDEPRESSANT therapy, both pharmacologic and nonpharmacologic in type. The reason for this is unknown, but may be related to adverse social complications of the alcoholic behavior (e.g., legal problems, job difficulties, marital separation, and divorce) (Cook, 1991).

Mania and Alcoholism. Individuals with bipolar affective disorder (manic-depressive disorder) have been noted to have increased use of alco-

hol during their manic episodes. Two studies have suggested that alcoholic bipolar patients have high rates of alcoholism in their families as compared to nonalcoholic bipolar patients (Morrison, 1974; Dunner, 1979). This fact suggests that the risk for alcoholism in bipolar disorder may occur due to a familial predisposition (e.g., genetic predisposition, behavior modeling, etc.), and not necessarily from a complication of the manic episode itself. Regardless, impulsive behavior during manic episodes clearly includes risk for excessive alcohol use.

SUBSTANCE ABUSE AND MOOD DISORDERS

Nearly all substances of abuse have the potential to alter mood symptoms. Classically, PSYCHOSTIMULANTS, such as AMPHETAMINES and COCAINE, may induce an appearance of elevated mood, racing thoughts, increased energy, and sense of well-being. Individuals who have developed tolerance to stimulants will experience, upon their discontinuation, withdrawal. These withdrawal symptoms will overlap characteristic depressive symptoms, including severe dysphoria, insomnia followed by hypersomnia, irritability, and fatigue. OPIATES induce a sense of elevated mood, and increased self-esteem. A sense of decreased anxiety is also frequently reported. Upon withdrawal, depressive symptoms are accompanied by characteristic physical symptoms such as muscle aches, drug CRAVING, lacrimation (secretion of tears), and piloerection (goose flesh).

SUBSTANCE ABUSE AND PERSONALITY DISORDERS

Personality disorders by definition involve maladaptive patterns of relating to one's environment and self that lead to conflict. To meet the definition of a disorder, these patterns should be enduring qualities, and the onset of such disorders is late adolescence. Behavior induced by substance abuse should be carefully separated from behavior demonstrated during periods of abstinence. This is important, since maladaptive behavior associated with personality disorders will persist through adulthood, while maladaptive behavior that is induced by substance abuse should subside during abstinence from the substance. Two personality disorders that are closely associated with substance abuse are ANTISOCIAL PERSONALITY DISORDER and borderline personality disorder.

Antisocial Personality and Alcoholism. A great deal is known regarding the relationship of antisocial personality disorder and alcoholism. This diagnostic combination is estimated to involve as many as 2 percent of the male population of the United States. Most studies of this combination of illnesses indicate that the antisocial alcoholic has an earlier onset of drinking difficulties, more family history of alcoholism, more social complications of alcoholism, and a greater number of symptoms of other psychiatric disturbances, e.g., drug abuse, depression, mania, schizophrenia, and psychotic symptoms. Antisocial alcoholics have also been reported to attempt suicide more frequently. In addition to these more severe symptoms at the time of initial evaluation, antisocial personality disorder influences the natural history of the substance use disorders and alcoholism. This change in course is demonstrated by the following studies.

Schuckit (1985) utilized standardized research criteria to divide a group of 541 alcoholics into those who were primary alcoholics, primary drug abusers, primary antisocials, and primary affective disorders. Intake and one-year outcome were then evaluated. The primary antisocial, along with the primary drug abusers, had a poorer one-year outcome in terms of drug use, police and social problems, and higher scores (worse outcome) on a clinical-outcome scale. Schuckit concluded from this study that antisocial personality predicted a poor prognosis in terms of continued alcohol problems.

In a carefully designed study, Rounsaville and coworkers (1987) evaluated 266 alcoholics one year after treatment. Multiple-outcome measures were utilized in this study and over 84 percent of the original cohort were reevaluated. In this study, it was found that in males, an additional diagnosis of major depression, antisocial personality disorder, or drug abuse were associated with poor prognosis at one year. Further analysis in this study also supported the conclusion that the diagnosis was the factor that conveyed the poor prognosis, not general severity of psychopathology or degree of alcohol dependence.

Another study that looked at outcome of alcohol problems in subtypes of antisocials was conducted by Liskow (1991). In this study, antisocial alcoholics were subtyped as to presence of additional diagnoses of drug abuse and depression. An alcoholism-

only group was included as a control group. In this study, the alcoholism-only group had the best outcome on a number of measures, while the antisocial alcoholic with drug abuse had the worst outcome. The antisocial alcoholic with no other diagnosis and the antisocial alcoholic with depression were similar in outcome—and intermediate in outcome. Overall, the differences in the alcoholism-only compared to the antisocial-only and the antisocial depressed alcoholics were small compared to the differences between the antisocial plus drug group and all other groups. This study suggests that the poor prognosis in antisocial alcoholics may depend in part on other additional psychopathology (i.e., drug abuse).

Antisocial Personality Disorder and Substance Abuse. Individuals with antisocial personality disorder have high rates of drug use. Conversely, the dysfunctional lifestyle of an individual actively involved with substance abuse frequently involves lying, joblessness, and the inability to comply with social norms concerning issues such as child care, finances, and the legal system. This makes disentangling these disorders difficult. If one looks for evidence of conduct disturbance, particularly during the late adolescence that predates the substance abuse, then the diagnosis of antisocial personality disorder is much more reliable. The implication for this distinction involves improved ability to predict changes in the individual should long-term abstinence be achieved. The abstinent antisocial will likely continue to demonstrate behavior problems in a variety of areas, whereas someone with an intact personality would be expected to have a better prognosis.

Borderline Personality Disorder. Less is known about borderline personality disorder and substance abuse or alcoholism. Individuals with borderline personality disorder clearly have high rates of substance abuse, and the criteria published in the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, revised (DSM-III-R) (American Psychiatric Association, 1987) for this personality disorder include self-damaging behavior—such as substance abuse—as one of the five symptoms required to make the diagnosis. Dulit (1990) has suggested that drug abuse may be an important factor in the development of this disorder.

POSTTRAUMATIC STRESS DISORDER (PTSD) AND SUBSTANCE ABUSE

Following extreme stresses beyond the realm of normal human experience, symptoms of anxiety including intrusive recollections of the trauma, autonomic hyperactivity, and nightmares have long been observed, but PTSD as a psychiatric diagnosis is much newer. Following recognition of this disorder, the link with substance abuse has been the subject of a number of studies. Rates of alcoholism in PTSD range from 40 to 80 percent, while other forms of substance abuse may range from 20 to 50 percent. This high rate of substance abuse has led to the hypothesis that the drug use may be explained by a self-medication theory. Jelinek (1984) has proposed that in the treatment of PTSD, those with substance abuse be divided into groups with abuse that preceded the trauma and those whose abuse followed the trauma. This latter group is considered as a "self-medication group," and in this group treatment of the PTSD is felt to be the primary goal. Following treatment for the PTSD, it is believed that the substance abuse in this group will then decrease or end. In the former group, detoxification from the substance abuse and abstinence is felt to be the primary goal, and that following this the PTSD symptoms will improve.

SUBSTANCE ABUSE AND OTHER ANXIETY DISORDERS

Alcohol and Anxiety. Individuals with anxiety disorders (e.g., generalized anxiety disorder, panic disorder, phobic disorders, obsessive-compulsive disorder) find that alcohol provides temporary relief from some of their anxiety symptoms. Large community studies of individuals with phobias suggest over a twofold increase in alcoholism risk. Panic disorder patients have rates of alcoholism approaching 20 percent, and male relatives of individuals with panic disorder have a two to three times increased rate of alcoholism when compared to controls, further suggesting a relationship between alcoholism and anxiety disorders. Another known fact is that anxiety symptoms are experienced during withdrawal. Schuckit (1990), in a study of anxiety symptoms during withdrawal, evaluated 171 alcoholics for anxiety and panic symptoms. Nearly all subjects had at least one

anxiety symptom during heavy drinking, or upon abrupt discontinuation of drinking, but only 4 percent fulfilled DSM-III-R criteria for generalized anxiety disorder when three or more months of abstinence were achieved.

Anxiety and Substance Abuse. Panic attacks have been shown to be induced by psychostimulants, particularly COCAINE. The rate of panic attacks among users of cocaine has been reported to be as high as 64 percent. Anxiety symptoms during the withdrawal phase from cocaine also increases the risk for alcohol abuse and/or benzodiazepine abuse. These substances are frequently used to ease the "crash" phase.

SUBSTANCE ABUSE AND SCHIZOPHRENIA

Only recently has the high prevalence of alcoholism in schizophrenia been noted. Likewise, the recognition of high rates of other substance abuse in the schizophrenic population was not appreciated until the 1980s. A review of published estimates of the prevalence of alcohol abuse in schizophrenia reported a range of 8.4 to 47 percent (Mueser, 1990). Stimulant abuse in this review was reported between 4 and 15 percent. The question of whether substance abuse induces a chronic schizophrenic-like psychosis even after the drugs are stopped is still open to debate. It is generally held, however, that individuals who develop schizophrenia coupled with drug abuse would most likely have developed schizophrenia regardless, but the abuse may have caused an earlier onset. The early drug use may represent efforts at self-treatment. Treatment of the schizophrenic with drug abuse presents a major clinical challenge. Such patients tend to be disruptive, prone to frequent relapse of psychosis and drug use, and do not easily fit into conventional treatment settings. Optimal care is thus difficult, and improved strategies for treatment are needed.

SUBSTANCE ABUSE AND EATING DISORDERS

Individuals with eating disorders (ANOREXIA and BULIMIA) abuse a number of drugs and alcohol. During the course of their lives, they often use agents to reduce weight, such as laxatives, emetics, diet pills, and diuretics. Of those individuals with eating disorders who seek psychiatric treatment, as many as 35 percent have a significant substance-abuse history. Alcoholism, particularly in bulimia and bulimic anorectic patients, appears to be common. Substance abuse in eating disorders is generally thought to convey a poor prognosis for recovery.

SUBSTANCE ABUSE AND ATTENTION DEFICIT DISORDER

Children with attention deficit hyperactivity disorder (ADHD) have been noted to be at risk for development of alcoholism and cocaine abuse as they grow into adolescence and adulthood. Family studies of children with ADHD and alcoholism have demonstrated higher rates of alcoholism in family members than that seen in the general population. Goodwin (1975) compared previously hyperactive adult adoptees with and without alcoholism. As children, these alcoholics were hyperactive, truant, shy, aggressive, disobedient, and friendless. In these adoptees, those with alcoholism clearly had an excess of alcoholism in their biological parents. No alcoholism was found among the biological parents of the nonalcoholic hyperactive adoptees. These findings suggest that in the case of alcoholism and hyperactivity, the risk for alcoholism comes from a genetic basis and not necessarily from just having ADHD.

It has been estimated that 15 to 20 percent of cocaine users might also be afflicted with ADHD. Studies have been conducted using Ritalin to treat cocaine users suffering ADHD—indeed, one such study by the New York State Psychiatric Institute showed a 66 percent drop of ADHD symptoms and a decline of 75 percent in the craving of cocaine (New York State Psychiatric Institute (1996). Despite those encouraging statistics, counseling and other treatment methods are obviously still very much in need.

SUBSTANCE ABUSE AND OTHER COMPLICATIONS

Suicide. Alcoholics have a 15 percent lifetime risk of SUICIDE. Alcohol is involved in at least 50 percent of successful suicides. Substance abusers are also recognized to have an elevated risk of suicide, and it has been reported that 70 percent of suicides in young people are associated with sub-

stance abuse. Studies of successful suicides demonstrate the ambivalent nature of this act. Alcohol or substance abuse may act as the weight that tips the scale toward suicide, or may induce the psychiatric symptoms that elicit the suicidal urge. Regardless, substance abuse is among the strongest risk factors for suicide.

Organic Brain Syndromes. A variety of organic brain syndromes, including DELIRIUM and dementia are associated with acute and chronic use of drugs and alcohol. Abrupt WITHDRAWAL from alcohol or sedative-hypnotic drugs can cause withdrawal delirium (DTs). These organic effects from drug use must be carefully separated from the psychiatric conditions discussed earlier, and from neurologic conditions which can overlap their symptoms. The impact of chronic drug use and personality is an area in need of further study.

Acquired Immunodeficiency Syndrome (AIDS). Intravenous drug use, needle sharing, and high-risk sexual practices among drug users are major risk factors for AIDS. Psychiatric manifestations of AIDS may present in a number of ways, including mood disorders, dementia, psychosis, and behavioral impairment. Suicide risk among AIDS victims is high. In evaluating the substance abuser with neuropsychiatric changes, HIV testing should be completed and treatment for AIDS should incorporate educating the patient about these risks.

SUMMARY

Substance abuse of all kinds and many psychiatric disorders have been shown very conclusively to be associated one with the other. The combination of these disorders, as generally agreed, make such individuals more difficult to treat from the standpoint of both their psychiatric and their substance-abuse problems. Research is being conducted to determine better ways of understanding the origins of these associations.

(SEE ALSO: *Conduct Disorder; Research: Mood and Drugs; Social Costs of Alcohol and Drug Abuse; Structured Clinical Interview for DSM-III-R; Vulnerability*)

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Neurological Alcohol (ethanol, also called ethyl alcohol) and other psychotropic drugs are taken because of their ability to affect the central nervous system (CNS) and thereby alter mental functioning. However, the possible reinforcing effects are offset by a cost: it is now well established that CNS structural and functional integrity can be compromised by heavy or prolonged intake of many abused substances. This article addresses the effects of alcohol and other psychotropic drugs on nervous system structure and function. It will briefly review and synthesize information from studies using various methods and technologies, including neurological examination, postmortem examination of the brain, neuropsychological tests, and neuroradiological techniques such as magnetic resonance imaging (MRI), which shows the living brain in fine detail, and positron-emission tomography (PET), which indicates the level of functioning of particular brain regions while the individual is at rest or is engaged in a cognitive task. Both acute and chronic effects of substances on brain and behavior and the reversibility of drug-related impairments are addressed. Because most of the relevant research on this issue has been conducted with ALCOHOL, more limited information is presented regarding the effects of other drugs of abuse on CNS.

ALCOHOL: ACUTE EFFECTS

BLOOD ALCOHOL CONCENTRATIONS (BAC) above the legal limit (0.08%) typically impair the operation of complex machinery, as should be obvious from public information and programs regarding DRIVING while intoxicated. The signs of intoxication, such as impaired judgment, slurred speech, and motor incoordination, are due to CNS depression. Sensitive testing also reveals impairments in a number of specific cognitive operations, including selective attention, decision making, and hand-eye coordination, at lower blood alcohol concentrations. Intoxication can increase risk-taking, aggressive, or dangerous behaviors because of diminished inhibitory control coupled with the person's inability to evaluate the consequences of his or her actions. Therefore, it is not surprising that intoxication is frequently associated with traumatic injuries, including traumatic brain injuries, and is a common factor in fatal motor vehicle accidents and violent incidents.

A binge of heavy drinking can lead to MEMORY lapses or alcoholic blackouts, in which the individual is unable to recollect events that took place during the period of intoxication even though he or she may have seemed "normal" to observers at the time. Although the pathogenesis of these episodes is not yet defined, it appears that the mechanisms underlying memory storage are temporarily disrupted during the blackout. Less severe difficulties with storage of new information can be seen even when drinking is below the legal limit of intoxication.

Very high doses of alcohol depress consciousness, leading to sleepiness, coma, respiratory depression, and death. The acute effects outlined here are clearly dose-dependent and are due to depression of successively more regions of the nervous system with the increasing dose.

ALCOHOL: TOLERANCE AND WITHDRAWAL

Dependence on alcohol, and many other drugs, is characterized by TOLERANCE and WITHDRAWAL. Tolerance refers to the fact that with chronic use, increasing doses of the drug are needed to achieve the same behavioral effects. Thus, the degree of acute impairment outlined above will vary with the individual's tolerance. People who have developed

alcohol tolerance also show cross-tolerance to other CNS depressants, including general anesthetics. Loss of tolerance appears to occur in the ELDERLY and in alcoholics who have developed organic brain impairments due to alcohol use or other factors, such as head injury. However, tolerance does not appear to develop to the direct neurotoxic effects of long-term alcohol abuse.

Following heavy drinking, many alcoholics experience a tremulous-hyperexcitable withdrawal syndrome, which is characterized by postural tremor, agitation, confusion, and ataxia. Generalized seizures can also appear in withdrawal, typically 10 to 48 hours after cessation of drinking. It has been hypothesized that long-term alcohol use may establish an epileptogenic state of the brain that becomes manifest upon alcohol withdrawal. For this reason, it has become common practice in many treatment facilities to guard against withdrawal seizures in patients with known susceptibility by giving prophylactic anticonvulsants or tranquilizers. Long-term treatment is usually not indicated because the withdrawal syndrome is self-limiting. In some patients, the acute withdrawal syndrome can progress to DELIRIUM TREMENS (DTs). This more severe form of withdrawal is characterized by delirium, HALLUCINATIONS, and a hyperautonomic state manifested by sweating and tachycardia. DTs are associated with approximately 15 percent mortality rate, possibly due to cardiac toxicity caused by the hyperadrenergic state. Treatment of the disorder involves rehydration and haloperidol (a neuroleptic drug) as well as medication to control withdrawal.

ALCOHOL: CHRONIC EFFECTS

Alcohol has direct toxic effects on neurons and, in association with other medical consequences of alcohol abuse, such as liver damage and inadequate nutrition, can result in significant and lasting cognitive deficits. There is no clear indication of the level of consumption that might put one at risk for such consequences, but "safe" drinking guidelines of no more than twelve to fourteen drinks per week probably represent a minimum level. Although there are no precise data on the incidence of neurological or cognitive impairment in alcohol abusers, it is estimated that 50 to 70 percent of individuals seeking treatment may present with some form of neurocognitive impairment. Most of these report drinking more than thirty standard drinks (each containing 13.6 grams of ethanol) per week and at least a five-year history of such use.

Victor and his colleagues (1989) have made the definitive studies of the best-known disorder associated with alcohol abuse, the Wernicke-Korsakoff syndrome (WKS). There are three major symptoms in the acute phase, known as Wernicke's encephalopathy: abnormalities of eye movements; ataxia; and a confusional state that includes poor responsivity, disorientation, and deficits in attention and memory. The disorder has been demonstrated to be caused by a thiamin (Vitamin B_1) deficiency that is probably due to decreased B_1 in the diet and decreased absorption or utilization of B_1 induced by alcohol-related gastrointestinal disorders or other mechanisms. These symptoms usually improve substantially when the patient is immediately treated with thiamin. The chronic phase, known as Korsakoff's syndrome, is marked by a profound memory deficit that includes both retrograde amnesia (an inability to recall information from the remote past) and anterograde amnesia (an inability to learn and retain new information). It should be noted that there is frequently no prior Wernicke's encephalopathy recognized in Korsakoff patients. Although there may be difficulties in other cognitive capacities, the levels of general intellectual functioning, verbal abilities, and many other specific skills remain intact in these patients. Although partial or complete recovery from the amnesia is seen in some individuals, at least 50 percent of cases show slight or no recovery.

Postmortem analysis indicates that the lesions in Korsakoff patients generally involve diencephalic areas known to be important to memory functioning. These include the mammillary bodies and the dorsomedial nuclei of the thalamus. Neuronal loss is also prominent in other areas surrounding the cerebral ventricles, such as the periaqueductal gray of the mesencephalon, hippocampus, and basal forebrain. Modern imaging techniques that permit in vivo examination of the neuropathology of WKS are consistent with the neuropathological data. Analysis of MRI scans reveals small or absent mammillary bodies, as well as more general cerebral atrophy.

Estimates of the prevalence of WKS, based on hospital records, suggest that it is relatively rare. However, it appears that the diagnosis is often missed during life, despite its seemingly dramatic

presentation. Autopsy series by Harper and his colleagues (1987) have indicated that less than 20 percent of patients with the characteristic brain lesions of WKS had been correctly identified antemortem.

A second profile of alcohol-related brain dysfunction, which is much more common than WKS, has been described by many investigators since the 1970s. These individuals may not show overt neurological symptoms, but selective impairments are seen in cognitive functions when sensitive neuropsychological tests are used. Extensive reviews of these effects are available in a book edited by Parsons, Butters, and Nathan (1987). The most prominent deficits are in complex visual-motor functions, particularly when speed of response is important. Thus, visual search, manual tracking, symbol copying, and other psychomotor functions are marked by imprecision and slowness. Problem-solving abilities, such as abstraction, hypothesis generation, and mental flexibility are also deficient. Mild deficits are apparent in new learning and memory, especially for nonverbal material. The memory difficulties are increased when the task requires the patient to use strategies for organizing and retrieving the information.

Studies of BRAIN STRUCTURES in these chronic alcoholics reveal apparent atrophy of the cortex, with enlargement of ventricles and sulci. For example, autopsy studies demonstrate that cerebral atrophy and low brain weight are associated with alcoholism, and some studies even show loss of cerebral tissue in "moderate" drinkers. Although such damage may be relatively widespread, several lines of research implicate a predominant involvement of frontal cortical regions in alcohol-related cerebral dysfunction. Autopsy studies show significant reduction in neuronal counts in the superior frontal cortex but not the motor cortex. Research with MRI and PET scanning in vivo reveals a consistent decrease in brain volume and functioning at rest in frontal regions, although most studies have implicated other brain areas as well. This evidence is generally consistent with studies using neuropsychological techniques, which also suggest impairments through tests sensitive to frontal-lobe dysfunction. However, further research is necessary to refine and correlate these different sources of evidence before strong conclusions can be made regarding selective effects of alcohol on localized cortical regions.

Many investigators recognize a more severe and global impairment in mental functioning that is different from both the "typical" picture of chronic alcoholic brain dysfunction and from WKS. This is generally referred to as alcoholic dementia, to underscore the severe and global nature of the cognitive deficits. However, it is not known whether alcoholic dementia exists as a separate pathological entity, which represents the end point of chronic alcoholism in some older individuals, or is an extension of WKS to other brain regions and cognitive domains. Some research at the end of the 1990s suggested that alcoholic dementia may be a more severe form of WKS.

There is no clear set of clinical diagnostic or neuropathological criteria for alcoholic dementia. However, a relatively high prevalence of alcohol-related dementias among residents of several long-term care institutions in northern Ontario were found. In that study, 24 percent of cognitively impaired residents fit this diagnostic profile, a figure substantially higher than had been reported previously. Given that the proportion of elderly individuals in North America is growing, we might expect an increase in research activity associated with this disorder in years to come.

ALCOHOL: RECOVERY OF FUNCTION

When alcoholics stop drinking, their presenting neurocognitive impairment can often show marked recovery over weeks to months with maintained abstinence. Substantial recovery has been demonstrated in only a minority of patients with WKS, given appropriate thiamin treatment and abstinence from alcohol use. In 1978, Carlen and colleagues were the first to report reversibility in measured cerebral atrophy on computerized tomography (CT) scans in chronic alcoholics after several months of abstinence. This reversibility has been replicated by several other research groups. Many studies have also reported recovery in the cognitive performance deficits for a majority of patients, with improvements depending critically on abstinence. In general, the more novel, complex, and rapid the information-processing requirements of the task, the longer the time for recovery to normal levels of function. As of the mid-1990s, only modest correlations between the measures of brain atrophy and cognitive functioning had been shown. The mechanisms underlying the pathogen-

esis and reversibility of cerebral atrophy remained under study.

ALCOHOL: SUMMARY AND FURTHER QUESTIONS

Chronic alcohol ingestion has potentially devastating effects on neurocognitive functioning. Impairments associated with alcohol use range from transient deficits observed in acute intoxication to potentially permanent and severe disorders, such as alcoholic dementia. There are also various other neurological conditions associated with chronic alcohol abuse. Some of these are relatively common in alcoholics, including cerebellar degeneration, peripheral neuropathies, movement disorders, and hepatic encephalopathy. Alcohol-related conditions that are relatively rare include central pontine myelinolysis, pellegra encephalopathy, and Marchiafava Bignami disease. Although some important relationships between alcohol misuse and neurocognitive functioning have been discerned since the 1970s, many important questions remain. Outstanding issues include the prevalence of impairment in the alcohol-dependent population; individual risk factors that mediate the expression of deficits; the relation between levels and patterns of consumption and resulting impairments; the rate and extent of recovery of function and treatments that may enhance recovery; precise specification of the profiles of cognitive impairment in different clinical syndromes and their relation to measures of brain damage; and implications of cognitive dysfunction for prevention and treatment of substance abuse.

OTHER CNS DEPRESSANTS

Several other classes of drugs act as depressants on the central nervous system. The profile of impairment with barbiturate intoxication largely resembles the acute effects of alcohol. Because of the relatively high abuse potential and severe withdrawal associated with these drugs, the BENZODIAZEPINES have largely displaced BARBITURATES in prescriptions for SEDATIVE-HYPNOTIC drugs. Currently the most prescribed class of PSYCHOACTIVE drugs in Western industrialized countries, they are typically used for muscle relaxation, sedation, and reduction of ANXIETY. It has become clear that benzodiazepines (e.g., Valium) can be associated with adverse behavioral changes, particularly in older individuals. In acute administration, they can cause impaired memory, slowing of reaction time and decision making, and disrupted attention. These effects are similar to those produced by drinking alcohol, and the effects of these two drugs taken together can be additive. Although patients appear to develop some tolerance to the sedating effects of benzodiazepines when they are administered for long periods, new evidence suggests that memory and cognitive impairments can remain or even increase with chronic administration. At present, it does not appear that these drugs have direct toxic effects on brain structures, so that their effects on behavior are likely mediated by a temporary and reversible pharmacological blocking of normal routes of neural information processing.

CANNABIS

Cannabis (MARIJUANA) intoxication leads to widespread changes in cognitive functioning, including disrupted attention, memory, and perceptual-motor abilities. For example, individuals may be unable to remember information learned while intoxicated, even when tested in drug-free conditions. Like alcohol, there is also impairment in the complex visual, motor, and decision-making skills needed to operate complex machinery. Numerous reports from the 1970s indicated a lack of enduring cognitive impairment associated with chronic cannabis use. However, later work reported poor learning and memory in newly abstinent users, with recovery of function documented over a six-week period. Although further research is needed, there is clearly not the same degree of brain and behavioral dysfunction that has been associated with alcohol.

OPIOIDS

Primarily used for therapeutic management of severe pain, the OPIOIDS can have a profound mood-altering euphoric effect that can lead to dependence. Administration of opioids (HEROIN, MORPHINE, or Demerol) to relatively naive users leads to a generalized depression of cognition that can be referred to as "mental clouding." Impairments in perception, learning and memory, and reasoning accompany the drowsiness and mood

changes induced by the drug. Generally, tolerance develops to the depression in mentation with chronic administration. Thus, there appears to be little long-term performance or brain dysfunction associated with this drug class.

AMPHETAMINES AND COCAINE

AMPHETAMINES and COCAINE act as CNS stimulants, which means that they generally increase arousal and psychomotor activity. As might be expected, acute administration can improve performance on many tasks, particularly when vigilance or speed of response is important. It can also reverse the effects of fatigue, which suggests that attentional resources are enhanced. However, this increase in arousal can be coupled with a dysfunction in higher-order control processes used to monitor or inhibit ongoing behavior, such that there is a corresponding increase in errors, impulsivity, and hyperactivity. Neurological consequences of a single dose of cocaine can include intracerebral hemorrhages, seizures, and strokes. These complications appear in only a small proportion of cocaine users, but the factors that place one at risk are not yet known. Although there is little research on the existence or nature of cumulative effects of chronic stimulant use, some recent evidence indicates mild impairments in memory and attention. A few studies have documented the recovery of function with abstinence and a correlation between the level of consumption and impairment, suggesting a direct relationship between drug use and behavioral deficiency. Convergent evidence for a transient disruption in brain function is provided by the work of Volkow and colleagues (1988). Their PET studies indicated decreased blood flow in the prefrontal cortex of cocaine users and an apparent return to normal levels with abstinence. In animal models, high doses of amphetamines, particularly METHAMPETAMINE, can produce damage to serotonergic and dopaminergic neurons.

SOLVENTS

Solvents are chemical compounds, such as benzene and toluene, typically used to dissolve oils or resins. Although a small proportion of young people voluntarily abuse or inhale these substances (e.g., sniffing glue or gasoline), many more individuals are exposed to them as workers in an industrial setting. These chemicals are unique in their ability to cause damage to the CNS after fairly limited exposure. Clinical observation of acute effects of these drugs shows that users experience euphoria, dizziness, and "drunkenness," which are usually accompanied by fatigue, muscle weakness, and impairments in concentration, memory, and reasoning. This can progress through loss of self-control, disorientation, and coma. Chronic neuropsychological impairments are seen in a variety of domains, including motor coordination, memory, and attention, and can resemble the symptoms of dementia. Neurological impairments include diminished sensitivity to pain and touch, shrinkage of the cortex, and lesions in the cerebellum. Although there is not yet sufficient evidence to be conclusive, it is likely that much of the damage and disruption to function are permanent.

SUMMARY

Although much is known about the effects of drugs on the brain and behavior, many important questions remain to be answered. Even given the intensive research on alcohol, there is still lack of consensus regarding the durability of impairments, the risk factors that determine individual susceptibility, and the relationship between consumption and brain damage. In particular, the separation of any neurocognitive dysfunction that may precede drug abuse from that which is consequent to chronic drug use remains an important issue that is difficult to address without prospective studies. Research attempting to discover the relationships among a given drug's effects on various indices of brain integrity is also a relatively new area and requires further elaboration. For example, only a few studies attempting to relate brain atrophy in specific regions to particular cognitive impairments in chronic alcoholics have shown significant and reliable correlations. It is also increasingly common to find that individuals will use and abuse several different drugs, yet research on the interacting effects of various drug combinations is in its infancy. In the final analysis, much of the work summarized here prepares us to ask better questions regarding the consequences of drug use on neurocognitive functioning.

(SEE ALSO: *Accidents and Injuries from Alcohol; Imaging Techniques*)

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Nutritional This entry discusses nutritional complications in alcoholics, smokers, and abusers of other addictive drugs.

ALCOHOL

Alcoholic beverages were long used as a source of nourishment for the sick, as a means of promoting appetite, and as a treatment for pain and infection—all before other means and medications were developed for these situations. Traditionally, wine and BEER were foods, used ceremonially and as part of ceremonial healing for the ailing (and pregnant) who refused or could not tolerate a solid diet. Eventually, alcoholic beverages moved from purely ceremonial occasions to a reason for social occasions in some cultures, among some classes, and for some individuals. Alcoholic beverages have a habit-forming or addictive element for some people that may become life threatening to fatal.

Use of Alcohol in Medicine: Recent History. In 1900, Atwater and Benedict reported on their experiments at Wesleyan University, which attempted to define whether ALCOHOL could actually be considered a food; they showed that alcohol is oxidized in the body and that the energy so derived can be used as a fuel for metabolic purposes. Before that, F. E. Anstie (1877) had written his treatise *On the Uses of Wine in Health and Disease*, and, in fact, the long tradition of using alcoholic beverages within the medical profession persisted into the twentieth century. Sir Robert Hutchison, a noted British physician, wrote in 1905 that there was reason *to believe* (not that there was evidence) that alcohol increases disease resistance. Alcohol was actually used to treat serious infectious disease, such as typhus, into the late 1920s—until it was shown that patients treated with milk and beef tea had greater survival rates.

The use of alcohol to treat such disease was linked to a supposition that debility would somehow be overcome and strength regained. Other than this, the major indication for alcohol was for analgesia (pain suppression). The basic analgesic properties of alcohol and alcoholic beverages were utilized for hundreds of years in the management of the injured and those requiring surgery. For example, prior to the time of anesthetics, patients were offered brandy to reduce the agonizing pain of amputations. Decline and cessation of these medical uses of alcohol came about with the development of

inhalation anesthetics and more efficient analgesics.

Alcohol, Obesity, and Wasting. In nonalcoholics, calories from alcohol are utilized as efficiently as calories from carbohydrates or fats (and alcohol provides more calories per gram than does carbohydrate). Indeed, while carbohydrate yields 4 kilocalories per gram (kcal/gm) on combustion, when alcohol is combusted in a bomb calorimeter it yields 7.1 kcal/gm. This suggests that when alcohol is consumed in addition to a diet that maintains body weight, weight gain occurs. Fictitious characters such as Shakespeare's Sir John Falstaff provide evidence that obesity was already in the 1600s considered a characteristic of heavy drinkers.

The realization came about gradually that in fact chronic heavy drinking leads not to obesity but to weight loss and an inability to sustain adequate nutritional status. Wasted alcoholics were first portrayed by artists such as William Hogarth (1697–1764) who were intent on showing both the social and medical evils of drinking gin—a recent import from Holland that became a fad. All ages and classes indulged in the new drink at all hours of the day and night. In eighteenth and nineteenth century England, when artists were portraying the physical deterioration associated with heavy gin drinking, it was assumed that drinking eventually led to wasting only because the drunkard was disinterested in food. This idea persisted into the twentieth century. By the 1940s, it was also well recognized that chronic alcoholics are malnourished because of impaired utilization of nutrients.

It is well-known today that, whereas obesity may occur in heavy eaters who consume alcohol, chronic alcoholics are undernourished. Furthermore, studies have shown that long-term, heavy consumption of alcohol in addition to food is not associated with the gain in body weight that would be expected from the calorie intake (Lieber, 1991). In addition, if dietary carbohydrate is replaced by alcohol, weight loss occurs (as in the so-called Drinking Man's Diet of the 1960s and 1970s). This energy deficit has been attributed to induction of the system that metabolizes alcohol and at the same time uses chemical energy and generates heat. Lieber, in reviewing current knowledge of the question, notes that this does not explain the fact that there is little or no weight deficit when alcohol is consumed with a very low-fat diet.

Alcohol and Malnutrition. Diet-related causes of malnutrition in alcoholics include low dietary intake of calories and nutrients—because of poor appetite, inebriation, and diversion of food dollars into support of the alcohol habit. In addition, malnutrition may be caused by impaired absorption of nutrients, poor nutrient utilization, and increased nutrient losses in body wastes. In 1940, it was suggested that ALCOHOLISM is the major cause of malnutrition in the industrialized world (Jolliffe, 1940). Malnutrition in alcoholics may be caused by impaired absorption of nutrients because of the reduced absorptive capacity of the alcohol-damaged gut. Nutrients that are poorly absorbed by alcoholics include the B vitamins—folic acid, thiamin (Vitamin B_1), and riboflavin (Vitamin B_2). Folic acid deficiency, which causes an anemia, is particularly common in heavy drinkers. Multiple nutritional deficiencies, including deficiencies of water and fat-soluble vitamins, are also common in those alcoholics who have pancreatic and liver disease. Chronic alcoholic pancreatitis (inflammation of the pancreas) develops commonly in people who consume 150 grams or more of alcohol per day for at least ten years and at the same time eat a high-fat diet. The digestive functions of the pancreas become impaired, and therefore food is not broken down into nutrients that can be absorbed. This type of pancreatitis is a major cause of malabsorption of nutrients in alcoholics. Alcoholic cirrhosis is a condition in which liver cells that are responsible for the conversion of nutrients to active forms are replaced by fibrous tissue. Cirrhosis develops slowly in heavy drinkers and is a special risk in those who consume about 35 percent or more of their total caloric intake as alcohol. Cirrhosis is the chief cause of impaired nutrient utilization (Morgan, 1982); however, cirrhosis is caused not by a nutritional deficiency but by the toxic effects of alcohol on the liver (Lieber, 1988).

Mineral and trace-element deficiencies, particularly zinc deficiency, are common in alcoholics. Contributory causes are low intake and increased losses in the urine.

Alcohol, Nutrition, and Brain Damage. Alcoholics are at risk for brain damage when they go on drinking sprees without food. Evidence exists for this condition only in Caucasians who are genetically predisposed. An acute confusional state may occur, called Wernicke's encephalopathy; this condition can be rapidly reversed if the patient is

given massive doses of thiamin, intravenously, within a period of forty-eight hours from the onset of the symptoms. If this acute condition is not treated with thiamin, a chronic state of irreversible brain damage develops, in which there is moderate to severe dementia (Victor et al., 1957).

Alcohol and Heart Disease. Through the 1990s, evidence indicated that while moderate drinking may reduce the risk of heart disease, alcohol abuse is associated with an increased risk of heart disease. Alcohol has the effect of increasing blood (plasma) levels of high-density lipoproteins (HDL)—and elevation of these blood lipids is associated with a lower risk of heart disease. In a British study (Razay et al., 1992), it was shown that women consuming a moderate amount of alcohol (1–20 gm/day) have lower fat (triglyceride) levels in their blood and higher HDL levels. The authors consider this strong evidence for supporting a lower risk of heart disease. It is important to note that in this study the women who were the moderate drinkers were slimmer than the non-drinking group. Lower body weight, found in this study among the moderate drinkers, is also a known factor in reduced risk of heart disease. Heart disease in alcoholics is due to the direct toxic effects of alcohol on heart muscle (Brigden and Robinson, 1964).

Alcohol and Osteoporosis. The formation of new bone tissue is reduced in heavy drinkers, and this causes a marked decrease in bone mass and strength, leading to severe osteoporosis. Alcohol abuse is recognized as a risk factor for osteoporosis in both men and women. Because inebriation is also associated with a high risk of falls, alcoholics who have osteoporosis are likely to sustain hip fractures. Low intake of calcium in foods is an additional risk factor for osteoporosis in alcoholics (Bikle et al., 1985).

Methods for Assessing Nutritional Status in Alcoholics. The methods required for the assessment of caloric and nutrient intake in actively drinking alcoholics include direct observation (seldom feasible outside a treatment facility) and so-called tray weigh back (also feasible only in the detoxification section of a rehabilitation facility, hospital, or nursing home). The term *tray weigh back* means weighing the food served to a patient, weighing the uneaten food, then computing intake from the difference.

When alcoholics are asked to recount what they have eaten, they tend to confabulate: When asked leading questions, they provide answers that the question indicates are correct or ideal. They may provide the questioner with an account of a make-believe diet, or they exaggerate the amounts of food they have eaten. These responses, which are worthless for the purpose of assessing the amount of calories consumed from food or for assessing the nutrients consumed, are given by alcoholics who may not remember what was eaten and also because they may want to please the dietitian, physician, or nurse seeking information. Not only do alcoholics confabulate, they may also exaggerate the amount they eat, reporting what is served to them rather than what was consumed. (This is also the type of over-reporting of food intake frequently found in people consuming other drugs that suppress appetite.)

The presence of malnutrition is assessed in alcoholics (as well as nonalcoholics) by using anthropometric (body) measurements—including weight-for-height measurements, calculation of the body—mass index (the weight/height squared), and the circumference of the upper arm and the thickness of the fat on the back of the arm. Alcoholics show muscle wasting in the upper arms, which may suggest malnutrition even when body weight is not markedly decreased. Although alcoholics with advanced liver disease are frequently wasted, weight loss may not register in numerical terms because of fluid retention within the abdominal cavity (ascites).

Biochemical measurements are valuable for assessing the nutritional status of alcoholics. The measurement of plasma albumin levels is particularly important—a value of less than 3.5 grams per 100 milliliters of plasma indicates that protein-energy malnutrition exists.

Nutrient Intolerance in Alcoholics with Liver Disease. Alcoholics with liver disease are very intolerant of high-protein diets. If high-protein diets are provided during periods of nutritional rehabilitation, such alcoholics may develop signs of liver failure. Such alcoholics are also intolerant of Vitamin A if this vitamin is taken in amounts that exceed 10,000 international units (IU) per day. Continued intake of Vitamin A at a high daily dosage level leads to further liver damage and may also precipitate liver failure (Roe, 1992).

Nutritional Rehabilitation of Alcoholics. Nutritional rehabilitation of alcoholics can be carried out successfully only when abstinence is en-

forced or the alcoholic voluntarily stops drinking. If the alcoholic has advanced liver disease or impairment of pancreatic function such that digestion and absorption of nutrients is impaired, optimal nutritional status cannot be maintained. The goal of nutritional rehabilitation is the treatment of existing protein-energy malnutrition by increasing caloric intake from carbohydrates and the treatment of existing vitamin, mineral, and trace-element deficiencies. Appetite returns after alcohol withdrawal symptoms have abated; however, recovery of efficient absorption of vitamins may not occur until ten to fourteen days after drinking ceases. Initially, intolerance of milk and other dairy foods is common during rehabilitation, because of lactose intolerance, and extreme caution has to be exercised in diet prescription because of protein intolerance (Roe, 1979).

TOBACCO

Smoking diminishes appetite and on average, smokers have lower body weights than nonsmokers. Nevertheless, on average, smokers have greater waist-to-hip circumference ratios than nonsmokers. This suggests that smoking may have an effect on body-fat distribution. Central (torso) adiposity, reflected by this change in circumferential measurements, has been shown to worsen the risk of cardiovascular disease. Cessation of smoking is usually associated with moderate weight gain, caused at least in part by increased food intake (Troisi et al., 1991).

OTHER ADDICTIVE DRUGS

Multiple Substance Abuse and Nutrition. Drug abuse includes the experimental use of various addictive drugs as well as chronic addiction to one or more of these social drugs. The term *addiction* here refers both to PHYSICAL DEPENDENCE on the drug, such that when the drug is withdrawn specific physical withdrawal symptoms occur, and to PSYCHOLOGICAL DEPENDENCE on the drug—even without physical dependence. Alcohol has been called *the* GATEWAY DRUG, because its early use is frequently accompanied by and/or followed by use of other drugs.

Effects of multiple-drug use on nutrition depend on the properties and toxic characteristics of the drug most used, as well as on doses, frequencies, and duration of use/abuse, and the time in life when the drug or drugs are abused. NARCOTIC drugs, such as HEROIN, impair appetite—so food intake is often diminished. If the drug is injected intravenously, malnutrition may be secondary to blood-borne bacterial infection or ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS). AMPHETAMINE ("speed") is the stimulant drug that has the most inhibitory effect on appetite; if taken in large doses, it also prevents sleep and stimulates activity—therefore energy expenditure may be high and weight loss is common. COCAINE and CRACK are also stimulants, they reduce appetite and may in addition induce gastrointestinal symptoms such as nausea, which further lessen food intake (Brody et al., 1990).

Substance Abuse and Nutrition in Pregnancy. Relationships between substance abuse and impaired nutrition of the fetus and newborn have been summarized in a 1990 report by the National Academy of Sciences, *Nutrition during Pregnancy*.

Alcohol use during PREGNANCY has led to poor birth outcomes. One condition is infants with specific defects in neuronal and cranial development, designated FETAL ALCOHOL SYNDROME. Even the daily drinking of more than two glasses of wine or a daily mixed drink has led to fetal alcohol syndrome, but this condition is most common among the offspring of mothers who are chronic drinkers or binge drinkers.

Alcohol use during pregnancy is also known to be associated with prenatal and postnatal growth retardation. After birth, infants of heavy drinkers may fail to suck, either because of the presence of withdrawal symptoms or because of cleft palate (which may be part of the fetal alcohol syndrome).

Cigarette smoking during pregnancy can affect both maternal and fetal nutrition (Werler et al., 1985). Effects are due to increased metabolic rate in smokers and to toxic effects from tobacco that impair the mother's utilization of certain nutrients, including iron, Vitamin C, folic acid (part of the B complex), and zinc. Low-birthweight infants are more likely to be the offspring of smokers than of nonsmokers—because their caloric intake is likely to be less and because the transfer of nutrients from the mother to the fetus via the placenta may be reduced in smokers.

Cocaine and amphetamine use in pregnancy also lead to increased numbers of low-birthweight in-

fants. This may be caused by low food intake by the mother, since these drugs reduce appetite. The risk of malnutrition in the newborns of women who have used cocaine during pregnancy is caused by the abnormal development of the infant's small intestine. These intestinal disorders in the infant may be extremely severe and may be associated with enterocolitis or bowel perforation, which may be fatal. If these infants survive, special methods of feeding via a vein are required. Although drugs other than cocaine are known to cause constriction of blood vessels in the pregnant woman, none other than cocaine have been shown to produce these bowel disorders in infants (Telsey et al., 1988; Spinazzola et al., 1992).

(SEE ALSO: *Alcohol History of; Complications: Liver* [*Alcohol*]; *Overeating and Other Excessive Behaviors*)

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DAPHNE ROE
REVISED BY MARY CARVLIN

Route of Administration The mode of drug administration—ingestion (by mouth), insufflation (snorting), inhalation (smoking), or injection (intravenous, subcutaneous, or intramuscular)—can be responsible for a number of medical complications to alcohol and other drug use. In the following, these complications are discussed as direct and indirect results of the various modes (route) of administration in the above order.

COMPLICATIONS DUE TO INGESTION

Ingestion is the way ALCOHOL, liquid medicines, pills and capsules are usually taken. Ingested drugs enter the gastrointestinal (GI) system, undergo some digestive processing, and enter the bloodstream through the walls of the stomach and intestines. Most medical complications from drug ingestion are a result of the corrosive and irritant effects of the drugs on the GI system. Alcohol and a variety of medicines, including aspirin, can cause intense, localized irritation to the GI mucous membranes,

leading to ulceration and GI bleeding. Pharmaceutical manufacturers attempt to decrease the danger of GI irritation by adding buffers to their pills and capsules. Buffers are inert or nonactive ingredients that cushion the corrosive effect of the active ingredients. However, if drug users attempt to dissolve pills intended for oral use and inject them, these buffers will often cause problems, such as abscesses or embolisms.

COMPLICATIONS DUE TO INSUFFLATION (SNORTING)

Medical complications from insufflation (snorting) are usually caused by stimulant drugs, such as the AMPHETAMINES or COCAINE. These drugs are breathed into the nose and absorbed into the bloodstream through the capillaries in the nasal mucous membrane. While these drugs cause a certain amount of surface irritation, the major damage is caused by their action as vasoconstrictors—they reduce the diameter of blood vessels, and with chronic use can severely limit the delivery of blood through the capillaries to the inner membranes of the nose. The result of this is that tissue damaged by contact with the drugs is unable to repair itself, and progressive necrosis (tissue death) follows. With chronic cocaine use, this process can result in actual holes through the septum (the dividing tissue) between the nostrils. When tobacco is insufflated as snuff, the risk of cancer of the nasal passages is increased.

COMPLICATIONS DUE TO SMOKING (INHALING)

The fastest delivery of large amounts of drug directly to the brain is through smoking (inhaling). Drugs taken in this way go directly to the lungs and are absorbed along with oxygen directly into the blood heading for the brain. The two terms, *smoking* and *inhaling*, as a means of drug intake, are clearly differentiated when, on the one hand, material is actually burned and the resulting smoke is taken into the lungs—as with TOBACCO or MARIJUANA—or on the other hand, when fumes from volatile substances are inhaled, such as glue or gasoline. They may be confused or used interchangeably, however, when material is vaporized through heat and the vapor is inhaled—as with cocaine FREEBASE (crack).

Smoking. Smoke from any material will act as an irritant to the lungs and bronchial system, eventually causing problems that can range from chronic bronchitis to emphysema or cancer of the mouth, throat and/or lungs. Both tobacco and marijuana contain a number of tars and potential carcinogens (cancer-triggering substances) and both produce potentially toxic concentrations of carbon monoxide. While it has been argued that tobacco is the worst danger because it is smoked very frequently, it has also been pointed out that the use mode of marijuana is worse—holding the smoke in the lungs for a long time. The argument is moot, since both can produce profound damage. As a vasoconstrictor, nicotine in tobacco promotes mouth ulceration and gum disease. It can be said that people who smoke lose their teeth, while those who don't, don't. Besides its irritant effects, the smoking of tobacco may also promote respiratory disease by weakening the immune system and by paralyzing the cilia (the tiny hairlike organs) in the lungs that push out foreign matter.

Inhalation (Sniffing). The inhalation (sniffing) of volatile hydrocarbons, such as solvents, can cause death by asphyxiation or suffocation, can impair judgment, and may produce irrational, reckless behavior. Abnormalities also have occurred in liver and kidney functions, and bone-marrow damage has occurred. These may be due to hypersensitivity to the substances or chronic heavy exposure. Chromosome damage and blood abnormalities have been reported, and solvents have been cited as a cause of gastritis, hepatitis, jaundice, and peptic ulcers—such effects are due more to the actions of the drugs than to the route of administration. Chronic users have developed slow-healing ulcers around the mouth and nose, loss of appetite, weight loss, and nutritional disorders. Irreversible brain damage has been reported, too. Many deaths attributed to solvent inhalants are caused by suffocation when users pass out with the plastic bags containing the substance still glued to their noses and mouths. There is also a very real danger of death from acute solvent poisoning or aerosol inhalation. The mere provision of adequate ventilation and the avoidance of sticking one's head in a plastic bag are by no means sufficient safeguards against aerosol dangers.

Other hazards may include freezing the larynx or other parts of the airway when refrigerants are inhaled, and potential spasms as these areas de-

frost. Blockage of the pulmonary membrane, through which oxygen is absorbed into the lungs, can occur. Death may also result from the ingestion of toxic ingredients along with the aerosol substance. The possibility is made more likely by the fact that commercial products not produced for human consumption are not required to list their ingredients on the label. Individual substances may produce a spectrum of toxic reactions depending on their contents. These have included gastric pain, headaches, drowsiness, irritability, nausea, mucous-membrane irritation, confusion, tremors, nerve paralysis, optic-nerve damage, vomiting, lead poisoning, anemia, and so on. The inhaling of aerosol fluorocarbons can cause "sudden-sniffing death" (SSD), wherein the heart is hypersensitized to the body's own hormone epinephrine (adrenaline), leading to a very erratic heartbeat, increased pulse rate, and cardiac arrest.

The inhaling of amyl, butyl or isobutyl nitrites can cause intense headaches, an abrupt drop in blood pressure, and loss of consciousness through orthostatic hypotension (increased heart rate and palpitations), with a threat of myocardial infarction (heart attack).

COMPLICATIONS DUE TO INJECTION

The injection of drugs generally involves the use of the hypodermic needle, first invented in the early nineteenth century and used initially for the medical delivery of the opiate painkiller MORPHINE, for the rapid control of intense PAIN. This combination was first used extensively for battlefield wounds during the Crimean War (1853–1856) and the American Civil War (1861–1865). As its name implies, the hypodermic needle pierces the skin—the dermis. Hypodermic injections may be subcutaneous, directly beneath the skin surface; intramuscular, into the muscle tissue; or intravenous, into a blood vessel. (*Note*: Although a number of injection-related medical complications are directly skin-related, these are discussed in the article *Complications: Dermatological*.

While the hypodermic needle is the primary means of drug injection, drug addicts who do not have access to hypodermics have made use of a number of ingenious, and often very dangerous, substitutes. Nonhypodermic-needle means of injection may involve such paraphernalia as lancets or scalpels, or any small sharp blade to make an opening, and the insertion of an eyedropper, tubing and bulb, or any means of squirting the drug into the resultant wound. In extremes, addicts have used such implements as a pencil, ballpoint or fountain pen, or the sharpened end of a spoon.

Intra-arterial Injection. Injections are never made intentionally into arteries. Accidental intra-arterial injection will produce intense pain, swelling, cyanosis (blueness), and coldness of the body extremity injected. Intra-arterial injection resulting in these symptoms is a medical emergency and, if untreated, may produce gangrene of the fingers, hands, toes, or feet and result in loss of these parts.

Transmittal of Disease through Injection. The greatest number and variety of medical complications of drug use caused by the mode of administration occur as a result of injection. Among the highest risk, and that with the most frequent fatal and disabling consequences is the transmittal of disease through the use of unsterile needles and the sharing of such needles.

Human Immunodeficiency Virus (HIV). Needle-using drug abusers comprise one of the primary high-risk populations for contracting human immunodeficiency virus (HIV). The primary recognized routes of transmission for HIV are (1) sexual contact through unprotected anal or vaginal intercourse—particularly if there are damaged tissue or sores present that provide direct access to the bloodstream; (2) contact with infected blood through needle sharing or through transfusions of blood or blood products; and (3) in utero or at-birth transmission from a mother to her baby. ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS), the most severe and life-threatening result of HIV infection, involves the destruction of a person's immune system and the development of cancers and infections that can no longer be fought off.

The incidence of HIV infection among needle-using drug abusers is closely related to local use traditions, habits, and the prevalence of HIV infection among other addicts. The highest incidence is in areas such as New York City, where there is a tradition of needle sharing or where "shooting galleries"—places where users can rent or share "works"—are commonly utilized and where there was a high prevalence of HIV among the homosexual population. Users in other geographical locations, such as San Francisco, seem to be more conservative in their social-usage patterns, and when they do share needles, tend to keep the same

"shooting partners" over a longer period of time. HIV-prevention efforts in some areas have focused on NEEDLE AND SYRINGE EXCHANGE, while others, particularly where needle exchange is not legalized, have community-outreach workers teaching users how to sterilize their needles between each use with household bleach. The gist of both campaigns is that users who share their needles or who use dirty needles are at risk for contracting HIV through their drug use. Those who use sterile needles are not. Both approaches are considered stopgap, however, and are apt to be condemned as "encouraging of drug abuse."

All needle-using drug abusers are considered at extremely high risk for HIV infection, and HIV screening is performed routinely at most drug-treatment centers. The virus has a very long incubation period and may be present for seven or more years before active symptoms of opportunistic disease appear. Early symptoms may include: a persistent rash or lesion; unexplained weight loss; persistent night sweats or low-grade fever; persistent diarrhea or fatigue; swollen lymph glands: DEPRESSION or states of mental confusion.

Hepatitis and Other Liver Disorders. Hepatitis B, and related strains, often referred to as serum (fluid-related) hepatitis, are the most common medical complication of needle drug use. Like HIV, hepatitis can spread in other ways than needle use, such as sexual intercourse or other direct sharing of blood and bodily fluids. Several strains, however, can be spread by contaminated foods, particularly shellfish, or by unhygenic practices in food handling. Current research indicates that some forms of hepatitis spread via an anal/oral progression—so it is recommended that hands are washed thoroughly after all bowel movements or any other anal-area or fecal-matter handling, as a means of prophylaxis.

Unlike AIDS, hepatitis is often not fatal if it is detected and treated at an early stage. Symptoms of all forms of hepatitis include fatigue, loss of appetite, pain in the upper abdomen, jaundice—yellow skin and a yellowish-to-chartreuse tinge to the sclerae (white of the eye), general itching, dark urine reaching the color of cola drinks with light-tan to cream-colored feces, and mental depression. Gamma globulin injection can provide short-term immunity to all forms of hepatitis and can reduce the symptoms of serum hepatitis if it is given during the gestation period. Treatment includes bed rest, nutritional support, and avoidance of alcohol or any other substance that may further irritate the liver. Caregivers should wear rubber gloves for handling patients. Patients with any form of hepatitis should avoid preparing food for others and use separate towels, bedlinens, and eating utensils until symptoms disappear. Toilet seats and any spilled bedpan matter should be disinfected and hands should then be washed thoroughly with soap. Condoms should be used for any genital contact.

Hepatitis can cause hepatic fibrosis—the development of fibrous tissue in the liver. It can also cause or exacerbate cirrhosis (scarring of the liver), although this is most often a result of chronic alcohol abuse. Symptoms of cirrhosis include jaundice (yellowish skin and eye whites), fatigue, ankle swelling, enlargement of the abdomen, and a full feeling in the right upper abdomen.

Tetanus and Malaria. According to Senay and Raynes, the first case of tetanus associated with needle-using substance abuse was reported in England in 1886. By the 1990s, between 70 and 90 percent of tetanus cases have occurred to drug abusers. As a medical complication to drug injection, tetanus most often occurs from "skin-popping"—which is cutaneous injection. A majority of cases occur in women, and this is attributed to less-substantial venous development than in men and a smaller population with tetanus immunization.

Malaria (caused by the *Plasmodium* parasite) was first reported among drug users in the United States in 1926. It affects intravenous drug abusers and was brought to this country by needle-sharing sailors who had been exposed to malaria in Africa. The initial outbreak in New Orleans spread to New York City in the 1930s and resulted in several hundred deaths from tertian malaria among drug abusers. A second outbreak occurred in the 1970s, as a result of malaria-infected veterans returning from Vietnam.

The spread of both these diseases among needle-sharing drug abusers has been kept somewhat in check, particularly on the East Coast and in Chicago, by the inclusion of 15 to 30 percent quinine (a natural antimalarial), as filler, to stretch profits in illicit opioid drug mixtures in those areas. Quinine (an alkaloid from chinchona bark) is a protoplasmic poison that prevents the germination of the fastidious tetanus anaerobe, *Clostridium tetani*, under the skin and in adjacent muscle tissue. Although the quinine amount is not sufficient to erad-

icate malaria once it has taken hold in the body, it does help prevent the disease by killing the malarial parasites in the hypodermic syringe.

COMPLICATIONS TO HEART AND BLOOD VESSELS

Drug abuse is related to a number of heart and blood vessel medical complications. Some of these, such as alcohol cardiomyopathy, are a direct result of the drug's toxic effects. Others are at least partially related to needle use.

Endocarditis, an infection of the tissues in the heart, usually a heart valve, is a progressive disease characterized by frequent embolization (obstruction of blood vessels) and severe heart-valve destruction that can be fatal if not treated. This disease can result from repeated injection of the infective agents into the blood system, usually from nonsterile needles and/or unusual methods of injection. Infective endocarditis is highly prevalent among drug abusers and should be suspected in any needle-using abuser who shows such symptoms as the following: fever of unknown origin; heart murmur; pneumonia; embolic phenomena; blood cultures that are positive for *Candida*, *Staphylococcus aureus* or *enterococcus*, or Gram-negative organisms.

MISCELLANEOUS COMPLICATIONS

Blood-vessel changes caused by necrotizing angiitis (polyarteritis—the inflammation of a number of arteries) or a swelling that leads to tissue loss have been demonstrated in intravenous amphetamine abusers, resulting in cerebrovascular occlusion (blockage in brain blood vessels) and intracranial hemorrhage or stroke.

Problems in the lungs often develop from inert materials that are included as cutting agents or as buffers and binding agents in drugs that come in pill form but are liquified and injected. These substances do not dissolve, so their particles may become lodged in the lungs, causing chronic pulmonary fibrosis and foreign-body granulomas. These same buffers and binding agents may as well become lodged in various capillary systems, including the tiny blood vessels in the eye.

Finally, injection-induced infections reaching the skeleton can be responsible for such bone diseases as septic arthritides and osteomyelitis. Gangrene can develop from cutting off circulation to the extremities and may necessitate amputation or be fatal.

(SEE ALSO: *Inhalant Abuse and Its Dangers; Needle and Syringe Exchange and HIV/AIDS*)

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COMPULSIONS A compulsion is a persistent, irresistible impulse to perform a repetitive, irrational behavior or mental act. Common behavioral compulsions include hand-washing, cleaning, checking, ordering, and touching. Common mental act compulsions include counting, praying, and repeating words silently. Compulsive acts may need to be performed to exacting specifications. The goal of compulsive behaviors or mental acts is to prevent or reduce anxiety. There is no pleasure or gratification derived from performing the compulsive behavior or mental act. Often, the person feels compelled to perform the compulsive act in order to reduce the anxiety associated with an obsessive thought. Alternatively, compulsive acts are performed as a way to prevent a feared event or situation. Compulsions are excessive (e.g. washing the hands until the skin is raw in order to relieve obsessive fears of contamination) or they are unrelated to the obsessive thought they were designed to negate or prevent.

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COMPULSORY TREATMENT *See* Civil Commitment; Coerced Treatment for Substance Offenders; Narcotic Addict Rehabilitation Act (NARA)

CONDITIONED TOLERANCE *Tolerance* refers to the diminishment or the loss of a drug effect over the course of repeated administrations. Some researchers have postulated that an important factor in the development of tolerance is Pavlovian conditioning of drug-compensatory responses. The administration of a drug may be viewed as a Pavlovian conditioning trial. The stimuli present at the time of drug administration are the conditional stimulus (CS), while the effect produced by the drug is the unconditional stimulus (UCS). Many drug effects involve disruption of the homeostatic level of physiological systems (e.g., alcohol lowers body temperature), and these disruptions elicit compensatory responses that tend to restore functioning to normal levels. The compensatory, restorative response to a drug effect is the unconditional response (UCR). Repeated administrations of a drug in the context of the same set of stimuli can result in the usual predrug cues coming to elicit as a conditional response (CR) the compensatory, restorative response. The conditional drug-compensatory CR would tend to reduce the drug effect when the drug is administered with the usual predrug cues—thus accounting for tolerance, or at least some aspects of tolerance.

One test of the Pavlovian conditioning model of tolerance is whether conditional drug-compensatory responses are elicited by predrug cues. In one experiment with rats (Crowell, Hinson, & Siegel, 1981), injections of alcohol in the context of one set of stimuli were alternated with injections of saline solution in the context of a different set of stimuli for several days. Each day, the rats' body temperatures were measured. Alcohol lowered body temperatures the first time it was given, but this effect diminished over the course of the repeated alcohol administrations—that is, tolerance developed to the hypothermic effect of alcohol. To determine if a drug-compensatory CR was elicited by the usual predrug cues, the rats were given a placebo CR test. In a placebo CR test, saline solution is administered instead of the drug. The placebo CR test was given to some rats under conditions where they were expecting alcohol; that is, saline was administered with the usual predrug cues. For the remaining rats, the placebo CR test was given under conditions where there should have been no expectancy of alcohol, that is saline was administered with cues that had previously signaled only saline. Rats given saline with the usual predrug cues had elevated body temperatures, while rats given saline without the usual predrug cues showed little temperature change. Thus, it was possible to directly observe the drug-compensatory CR, in this case hyperthermia opposed to the hypothermic effect of alcohol. Other experiments similar to the one just described have found drug-compensatory CRs following the development of tolerance to various effects of Opiates, Barbiturates, and Benzodiazepines (Siegel, 1983).

Conditioned responses occur only when the conditional stimulus is presented. If drug-compensatory CRs contribute to tolerance, then tolerance should only be evident in the presence of the usual predrug cues that are the CS. This expectation was tested in the experiment by Crowell, Hinson, and Siegel (1981), involving tolerance to the hypothermic effect of alcohol. After all rats had developed tolerance to the hypothermic effect of alcohol, a test was given in which some rats received alcohol with the usual predrug cues, while other rats received alcohol when the usual predrug cues were not present. Although all rats had displayed tolerance prior to the test, only those rats given alcohol in the presence of the usual predrug cues (i.e., with the CS) showed tolerance during the test. The explanation of this "situational specificity" of tolerance is that when alcohol is given with the usual predrug cues, the drug-compensatory CR occurs and reduces the drug effect—but when alcohol is given without the usual predrug cues, the drug-compensatory CR does not occur and the drug effect is not

reduced. Other research has demonstrated situational specificity with regard to tolerance to opiates, barbiturates, and benzodiazepines (for a complete review see Siegel, 1983).

In order to eliminate a CR, it is necessary to present the CS not followed by the UCS, a procedure termed *extinction*. Research indicates that the loss of tolerance occurs as a result of extinction of drug-compensatory CRs. Again referring to the experiment of Crowell, Hinson, and Siegel (1981), rats were given alcohol in the presence of a consistent set of cues until tolerance developed. Then, all drug injections were stopped for several days. During this period some animals were given extinction trials, in which the usual predrug cues were presented but only saline was injected. The other animals did not receive extinction trials and were left undisturbed during this time. Subsequently, all animals were given a test in which the drug was given with the usual predrug cues. The animals that had received extinction trials were no longer tolerant, whereas animals that had not been given extinction trials retained their tolerance. Similar results—in which tolerance is retained unless extinction trials are given—occur for tolerance to opiates, barbiturates, and benzodiazepines (Siegel, 1983).

The drug-compensatory CRs that contribute to tolerance may also be involved in withdrawal-like symptoms that occur in detoxified drug addicts. Detoxified addicts often report experiencing withdrawallike symptoms when they return to places where they formerly used drugs, although they are now drug free. The places where the addict formerly used drugs act as CSs and still elicit drug-compensatory CRs; even when the addict is drug free, the drug-compensatory CRs achieve expression. Thus, it is postulated that the drug-compensatory CRs elicited by the usual predrug cues in the drug-free postaddict result in a withdrawal-like syndrome (Hinson & Siegel, 1980). This conditional postdetoxification withdrawal syndrome may motivate the postaddict to resume drug taking (to alleviate the symptoms).

(SEE ALSO: *Addiction: Concepts and Definitions; Causes of Drug Abuse: Learning; Tolerance and Physical Dependence; Wikler's Pharmacologic Theory of Drug Addiction*)

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CONDUCT DISORDER AND DRUG USE

A behavior pattern characterized by such behaviors as stealing, violence, running away from home, and truancy occurs in about 10 percent of children under 16 years of age. Within the framework of the *DIAGNOSTIC AND STATISTICAL MANUAL of Mental Disorders*, 4th edition, (DSM-IV), this serious and persistent pattern of antisocial behavior is diagnostically labeled *conduct disorder* (CD). CD is the most common psychiatric disorder in emotionally disturbed youth, present in about 75 percent of cases. Boys outnumber girls in ratios of 4:1, but 8:1 for property and violent crimes. Emerging evidence suggests, however, that the gender gap is narrowing; and by adolescence, commonly associated problems include alcoholism, drug addiction, criminality, incarceration, sexually transmitted diseases (STDs), pregnancies, prostitution, traumatic injuries, dropping out of school, and comorbid psychiatric disorders.

DIAGNOSIS

In the American Psychiatric Association classification system for diagnosing mental disorder (DSM-IV), conduct disorder is defined as "a repetitive and persistent pattern of conduct in which the basic rights of others or major age-appropriate societal norms or rules are violated" (American Psychiatric Association, 1994). Conduct disorder has become one of the most valid and reliably diagnosed psychiatric disturbances. The problem behavior is transsituational—it is manifested in the

home, at school, and in daily social functioning. Often, CD youth are suspicious of others and, consequently, they misinterpret the intentions and actions of others. By adolescence, aggression may become so severe that violent assault, rape, and homicide are committed. Precocious sexual behavior and sexual misbehavior, especially among females, are also common. Denial and minimization generally occur when the youngsters are confronted about their behavior. Typically, feelings of guilt are not experienced.

Other, less severe, types of behavior disorders are also known. The most common that resemble CD are

(1) adjustment disorder with disturbances of conduct;
(2) childhood (or adolescent) antisocial behavior; and
(3) oppositional defiant disorder.

Substantial differences in the behavioral manifestations of CD have prompted efforts to develop subtypes. The most well-known subtyping criteria are

(1) socialized versus unsocialized;
(2) aggressive versus nonaggressive; and
(3) overt versus covert.

Just one variant of CD, the solitary aggressive type, characterizes approximately 50 percent of incarcerated youth; they are usually socioeconomically disadvantaged and typically derive from dysfunctional families. Moral development is arrested, cognitive abilities are low, and behavior is often dangerous both to self and others. This CD variant should not be confused with *adaptive delinquency*, in which the behavior is an attempt to adjust to the manifold disadvantages of inner-city living.

NATURAL HISTORY

Other psychiatric disorders frequently occur in conjunction with conduct disorder. The most prevalent comorbid (coexisting) psychiatric disorder is *attention deficit disorder*. By adolescence, the comorbid conditions of psychoactive substance use disorder with depressive disorders often emerge; however, virtually any type of psychiatric disorder can be present concurrently with CD (Rutter, 1984). By adulthood, an ANTISOCIAL PERSONALITY disorder is the most common outcome of CD; this disorder may also be accompanied by any other psychiatric disorder.

Among those who have CD with attention deficit disorder, the onset age of behavior problems tends to be earlier and more severe than in cases with either disorder alone. In the situation where both are present, children are also at greater risk for developing criminal behavior and substance abuse by adolescence or young adulthood.

The coexistence of CD and substance abuse has been frequently observed. It is estimated that as many as 50 percent of serious offenders are substance abusers. In these cases, CD usually preceded the onset of substance abuse. Some evidence has been marshaled to suggest that, for many individuals, substance abuse and CD are the overt expressions of a common underlying predisposition. Only in some cases, does the onset of CD follow the onset of substance abuse. Drug use during adolescence, by virtue of its pattern of illegal behavior plus association with nonnormative peers, increases the risk for violent assault as well as getting arrested and convicted for drug possession or distribution. In effect, the use of drugs in this circumstance socializes a person to a deviant lifestyle by early to mid-adolescence.

Approximately 30 percent of boys with CD also qualify for a diagnosis of DEPRESSION. In this comorbid condition, there appears to be a lower risk of depression in adulthood compared to cases of depression in childhood without CD. Since the outcome of depressed children with comorbid CD is similar to nondepressed CD children, this suggests that the affective disturbance is a secondary condition.

CD in childhood is associated with an increased risk for antisocial personality disorder in adulthood. Compared to other psychiatric disorders of childhood, CD is the most likely to remain stable. Persistence of conduct problems into adulthood is most likely if the behavior problems are serious, are generalized across multiple environments or situations, have an early age onset, and lead the person into the criminal-justice system (Loeber, 1991).

ETIOLOGY

Adoption and family studies implicate a genetic predisposition for the development of antisocial behavior in many. A genetic propensity does not, however, appear to invariably ensure this adverse

outcome. Other complicating factors include being a child in a dysfunctional family where the parents are abusive, neglectful, or absent or where there are poor parenting skills. Alcoholic and physically abusive parents have been frequently linked to CD in their own childhoods. Neurologic injuries (e.g., trauma) and neurodevelopmental disability (e.g., dyslexia) can exacerbate the expression of CD. Socioeconomic and ethnic factors (e.g., POVERTY, street GANGS) also influence the development of CD.

TREATMENT

The following are generally inadequate for the treatment of youth with CD: individual psychotherapy, behavior modification, group counseling, family therapy, milieu therapy, and immersion in a long-term therapeutic community. The most promising approaches emphasize training parents in the skills necessary to promote normal socialization in their children, accompanied by training children in the use of problem-solving strategies (Kazdin et al., 1987). The complexity and severity of CD-associated problems dictate the need for multimodal treatment. Primary consideration should be given to containment and limit setting—which create the conditions for treatment, to provide safety, and to delineate a comprehensive program of intervention encompassing behavioral and social-skill building, family therapy, and educational assistance.

(SEE ALSO: *Adolescents and Drug Use; Crime and Drugs; Families and Drug Use; Family Violence and Substance Abuse; Vulnerability As Cause of Substance Abuse*)

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CONDUCT DISORDER IN CHILDREN

One of several childhood behavioral disturbances, CONDUCT DISORDER refers to repeated patterns of conduct by a child that violate the basic rights of others or transgress age-appropriate societal rules. The behavior is socially disruptive and generally more serious in its consequences than typical childhood mischief. The duration of the behavior, its severity, and the kinds of actions involved distinguish conduct disorder from general misbehavior. Conduct disorder is the most common behavioral problem seen in child psychiatric settings in North America.

The behaviors that characterize this disorder include theft, vandalism, physical fights—sometimes with weapons—fire setting, running away from home, truancy, repetitive lying, forcing sexual activity on others, physical cruelty to animals and to people, and substance abuse. Legal involvement may ensue. Different children may manifest different combinations of these behaviors, and these in turn may change at different points of child development. Conduct disorder appears to be more common in boys than in girls.

The etiology of conduct disorder is considered multifactorial. Psychological and social factors believed to contribute to its development include the child's particular temperament, a family history of ANTISOCIAL PERSONALITY disorder or alcohol dependence (or both), poor parenting skills, a chaotic home environment, and lower socioeconomic status. Mild central nervous system abnormalities have been found in children with a history of violent behavior, and they are thought to contribute to the children's impulsivity. ATTENTION-DEFICIT HYPERACTIVITY DISORDER and specific developmental disorders are common associated diagnoses. Children who display significant antisocial behavior have a poorer long-term prognosis with greater psychiatric impairment in adulthood (including antisocial personality disorder), poorer educational

achievement, overt criminal behavior, higher rates of unemployment, impaired social functioning—and considerably higher rates of smoking and alcohol abuse, illicit drug use and dependence.

Treatment of conduct disorder in children and adolescents can include family therapy, parent management training, behavioral and cognitive therapies, residential treatment programs, and, less frequently, pharmacotherapy.

(SEE ALSO: *Crime and Drugs*)

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CONTROLLED SUBSTANCES ACT OF 1970 Until 1970, psychoactive drugs were regulated at the federal level by a patchwork of statutes enacted since the turn of the century. These statutes were shaped by an evolving conception of congressional power under the U.S. Constitution. The first federal law on the subject was the Pure Food and Drug Act of 1906, which required the labeling of substances such as patent medicines if they included designated NARCOTICS (e.g., OPIATES and COCAINE) and were shipped in interstate commerce. In 1909, Congress banned the importation of smoking opium. Then in 1914, in the HARRISON NARCOTICS ACT, Congress deployed its taxing power as a device for prohibiting the distribution and use of narcotics for nonmedical purposes. (The taxing power was used because U.S. Supreme Court decisions implied that Congress would not be permitted to use its power to regulate interstate commerce in banning "local" activities, such as the production and distribution of narcotics.) The scheme established by the Harrison Act required the registration and payment of an occupational tax by all persons who imported, produced, or distributed narcotics; it imposed a tax on each transaction; and it made it a crime to engage in a transaction without paying the tax. Mere possession of narcotics without a prescription was presumptive evidence of a violation of the act. The Marihuana Tax Act of 1937 utilized the same model.

In 1965, Congress prohibited the manufacture and distribution of "dangerous drugs" (stimulants, depressants, and hallucinogens) for nonmedical purposes. By this time, Congress's constitutional authority to enact such legislation under the commerce clause was no longer in doubt. (In 1968, Congress made simple possession of the drugs a misdemeanor.) All important feature of the 1965 "dangerous drug" legislation was its delegation of authority to the secretary of Health, Education and Welfare (HEW) to control previously uncontrolled

drugs if they had a "potential for abuse" due to their depressant, stimulant, or hallucinogenic properties. (In 1968, this scheduling authority was transferred to the U.S. attorney general.)

All this legislation was replaced by a comprehensive regulatory structure in the 1970 Controlled Substances Act (CSA). Under the new statutory scheme, all previously controlled substances were classified—in five schedules—according to their potential for abuse and accepted medical utility; an administrative process was then established for scheduling new substances, building on the model of the 1965 act. Schedule 1 lists drugs that have no traditional recognized medical use, such as HEROIN, LSD, and cannabis (MARIJUANA). Schedule 2 lists the drugs with medical uses that have the greatest potential for abuse and dependence, such as MORPHINE and cocaine. The remaining schedules use a sliding scale that balances each drug's ABUSE POTENTIAL and its legitimate medical uses.

Different degrees of control are applied to manufacturers, distributors, and prescribers—depending on the schedule in which the drug has been placed. The regulatory structure of the Controlled Substances Act is predicated on the assumption that tighter controls on legitimate transactions will prevent diversion of these substances and will thereby reduce the availability of these substances for nonmedical use.

The drafting of the Controlled Substances Act reflected a continuing controversy regarding the locus of administrative authority for scheduling new drugs and for rescheduling previously controlled drugs. Under the bill passed by the Senate, this responsibility would have rested with the U.S. attorney general, who was required only to "request the advice" of the secretary of HEW (now Health and Human Services, HHS) and of a scientific advisory committee; the attorney general was not required to follow this advice although the various criteria in the act require primarily scientific and medical judgments. The Senate rejected an amendment that would have made the recommendations of the "advisor" binding on the attorney general. Under the bill passed in the House of Representatives, however, the secretary's decision declining to schedule a new drug was binding on the attorney general, and the secretary's recommendation concerning rescheduling was binding as to its medical and scientific aspects. The House version prevailed in the 1970 law as it was finally adopted.

After enactment of the federal Controlled Substances Act, the National Conference of Commissioners on Uniform State Laws promulgated a Uniform Controlled Substances Act, which was modeled after the federal act. (Earlier state laws were modeled on the 1934 Uniform Narcotic Drug Act, which had also been promulgated by the National Conference.) Every state has enacted the Uniform Controlled Substances Act.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy; Controls: Scheduled Drugs; Legal Regulation of Drugs and Alcohol*)

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CONTROLS: SCHEDULED DRUGS/DRUG SCHEDULES, U.S. The Comprehensive Drug Abuse Prevention and Control Act of 1970, commonly known as the CONTROLLED SUBSTANCE ACT (CSA) establishes the procedures that must be followed by drug manufacturers, researchers, physicians, pharmacists, and others involved in the legal manufacturing of, distributing of, prescribing of, and dispensing of controlled drugs. These procedures provide for accountability for a drug from its initial production through distribution to the patient and are intended to reduce widespread diversion of controlled drugs from legitimate medical or scientific use.

CRITERIA FOR CONTROLLING AND SCHEDULING DRUGS

Several factors are considered before a drug is controlled under this act. These factors include the potential for abuse (i.e., history, magnitude, dura-

tion, and significance), risk to public health, and potential of physical or psychological dependence. Drugs controlled under this act are divided into five Schedules (I–V) according to their potential for abuse, ability to produce dependence, and medical utility. Drugs in Schedule I have a high potential for abuse and/or dependence with no accepted medical use or they lack demonstrated clinical safety. Those in Schedules II–V may have a high potential for abuse or ability to produce dependence but also have an accepted medical use. (However, some substances which have no accepted medical use but which are precursors to clinically useful substances may also be found in Schedules II–V. For example, thebaine, found naturally in OPIUM, has no medical use but it is a substance used in the manufacture of CODEINE and a series of potent OPIOID compounds as well as opioid ANTAGONISTS.) The potential for abuse and the ability to produce dependence is considered to be the greatest for Schedule I and II drugs and progressively less for Schedule III, IV, and V (see Table 1).

The amount of controlled drug in a product can also determine the schedule in which it is placed. For example, AMPHETAMINE, METHAMPHETAMINE, and codeine, as pure substances, are placed in Schedule II; however, these same drugs in limited quantities and in combination with a noncontrolled drug are placed in Schedules III and V. Drugs in Schedule V generally contain limited quantities of certain narcotic drugs used for cough and antidiarrheal purposes and can only be distributed or dispensed for medical purposes.

LISTS OF SCHEDULED DRUGS

Drugs controlled under the CSA are listed by schedule and drug class in Table 2 (Schedules I and II) and Table 3 (Schedules III, IV, and V). A listing of controlled chemical derivatives, immediate precursors (chemical which precedes the active drug), and chemicals essential for making a controlled drug, along with drugs exempt from control can be found in the most current edition of the *Controlled Substances Handbook*. Brand names for drugs in Schedules II–V are not included in the Tables but can also be found in the latest edition of the *Controlled Substances Handbook*.

PRESCRIBING AND DISPENSING OF CONTROLLED DRUGS

Medical practitioners have to follow specific rules for each schedule when prescribing or dispensing controlled drugs. Drugs in Schedule I can only be obtained, prescribed, and dispensed to an individual after special approval is obtained from the Food and Drug Administration (FDA). Drugs in Schedule II cannot be refilled or dispensed without a written prescription from a practitioner, except in an emergency. When they are dispensed in an emergency, a written prescription must be obtained within 72 hours. Drugs in Schedule III and in Schedule IV may not be dispensed without a written or an oral prescription. Prescriptions for these drugs may not be filled or refilled more than six months after their issue date or refilled more than five times unless authorized by a licensed practitioner. Drugs in Schedule V can be refilled, with a practitioner's authorization, without the limitation on number of refills or time. Certain Schedule V drugs may be purchased directly from a pharmacist, in limited quantities, without a prescription. The purchaser must be at least 18 years of age and furnish appropriate identification and the transactions must be recorded by the dispensing pharmacist.

When drugs in Schedule II, III, and IV are dispensed, a warning label stating "Caution: Federal law prohibits the transfer of this drug to any person other than the patient for whom it was prescribed," must be affixed to the dispensing container. The warning label regarding transfer does not apply to Schedule V drugs.

REGISTRATION, ORDERING, QUOTAS, AND RECORDS OF CONTROLLED DRUGS

Each individual or institution engaged in manufacturing, distributing, or dispensing any controlled drug must be authorized by and register annually with federal and state drug-enforcement agencies, unless specifically exempted. A unique registration number is assigned to each individual or institution registered under the act. A separate registration is required for practitioners who dispense narcotic drugs to individuals for the purpose of addiction treatment (such as METHADONE and

LAAM [L-ALPHA-ACETYLMETHADOL] for opioid detoxification or maintenance).

All orders for Schedule I and II drugs must be made using a special narcotic order form. Proof of registration is required when ordering Schedule III-V drugs.

Annual production quotas are established for drugs in Schedule I and Schedule II. Everyone registered to handle controlled drugs must maintain records, conduct inventories, and file periodic reports specific to their business or professional activity.

SPECIAL ISSUES

The development of designer drugs has raised many concerns about policing drugs of abuse. Underground chemists who develop designer drugs seek to achieve two results: the creation of marketable drugs that mimic the effects of restricted drugs of abuse; and the creation of drugs that are not specifically listed as controlled substances by the Drug Enforcement Administration. The most popular designer drug of the late 1990s was MDMA (methylenedioxymeth-amphetamine), popularly known as Ecstasy. Despite efforts to evade federal drug laws, the designers of these drugs eventually see them added to the CSA. For example, MDMA was placed on Schedule I on an emergency basis in 1985 because of its neurotoxic effects and abuse potential.

State and local laws either parallel the federal regulations as described by the CSA, or impose additional restrictions. Individuals registered to handle controlled drugs must abide by the law (state or federal) that is most stringent in governing their business or professional activity. Examples where state law may be more stringent than federal law include the requirement for TRIPLICATE PRESCRIPTION forms or the placing of a drug in a higher schedule.

(SEE ALSO: *Addiction: Concepts and Definitions; Legal Regulation of Drugs and Alcohol*)

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COPING AND DRUG USE Coping is the capacity to surmount negative emotional states, including ANXIETY, DEPRESSION, anger, loneliness, and alienation. These aversive states are induced by internal psychological conflict or by external STRESS. Effectiveness in appraising and overcoming emotional distress that results from predisposing or triggering stressors determines, to a large extent, psychological well-being. In contrast, ineffectiveness in coping, as well as a subjective perception of ineffectiveness, exacerbates emotional distress, which comprises for some people an important factor in promoting ALCOHOL, TOBACCO, and other drug (ATOD) consumption.

The association between ATOD use and coping is complex. In some individuals there is a direct connection. In effect, PSYCHOACTIVE DRUGS are consumed to reduce tension and associated negative emotions. The consumption of drugs is motivated by their palliative effects. In most individuals, however, the connection between drug consumption and coping is more complicated. Numerous factors such as psychiatric illness, low self-esteem, deviant social values, maladaptive learned behaviors, inadequate social support, poor social skills, and personality disposition moderate and mediate the relationship between ATOD use and coping. No specific association has been established between coping style and VULNERABILITY to drug use or abuse. Thus, whereas it is generally recognized that a substantial proportion of the ATOD-using population is deficient in coping capacity, it is important

to understand that many factors influence this association.

Coping and substance use and abuse become so intertwined over time that cause-effect relationships cannot always be discerned. Deficient coping capacity initially may, directly or indirectly, lead to ATOD consumption. Neurobehavioral, psychopathological, and social adjustment disturbances that occur along with chronic ATOD consumption may also diminish coping ability.

Substantial variation among individuals occurs with respect to both coping capacity and drug-use behavior across the life span. Drug consumption among youth is most frequently related to negative feelings such as depression and anxiety, social deviancy, and interpersonal problems—whereas substance use among the ELDERLY is more commonly associated with life crises, psychiatric disorder, bereavement, sleep disturbances, and unremitting pain.

Drug-abusing youth and adults, as a group, exhibit less ability to cope than the general population (Peele, 1985). It is essential to emphasize, however, that ATOD use and abuse may also be motivated by reasons other than the need to cope. In this context, ATOD consumption often stems from the desire for a euphoric effect or some other desirable state, a desire that may reflect accurate as well as inaccurate beliefs about the pharmacological effects of the chosen drug. For example, ATOD consumption may be motivated by perceived APHRODISIAC effects, energy or alertness enhancement, or social facilitation.

Among those whose ATOD consumption is motivated by deficient coping skills, it appears that augmenting competency improves the likelihood of successful treatment. In other words, treatments designed to enhance their coping skills are superior to treatments that emphasize their exploration of feelings (Getter et al., 1992). Furthermore, active coping strategies present 2 years after treatment are associated with a superior outcome at 10-year posttreatment follow-up (Finney & Moos, 1992).

The role of coping in ATOD use needs to be evaluated on a case-by-case basis. Assessment can be conducted using the Ways of Coping scale (Lazarus & Folkman, 1984) or the more comprehensive Constructive Thinking Inventory (Katz & Epstein, 1989). Severity of ATOD-use disorder can be efficiently quantified by employing the Drug Use Screening Inventory (Tarter, 1990). This brief self-report evaluates the severity of the disorder in ten key domains: (1) substance use, (2) psychiatric disorder, (3) behavior patterns, (4) health status, (5) family system, (6) work adjustment, (7) social competence, (8) peer relationships, (9) school adjustment, and (10) leisure/recreation. A treatment protocol to enhance coping has also been developed for alcoholics (Kadden et al., 1992); this practical approach to intervention is also applicable for treating individuals with other types of drug abuse.

(SEE ALSO: *Relapse; Treatment Types: Cognitive Therapy of Addictions*)

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RALPH E. TARTER

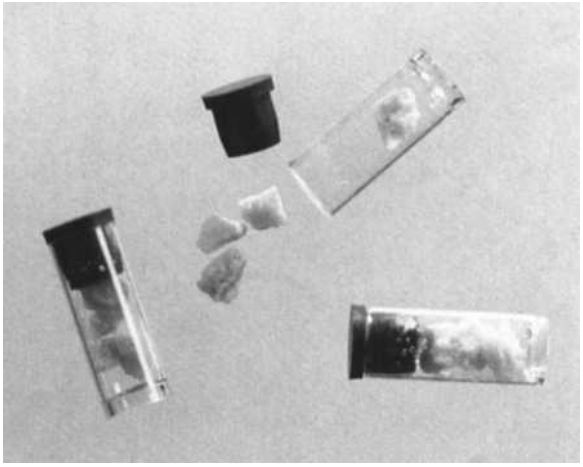
CRACK Crack (sometimes called crack-cocaine) is an illicit drug, the smokable form of COCAINE, made by adding the bases ammonia or baking soda and water to cocaine hydrochloride. The white powder illicitly purchased as cocaine is in the hydrochloride form; it cannot be smoked, because it is destroyed at the temperatures required for smoking. Therefore, in order to be used by the smoked route, cocaine must be converted to the

base state. A mixture is made and heated to remove the hydrochloride, resulting in a pellet-sized cake-like solid substance that can be smoked. This form of cocaine is inexpensive, available for purchase "on the street," and is called "crack," because of the cracks formed in the solid as it dries.

Although crack can be smoked in tobacco cigarettes or marijuana cigarettes, it is generally smoked in a special crack pipe. In its simplest form, this is a glass tube with a hole at the top of one end and a hole at the other end through which the smoke is inhaled. The crack pellet is placed on fine wire mesh screens that cover the hole distal to the smoker and a flame is applied directly to the pellet. Soda bottles, small liquor bottles, etc. are all used to manufacture crack pipes. They have in common the use of fine mesh screens so that the crack is not lost as it melts. Temperatures of approximately 200°F (93°C) are most efficient in providing the largest amount of cocaine to the user. Higher temperatures destroy more of the cocaine.

Smoking cocaine began with the use of FREEBASE cocaine, prepared by its users from the cocaine hydrochloride illicitly purchased by them. Soon after this form of cocaine had achieved its popularity, single doses of cocaine already prepared for smoking (i.e., crack), became available through the illicit drug market. Unlike the process for forming freebase cocaine, the crack manufacturing process does not rid the cocaine of its adulterants. Smoking cocaine rapidly became a popular route of administration once crack became readily available, since it was so convenient to use. Blood levels peak rapidly when cocaine is smoked, because of efficient respiratory absorption, and the smoked route of cocaine administration yields effects (peak, duration of effect, half-life) comparable to the intravenous route of administration. This means that the smoker of cocaine can achieve rapid onset of effect, including a cocaine "rush" and substantial cocaine blood levels, and can do this repeatedly using a more socially acceptable route of administration—one that requires none of the PARAPHERNALIA associated with hardcore illicit drug use (e.g., syringes, needles, etc.).

The more rapid the onset of the drug effect, the more likely it is that the drug will be abused. Thus, although the effects of smoking crack are no different than the effects of cocaine by any other route, the ease with which the drug can be taken, combined with its toxicity make this an extremely dangerous substance.



Smoking crack, the rock form of cocaine, produces effects comparable to intravenous injection; the effects, felt almost immediately, are very intense, and quickly subside. (Drug Enforcement Administration)

From a financial perspective, crack is more desirable for both the buyer and the seller. A gram of cocaine hydrochloride costs approximately 50 to 60 dollars. This gram can be turned into 10 to 25 crack pellets, each selling for 2 to 20 dollars. Thus, a gram of cocaine can generate a substantial profit for the seller, and, as well, is available in single-dose units to anyone with only a few dollars to spend.

(SEE ALSO: *Coca Paste; Freebasing; Pharmacokinetics; Street Value*)

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MARIAN W. FISCHMAN

CRAVING The term *craving* is generally delined as a state of desire, longing, or urge for a drug

that is responsible for ongoing drug-use behavior in drug-dependent individuals. Craving is also viewed by many drug-abuse researchers and clinicians as the main cause of relapse among drug users attempting to remain abstinent. During periods of abstinence, drug-dependent individuals often complain of intense craving for their drug. Several systems for diagnosing drug abuse include persistent desire or craving for a drug as a major symptom of drug-dependence disorders.

The belief that an addict's inability to control drug use is caused by craving and irresistible desire was a prominent feature of descriptions of addictive disorders provided by many nineteenth-century writers. Craving continued to be important in many models of addiction developed in the twentieth century. The use of craving as a key mechanism in theories of addiction peaked in the 1950s, supported largely by E. M. Jellinek's writings on the causes of alcoholism.

Jellineck contended that sober alcoholics who consumed a small amount of alcohol would experience overwhelming craving that would compel them to continue drinking. The proposal that craving and loss of control over drinking were equivalent concepts was adopted by many clinicians and addiction researchers. Equally popular was the position, also supported by Jellinek, that craving was a direct sign of drug withdrawal. WITHDRAWAL-based craving was often described as physical craving, distinguishing it from craving that led to relapse during long periods of abstinence after withdrawal had subsided. Craving that occurred after an addict no longer was experiencing withdrawal was typically viewed as the result of psychological factors. The craving concept was sufficiently controversial that a committee of alcoholism experts brought together by the World Health Organization in 1954 (WHO Expert Committees on Mental Health and on Alcohol, 1955) recommended that the term craving not be used to describe various aspects of drinking behavior seen in alcoholics.

The use of craving as a key process in theories of addiction decreased during the 1960s and early 1970s as a result of several factors. During this period, many studies showed that alcoholics did not necessarily engage in loss of control drinking when they drank small doses of alcohol. The failure to confirm Jellinek's conceptualization of alcoholic drinking cast doubt on the idea that craving was synonymous with loss of control over drug intake. Furthermore, withdrawal models of craving could not account for the common observation that many addicts experienced craving and relapsed long after their withdrawal had disappeared. Finally, addiction research was increasingly dominated by behavioral approaches that focused on the influence of environmental variables in the control of drug taking and avoided the use of subjective concepts, such as craving, to explain addictive behavior.

Even though many researchers questioned the value of using craving to explain addictive behavior, it persisted as an important clinical issue, as many addicts complained that craving was a major barrier to their attempts to stop using drugs. Craving continued to be cited as a major symptom of drug dependence in formal diagnostic systems of behavioral disorders, and the notion that craving was responsible for compulsive drug use remained at the core of several popular conceptualizations of drug addiction. Scientific interest on the role of craving in addictive disorders reemerged in the middle 1970s as a result of two developments. First, behavioral theories of addiction were increasingly influenced by social-cognitive models of behavior that were more sympathetic to the possibility that hypothetical entities such as craving might be useful in explaining addictive processes. Second, animal research on the contribution of learning processes to drug tolerance and drug withdrawal provided support for the hypothesis that learned withdrawal effects might produce craving and relapse in abstinent addicts.

THEORIES OF CRAVING

Although there is considerable disagreement across current theories regarding the processes that supposedly control craving, nearly all models describe craving as, fundamentally, a subjective state and agree on the impact of craving on drug use. With few exceptions, modern theories of craving assume that craving is a necessary, but probably not sufficient, condition for drug taking among addicts. These theories suppose that addicts are driven to use drugs because of their craving, and craving is generally described as the principal cause of relapse in addicts trying to remain abstinent. Moreover, all the comprehensive models of craving invoke some sort of learning or cognitive process in

their descriptions of the mechanisms controlling craving, and these models make little distinction between physical and psychological forms of drug craving. It is important to note that, at the present time, research on craving is not sufficiently advanced to fully evaluate the validity of any of the major models of craving.

Many modern theories associate craving with drug withdrawal and suggest that craving may be merely a part of drug withdrawal. For example, the diagnostic system published by the American Psychiatric Association in 1987 listed craving as one of the symptoms of withdrawal for nicotine and opiates. Other approaches assume that cravings are distinct from withdrawal, but represent an addict's anticipation of, and desire for, relief from withdrawal. To explain the presence of craving following long periods of abstinence, it has been posited that learning processes are responsible for the maintenance of withdrawal effects. For example, Wikler's conditioning model of drug withdrawal (see WIKLER'S PHARMACOLOGIC THEORY) hypothesizes that situations reliably paired with episodes of drug withdrawal become conditioned stimuli that can produce conditioned withdrawal responses. An addict who has been abstinent for an extended period may reexperience withdrawal if faced with these conditioned stimuli. This learned-withdrawal reaction will trigger drug craving, that, in turn, may lead to relapse. A similar theory is based on the suggestion that drug-tolerance processes can become conditioned to environmental stimuli. Some have hypothesized that conditioned drug-tolerance effects will produce withdrawal-like reactions that, as in Wikler's theory, should promote craving and relapse to drug use (Poulos, Hinson, & Siegel, 1981).

Another perspective on craving is that it is strongly associated with the positively reinforcing, or stimulating, effects of drugs. For example, Marlatt (1985) has suggested that craving is a subjective state produced by the expectation that use of a drug will produce euphoria, excitation, or stimulation. Similarly, Wise (1988) proposed that craving represents memories for the pleasurable or positively reinforcing effects of drugs. There are also multiprocess models, in which expectancies of positive reinforcement and anticipation of withdrawal relief, as well as other factors, including mood states and access to drugs, generate craving (Baker, Morse, & Sherman, 1987; Gawin, 1990).

In contrast to models that contend that craving is responsihle for all addictive drug use, a recent cognitive theory suggests that drug use may operate independently of craving (Tiffany, 1990). According to this theory, as a result of a long history of repeated practice, most of an addict's drug-use behavior becomes automatic. That is, drug use may be easily triggered by certain cues, difficult to stop once triggered, and carried out effortlessly with little awareness. Addicts attempting to withdraw from drug use will experience craving as they try to stop these automatized actions from going through to completion.

MEASURES OF CRAVING

Craving is generally measured through three types of behaviors—self-reports of craving, drug-use behavior, and physiological responding. In the most frequently used measure, self-report, addicts are simply asked to rate or describe their level of craving for a drug. Recently, questionnaires have been developed that ask addicts to rate a variety of questions related to craving. These questionnaires produce results that are considerably more reliable than a single rating of craving and tend to show that an addict's description of craving may have multiple dimensions. Measures of drug-use behavior have also been used to assess drug craving. This is entirely consistent with the common assumption that craving is responsible for drug use in addicts. Finally, as several theories posit that craving should be represented by particular patterns of physiological changes, physiological measures, primarily those controlled by the autonomic nervous system, have been included in several studies as an index of craving. These measures have included changes in heart rate, sweat gland activity, and salivation. In general, withdrawal-based theories predict that the physiology of craving should look like the physiology of drug withdrawal. In contrast, models that emphasize positive reinforcement in the production of craving would associate drug desire with physiology characteristic of the excitatory effects of drugs.

RESEARCH ON CRAVING

Two kinds of studies have been used to investigate drug craving. The first, naturalistic studies, examine changes in addicts' descriptions of craving as they are attempting to stop using drugs. These studies generally have shown that cravings are especially strong in the first several weeks of abstinence, but decline over time as addicts stay off drugs. They also reveal that craving rarely remains at a constant level throughout the day, but grows stronger or weaker depending on the situations the addict encounters. These situations tend to be strongly associated with previous use of drugs, such as meeting drug-using friends or going to locations where the addict used drugs in the past.

Laboratory studies attempt to manipulate craving by presenting addicts with stimuli or cues that have been associated with their previous drug use. For example, a heroin addict may watch a videotape of someone injecting heroin or smokers may be asked to imagine a situation in which they would want to smoke. These cue-reactivity studies allow the measurement of self-reports of craving, drug-use behavior, and physiological reactions under controlled conditions. Results from these studies indicate that abstinence from drugs, drug-related stimuli, and negative moods can influence craving measures.

Many of the results of naturalistic and laboratory studies have presented a challenge to the dominant assumption that craving is directly responsible for drug use in addicts (Kassel & Shiffman, 1902; Tiffany, 1990). For example, across many cue-reactivity studies, there is not a very strong correlation between addict's reported levels of craving and their level of drug consumption in the laboratory. Correlations between self-reported craving and physiological reactions also tend to be weak. Other studies reveal that, although addicts frequently complain that cravings are a major difficulty they face as they try to stay off their drugs, few addicts who relapse say that they experienced craving just before their relapse episode. These findings show that the exact function of craving in drug dependence remains a controversial issue.

Despite these negative indications, millions of dollars are spent each year to develop pharmacological agents that might be capable of blocking, preventing, or reducing craving for various drugs.

(SEE ALSO: *Addiction: Concepts and Definitions; Causes Drug Abuse: Learning; Research: Conditioned Drug Effects; Research, Animal Model: Conditioned Drug Effects*)

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STEPHEN T. TIFFANY

CREATIVITY AND DRUGS Accounts of alcohol and drug use to stimulate creativity are apocryphal and anecdotal. For example, Samuel

Taylor Coleridge reportedly composed much of his unfinished poem *Kubla Khan* while in an opium dream. In ancient Greece, however, the Pythian priestesses of the oracle at Delphi inhaled medicinal fumes to facilitate revelatory trances—as did the priests and peoples of most ancient societies. The institutionalized twentieth-century Native American Church continues to use the PEYOTE of their ancestors to promote profound religious experiences.

Psychedelic drugs, such as LYSERGIC ACID DIETHYLAMIDE (LSD), MESCALINE, PSILOCYBIN, and methylene dioxyamphetamine (MDA) have been used—both legally and illicitly—to increase aesthetic appreciation, improve artistic techniques, and enhance creativity. MARIJUANA has been used to heighten the sense of meaning, foster creativity, and heighten perceptions (also both legally and illicitly); and ALCOHOL has been employed by countless people worldwide to relieve inhibitions, increase spontaneity, and stimulate innovation and originality.

In the industrial West, the common belief in, and positive association between, alcohol or drug use and creativity is strengthened by the popular stereotypes of artists, writers, actors, and others in the creative and performing arts as heavy users or abusers of such substances. Despite these anecdotal claims, little scientific evidence supports the notion that alcohol and drug use actually increase creativity.

Part of the reason that creativity is attributed to drug use involves the actions of many psychoactive substances in producing altered states of consciousness. These altered states are characterized by some or all of the following features: (1) *alterations in thinking*, in which distinctions between cause and effect become blurred and in which logical incongruities may coexist; (2) *disturbances in time sense*, whereby the sense of time and chronology may become greatly altered; (3) a sense of *loss of control*, during which the person becomes less inhibited and self-possessed; (4) a *change in emotional expression*; (5) *body image change*, with a dissolution of boundaries between one's self and the world, resulting in transcendental or mystical experiences of "oneness" or "oceanic feelings"; (6) *perceptual distortions*, including illusions, pseudohallucinations, heightened acuity, and increased visual imagery; (7) *hypersuggestibility*, representing a decrease in the use of critical faculties; (8) a heightened *sense of meaning and significance*; (9) *sense of the ineffable*, in which the experience cannot be expressed in words; and (10) feelings of *rebirth and rejuvenation*. When people experience such features as these, it is understandable that they attribute creativity to certain drug experiences.

Samuel Taylor Coleridge (1772–1834), depicted here in a 1795 painting by Peter Van Dyke, reportedly composed much of Kubla Khan *while in an opium dream.* (© Bettmann/CORBIS)

The immediate problem, however, in evaluating whether this is really so depends on the definition of creativity. At the outset, three dimensions of creativity need to be distinguished: those pertaining to the creative person, those pertaining to the creative process, and those pertaining to the creative product. If creativity pertains to an attribute of the person (e.g., original thinking), then any unusual or extraordinary experiences should qualify as "creative," even if nothing of social value emerges. If creativity pertains to a process (e.g., discovery, insight), then the testing and validation of the insights must take place as well. If creativity pertains to a product, it not only should possess some measure of social utility but should embody such qualities as novelty, surprise, uniqueness, originality,

beauty, simplicity, value, and/or coherence. For both the creative process and the creative product, there is no substantive evidence to indicate that alcohol or drugs have benefit, despite the ongoing belief of many that they do. The experience of the *sense of meaning or significance* produced by drugs may have no bearing on whether that experience has *true* meaning or significance. The American philosopher and psychologist William James's claim that alcohol makes things seem more "utterly utter" is especially apt. This also happens with PSYCHEDELIC DRUGS, which have the capacity to induce a sense of profundity and epiphany (intuitive grasp of reality), but usually without any substantive or lasting benefit or practical value.

What, then, is the actual state of knowledge about the relationship between substance use and creative achievement? What few studies exist, in fact, indicate mostly detrimental effects of drugs on creativity, especially when these substances are taken in large amounts and over an extended period of time. The results of studies on the actions of alcohol typify this. As early as 1962, for example, Nash demonstrated that small doses (about equal to two martinis) of alcohol, in normal volunteers, tended to facilitate mental associations, while large doses (about equal to four martinis) had adverse effects. With the large doses, they had more trouble in discriminating and assimilating details and performing complex tasks. In another study, Hajcak (1975) found that male undergraduates permitted alcohol on an ad-lib basis (without limits or restraints) showed greater initial productivity than when not allowed to drink but showed decreased appropriateness and decreased creative problem solving when intoxicated.

In an anecdotal study with seventeen artists who drank, Roe (1946) found that all but one regarded the short-term effects of alcohol as deleterious to their work, but they sometimes used it to overcome various technical difficulties. The general sentiment was that alcohol provided the freedom for painting but impaired the discipline. In a more extensive study of thirty-four eminent writers, Ludwig (1990) found that more than 75 percent of artists or performers who drank heavily experienced negative effects from alcohol—either directly or indirectly, on creative activity, particularly when they did not refrain from drinking when they were working. More positive effects of alcohol were found in a small number of cases, among those who used it in moderate amounts early in their careers to remove certain roadblocks, to lessen depression or mania, or to modulate the effects of other drugs.

With many anecdotal accounts to the contrary, the weight of scientific and clinical evidence suggests that long-term alcohol and drug use exert mostly negative effects on creativity. That drugs and alcohol are used so widely within the creative arts professions seems to have less to do with creativity than with social expectations and other extraneous factors. In fact, people use pharmacological substances for many reasons other than the stimulation of their imaginations. These reasons include relaxation, the facilitation of sleep, self-medication, social rituals, pleasure, or simply habituation or addiction.

Because writers, artists, actors, or musicians may write about, portray, or act out certain aspects of their pharmacological experiences does not logically or necessarily mean that these experiences are essential for the creative process. Creative people often exploit all aspects of their experiences—whether pathological or healthy and whether drug-induced or not—in a creative way; they try to translate personal visions and insights within their own fields of expression into socially acceptable, useful, or scientifically testable truths. Without some measure of social utility, unique drug-induced experiences represent little more than idiosyncratic to quasi-psychotic productions, having value and meaning only, perhaps, to the substance user.

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Arnold M. Ludwig

CRIME AND ALCOHOL The relationship between Alcohol and involvement in crime is not a simple one. Drinking is a very common activity, and most drinking is not followed by criminal behavior. Understanding the alcohol-crime relationship requires an identification of those drinking effects and circumstances that are related to crime. Alcohol's relationship to crime also varies by the type of crime. The major crime-type distinction is between *violent* personal crime (such as homicide, forcible rape, and assault) and *property* crime (such as burglary and larceny). Alcohol's effects differ with respect to violent crime and property crime. Individual characteristics are also implicated in the alcohol-crime relationship. Age and gender, for example, affect whether drinking leads to criminal behavior. Young adult males are more likely than older adult males and females of all ages to engage in alcohol-related offenses.

According to the available evidence, drinking is more likely to be implicated in violent than in property crime. Moreover, violent offenses are often thought of as expressive or instrumental. Expressive violent offenses are typically those that result from interpersonal conflict that escalates from verbal abuse to physical Aggression. Such violence often involves a drinking offender or drinking by both (or multiple) parties in cases of violent conflict. Instrumental offenses have rational goals, typified by stealing to realize the value of the stolen money or goods. Alcohol is not thought to be an important causal factor in acquisitive crimes such as theft.

Research has shown that alcohol is an important factor in the occurrence of expressive interpersonal violence, that alcohol use increases the risk of being a crime victim, that the alcohol-crime relationship is complex (involving multiple factors in addition to alcohol), and that alcohol is often blamed without justification for criminal offenses.

HOW OFTEN DOES DRINKING PRECEDE THE COMMISSION OF CRIMES?

The Bureau of Justice Statistics (BJS), an agency of the U.S. Department of Justice, reviewed the role alcohol played in crime by looking at convicted offender data from 1996 (Greenfield, 1998). On an average day in 1996, an estimated 5.3 million convicted offenders were under the supervision of criminal justice authorities. Nearly 40% of these offenders, about two million, had been using alcohol at the time of the offense for which they were convicted. Whether the offender was on probation or was incarcerated in a local jail or a state prison, offenders were about equally likely to have been drinking at the time of the crime. What they consumed was similar, with beer being the most commonly used alcoholic beverage: 30 percent of probationers, 32 percent of jail inmates, and 23 percent of state prisoners said that they had been drinking beer or beer in combination with liquor prior to the commission of the current offense. Consumption of wine alone was comparatively rare among the surveyed offender populations.

Surveys of crime victims also indicate that offenders often had been drinking. The National Crime Victimization Survey (NCVS) is one of two statistical series maintained by the Department of Justice to learn about the extent to which crime is occurring. The NCVS, which gathers data on criminal victimization from a national sample of household respondents, provides annual estimates of crimes experienced by the public without regard to whether a law enforcement agency was called about the crime. Initiated in 1972, the NCVS was designed to complement what is known about crimes reported to local law enforcement agencies under the FBI's annual compilation known as the Uniform Crime Reporting Program (UCR). 1998 estimates from the NCVS indicate that victims of about three million violent crimes each year, or about a quarter of all violent crimes, perceived the offenders to have been drinking.

Most studies of alcohol and crime focus on offenses known to the police or on offenders serving sentences for crimes that resulted in conviction. A

notable exception is a community study in Thunder Bay, located in the province of Ontario, Canada. Pernanen (1976, 1981, 1991) collected information from a representative sample of 1,100 community residents. Among those who had been victimized, the assailant had been drinking in 51 percent of the occasions when violence occurred; two-thirds of the time (68%), the assailant was judged to have been "drunk." Pernanen also noted that the findings for the Thunder Bay study are consistent with many other North American studies using police records. It is usually found that half of all violent offenders had been drinking at the time of the offense.

The most common pattern found in studies of violent crimes is that 60 percent or more of the events involve drinking by the offender, both the offender and the victim, or the victim only. The results of Wolfgang's classic study (1958) of criminal homicides in Philadelphia are typical (see table 2). The most common pattern when alcohol was present was for both victim and offender to have been drinking.

If the foregoing findings indicated the extent to which drinking was *causally* implicated in violent crime, it would be remarkable. It could then be argued that alcohol accounts for a majority of violent offenses. But neither the presence of alcohol in a crime nor the intoxication of an offender is necessarily an indication that alcohol influenced the occurrence of the crime. Because drinking is such a common activity, it is likely that alcohol is sometimes simply present but not causally relevant. Drinking is also sometimes offered by offenders as an excuse for the crime, as a way of avoiding being held accountable.

ALCOHOL USE AND CRIME VICTIMIZATION

Alcohol use raises the likelihood that the drinker will be a victim of violent crime. Substantial percentages of homicide, assault, and robbery victims were drinking just before their victimization. Medical examiners have done a significant number of homicide studies by running toxicological tests of the body fluids of homicide victims. Separate reviews by Greenberg (1981) and by Murdoch, Pihl, and Ross (1990) found that the percentage of homicide victims who had been drinking ranges widely, but is usually about 50 percent. Goodman et al. (1986) tested the alcohol levels of several thousand homicide victims; they found that 46 percent of the victims had consumed alcohol in the period before being killed, and three of ten victims had alcohol levels beyond the legal intoxication level.

Roizen (1993) examined studies of alcohol use by robbery and rape victims. The percentage who had been drinking before their victimization ranged widely—from 12 to 16 percent for robbery victims and from 6 to 36 percent for rape victims. Abbey (1991) and Muehlenhard and Linton (1987) also found in their studies of date rape that both offenders and victims had commonly been drinking. Abbey suggested that drinking by the offender or by the victim contributes to rape by the impaired communication and misperception that results from alcohol's effects on cognitive ability (among other contributing factors). Males who have been drinking, for example, may mistakenly attribute sexual intent to their date.

Alcohol may increase the risk that the drinker will be a crime victim because of effects that alcohol has on judgment and demeanor. Someone who has been drinking may take risks that might not be taken when sober, such as walking in a dangerous area of a city at night. Alcohol also causes some individuals to be loud and verbally aggressive. Such demeanor can be offensive and might sometimes precipitate physical attack.

DOES DRINKING GENERATE FAMILY VIOLENCE?

Unfortunately, violence is common in American households, and alcohol is a contributing factor, according to research done by Kantor and Straus (1989) and by Straus, Gelles, and Steinmetz (1980), among others. Hotaling and Sugarman (1986) found that alcohol appears to be most relevant to the occurrence of husband-against-wife violence. Hamilton and Collins (1981) reviewed about 25 studies that examined the role of alcohol in spouse and CHILD ABUSE. They found alcohol to be most relevant to wife beating, where it was present in one-quarter to a half of all such events. (Alcohol was present in less than one in five incidents of child abuse.) The most common pattern was for only the husband to be drinking or for both parties to have consumed alcohol. It was uncommon for only the wife to have been drinking. Stud-

ies also indicate that husbands or partners with alcohol problems were more likely to be violent against their wives/partners.

A 1998 BJS study on the relationship between crime and alcohol found that two-thirds of victims who suffered violence by an intimate (a current or former spouse, boyfriend, or girlfriend) reported that alcohol had been a factor. Among spouse victims, three out of four incidents were reported to have involved an offender who had been drinking. By contrast, an estimated 31 percent of stranger victimizations where the victim could determine the absence or presence of alcohol were perceived to be alcohol-related.

Research by Jones and Schecter (1992) and by Barnett and Fagan (1993) on family violence suggests that violence against women may lead to their own use of alcohol and drugs as a coping mechanism. Drinking and/or drug use may be a response to the physical and emotional pain and fear that result from living in a violent relationship. Miller, Downs, and Testa (1993) found that women in alcohol-treatment programs had higher rates of father-to-daughter violence than did the women in the comparison group. These findings underline the importance of interpreting the meaning of alcohol's association with family (and other forms of) violence carefully. As previously noted, alcohol is often present but irrelevant to the occurrence of violence. Some recent literature on family violence indicates that alcohol use may sometimes be a *response* to violent victimization.

HOW OR WHY DOES ALCOHOL CONTRIBUTE TO CRIME?

There are a number of possible explanations offered for alcohol's role in crime:

The need for money to support drinking may cause some individuals to commit crimes to generate cash to support their habit.

The pharmacological effects of alcohol can compromise drinkers' cognitive ability and judgment and raise the likelihood of physical aggression.

Expectations that alcohol makes drinkers aggressive may increase the chance of violence.

Standards of conduct and accountability for behavior may differ for sober and drunken activities (these differences can result in an increase in the likelihood of criminal behavior after drinking).

These possible explanations are not mutually exclusive. All may sometimes accurately describe how drinking causes crime. Two or more of the explanations may even apply to the same incident.

Committing "income crimes"—to obtain money for drinking—is not thought to be an important explanation. Although the cost of maintaining an addiction to relatively more expensive drugs (e.g., HEROIN and COCAINE) is high, the price tag for supporting heavy drinking is usually modest. In most of the United States, for example, one could support a habit of daily heavy drinking for 10 dollars or less. The majority of individuals could maintain such a habit without resorting to crime, although many heavy drinkers spend more than this minimal amount on alcohol. There is virtually no information in the research literature about the likelihood or frequency of involvement in income crime to support drinking, but alcohol is not thought to be a major factor in income crimes. This does not mean it never happens, only that it is uncommon.

If alcohol is not an important factor in the occurrence of income-generating crime, why do so many property offenders (approximately 30 percent of inmates in 1996) report they were under the influence of alcohol at the time they committed such offenses? At least two explanations are possible for the high correlation between drinking and property crime. The first suggests that the correlation is simply coincidental, not causal. A second reason (put forward by both Collins, 1988, and Cordilia, 1985), is that a property offender who has been drinking is more likely to be caught than is a sober one. This reason makes sense based on the known impairment effects of alcohol. A drinking offender may not be as competent or careful as a sober one, so drinking offenders may be overrepresented among offenders who are caught and thus known to criminal-justice officials.

Alcohol impairs cognitive ability, including one's own capacity to communicate clearly and the capacity to understand the verbal and behavioral cues of others. In addition, a person whose abilities have been impaired by alcohol is less able to make decisions and carry out appropriate and effective actions. Pernanen, in his early work (1976), discussed how alcohol-impaired cognitive ability can lead to violence. When one or both parties who are interacting have been drinking, there is an increased potential for misunderstanding that can

lead to conflict and that may in turn escalate to violence. One factor in such a scenario is what may be called a "reduced behavioral response repertoire." Alcohol impairs a drinker's capacity to conceive and utilize the wide range of verbal and other behavioral options that are available to sober individuals. Alcohol-induced cognitive impairment may also diminish the drinker's capacity to foresee the negative implications of violent actions. In summary, one way that alcohol increases the likelihood of violence is its negative effect on cognitive capacities, and these effects lead to an increased risk of violence.

It has been demonstrated in laboratory experiments that both actual alcohol use and the *belief* that alcohol has been consumed can raise levels of aggression. In laboratory experiments using competitive encounters between opponents in which the winner can apply an electrical shock to the loser, subjects who have been given alcohol behave more aggressively. Evidence gathered by Bushman and Cooper (1990) and by Hull and Bond (1986) also indicates that subjects who have been told they have received alcohol, but who actually have been given a placebo, are more aggressive in their administration of electrical shocks. These findings suggest that beliefs about alcohol's behavioral effects can themselves affect behavior.

Expectations that alcohol use leads to aggressive behavior probably have sociocultural roots. Anthropologists such as Heath (1976a, 1976b) and MacAndrew and Edgerton (1969), for example, noted that societies differ in the behavior that occurs after drinking. Some of these differences may be attributable to racial or ethnic differences in physiological reactions to alcohol, but it is also clear that there are normative variations in what behaviors are expected or acceptable after drinking. In fact, behavioral norms after drinking may vary within societies. MacAndrew and Edgerton noted that during certain "time-out" periods, usual standards of behavior are suspended. For example, festivals or Mardi Gras celebrations are often characterized by high levels of drinking and behavior that is considered deviant or criminal during normal times.

Alcohol appears to be implicated in violence in another indirect way. Drinking is sometimes used as an excuse for crime or as a way to avoid accountability after the fact. McCaghy (1968) has referred to this phenomenon as "deviance disavowal." The deviance disavowal potential of alcohol can account for a drinker's involvement in crime in two ways: individuals may drink or say that they have been drinking as an advance excuse for their conduct, or drinking may be offered as an excuse after the fact.

SUMMARY

Drinking alcohol and involvement in criminal behavior frequently occur together. Some of the time alcohol has a causal role in crime, but often it is merely present. Drinking is most likely to be relevant causally to expressive interpersonal violence—including family violence. Drinking can increase the risk of being victimized as well. Drinking may also sometimes help account for the commission of crimes to obtain money to support the habit, but alcohol is not a major factor in the occurrence of income crime. Drinking leads to criminal behavior in a number of ways, including the effects that alcohol use has on cognition and the rules that govern behavior and accountability for behavior. The alcohol-crime relationship is complex. It is clear that drinking is rarely the only cause of criminal behavior, and that when it does contribute, it is usually only one of a number of relevant factors.

(SEE ALSO: *Complications: Cognition; Crime and Drugs; Drunk Driving; Economic Costs of Alcohol Abuse and Alcohol Dependence; Expectancies; Family Violence and Substance Abuse; Social Costs of Alcohol and Drug Abuse*)

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REVISED BY FREDERICK K. GRITTNER

CRIME AND DRUGS Because of widespread public and political concern over drug-related crime, there has been an urgent need to understand the drugs-crime relationship. However, despite numerous studies on this topic, only re-

cently have significant empirical advances in our understanding emerged.

Authors of a comprehensive literature review published in 1980 concluded that the drugs-crime relationship was far more complex than originally believed (Gandossy et al.). While acknowledging significant contributions of previous research, the authors felt that methodological problems in the studies they reviewed had obscured an understanding of the linkage between drugs and crime. As these and other reviewers have observed, perhaps the most serious of these weaknesses was the use of official arrest records as indicators of criminal activity. Studies using confidential self-report methods in settings in which there is immunity from prosecution have consistently documented that less than 1 percent of offenses committed by drug abusers result in arrest.

With a subsequent emphasis on confidential self-report data, studies conducted since 1980 have permitted more realistic estimates of the extent of criminality among drug abusers. In addition, victims of violent crime are now being asked whether they perceived the offender to be under the influence of drugs or alcohol. The annual Bureau of Justice Statistics (BJS) National Crime Victimization Survey asks this question of crime victims. Though a subjective inquiry, the 1998 survey revealed that 30 percent of victims could not determine whether the offender was under the influence of a substance. Of those who could make a determination, about 31 percent reported that the offender was under the influence of drugs or alchohol.

In 1997, the National Institute of Justice (NIJ) established the Arrestee Drug Abuse Monitoring (ADAM) Program to measure drug use among arrestees by calculating the percentage of arrestees with positive urine tests for drug use. ADAM data are collected voluntarily and anonymously at the time of arrest in booking facilities in thirty-five U.S. cities. The ADAM program has given researchers a powerful tool for obtaining empirical evidence of patterns of drug abuse. ADAM is the only national research program studying drug use that employs both drug testing and interviews, giving analysts the means of assessing validity of self-report data. Therefore, ADAM data are less susceptible either to exaggeration or denial of drug use than many other surveys. Moreover, ADAM is the only national drug research program built upon data collection at the local level. This data has revealed that there is no single national drug problem, but rather different local drug problems that vary from city to city.

THE CRIMINALITY OF DRUG ABUSERS

In examining the criminality of drug abusers, it is important to note that the onset of illicit drug use typically does not result in the onset of criminal behavior. Rather, it is the *frequency*, not the *onset*, of drug use that increases criminal activity. Further, the positive relationship between drug-use frequency and crime frequency is not consistent across all types of drug use and all types of crime. Such a relationship has been observed with respect to only three types of drug abuse: heroin addiction, cocaine abuse, and multiple-drug use. In addition, such associations are more common for property crime than for violent crime.

Narcotic Drug Use. Much of our knowledge about the relationship between drugs and crime comes from detailed self-report information on the type, extent, and severity of criminal activity of NARCOTIC (mainly heroin) addicts. Large-scale, independently conducted studies have convincingly shown that increases in property crime and robbery, which has components of both property crime and violence, are associated with increased heroin use. Such a relationship, however, is less clear for violent crimes other than robbery.

Prevalence and Scope of Property and Violent Crime. Several key studies reveal an exceptionally high prevalence of property crime among narcotic addicts. Anglin and Speckart (1988) found that 82 percent of a sample of 386 California male narcotic addicts reported involvement in property crime over an average five-year period of daily narcotic use. Anglin and Hser (1987) reported that 77 percent of a sample of 196 female narcotic addicts from California admitted to involvement in property crime during an average six-year narcotic addiction period. Inciardi (1986) noted that almost all of a sample of 573 male and female narcotic abusers from Miami had reportedly engaged in theft during the year prior to interview. Inciardi also found that these individuals reported involvement in more than 77,000 property crimes (on average, 135 per subject) over a 12-month period while at large in the community. This figure included 6,669 burglaries, 841 vehicle thefts, 25,045 instances of shoplifting, and 17,240 instances of

fencing. While these studies varied in sampling methods and definitions of property crime (e.g., including and not including robbery), all provided evidence that a substantial majority of narcotic abusers routinely engage in property crime.

Property crime comprises a considerable portion of the crime, other than drug distribution, committed by narcotic addicts. For instance, Nurco et al. (1991a) found that of the nondistribution crimes committed by a sample of 250 male narcotic addicts during an average 7.5-year addiction period, approximately 48 percent were property crimes.

Research has also consistently documented that among heroin addicts, violent crime is less prevalent and occurs with less frequency than property crime. Earlier studies noted that addicts tended to prefer property crime over violent crime and appeared to be less violent than other offenders. While findings from later studies have continued to show that violence accounts for only a small proportion of all addict crime (approximately 1% to 3%, a rate that is much smaller than the property-crime figure), the actual number of violent crimes is still quite large because addicts commit so many crimes. For example, in Inciardi's 1986 sample of 573 Miami narcotic abusers, violent crime comprised only 2.8 percent of all offenses (5% of nondistribution offenses) committed by the subjects in the year prior to interview. However, this relatively small percentage amounted to 6,000 incidents of violent crime (on average, 10.4 per subject), since a total of 215,105 offenses were committed.

Researchers have also suggested that heavy heroin use and, more recently, heavy cocaine abuse have contributed to record numbers of homicides in large cities in the United States. The ways in which drugs can contribute to violence is the basis for a prominent theory in the drugs-crime field, discussed later in this article.

Crime and Frequency of Heroin Use. Recent studies have provided consistent evidence of a direct, functional relationship between the frequency of narcotic drug (primarily heroin) use and the frequency of property crime. These investigations have employed a unique longitudinal design in which crime data are obtained for each subject over periods during which the frequency of narcotic use may vary. These studies of addiction careers reveal that property-crime rates are significantly higher during narcotic addiction periods than during periods of nonaddiction. Such a relationship tends to be linear, with the highest property-crime rates occurring at the highest levels of narcotic use (three or more times per day). In addition, although most addicts commit property crime prior to addiction, the frequency of such crime increases significantly from preaddiction to addiction, remaining high over subsequent addiction periods and low during intervening nonaddiction periods. While other factors also influence property-crime rates, the simplest explanation for these results is that property crime is functionally related to narcotic addiction—since addicts need cash to support their habits.

Evidence of a similar relationship between heroin use and violent crime is less conclusive. Studies have consistently shown that rates for robbery, in which there are property-crime features, are considerably higher during addiction periods than during either preaddiction or nonaddiction periods. However, when rates for composite measures of violence and rates of assault alone are examined, the relationship appears less clear.

In compiling composite measures of violence, Ball et al. (1983) found that for a sample of 243 male Baltimore addicts, the number of days on which violent crime was committed was considerably higher during the first addiction period than during the first nonaddiction period. However, in subsequent studies of 250 male addicts from Baltimore and New York City, most of whom had multiple periods of addiction, more complex relationships were observed. Over an addiction career, violent-crime rates for the total sample were significantly higher for combined addiction periods than for combined nonaddiction periods (Nurco et al., 1986; Nurco et al., 1988a). This result stemmed largely from high levels of crime committed during the first addiction period; violent crime actually decreased over subsequent periods of addiction, a finding that appeared to be age-related. The fact that mean rates for violence were found to be even higher for preaddiction (10 days per year) than for addiction periods (8 days per year) also reflected an inverse relationship between age and violent criminal activity.

The 1999 ADAM report on U.S. drug use of arrestees reveals that opiate use among adult arrestees remains relatively low compared to the prevalence of cocaine and marijuana in the overall sample. For female arrestees the median rate for

testing positive to opiates was 8 percent in 1999 and for male arrestees it was 6 per cent.

Nonnarcotic Drug Use. Investigation of the nonnarcotic drugs-crime relationship has only recently emerged as a major research question. In the 1980 literature review, Gandossy and associates found that, of the few studies conducted on the nonnarcotic drug-crime relationship, evidence linking the use of various nonnarcotic substances to either property crime or violent crime was weak. Another reason for the unclear relationship between nonnarcotic drug use and criminal behavior is that various narcotic and nonnarcotic drugs are often used in combination. Thus, disentangling their separate relationships to criminal activity, let alone determining cause and effect, is especially problematic. Despite these difficulties, significant advances have been made in understanding the nonnarcotic drugs-crime relationship since 1980.

Cocaine. Data analyses on a nationwide probability sample of 1,725 adolescents strongly supported a cocaine-crime connection (Johnson et al., 1991). Adolescents who reported using cocaine in the year preceding the interview (comprising only 1.3% of the sample) were responsible for a disproportionately large share of the property and violent crime committed by the sample during this period. The cocaine users accounted for 60 percent of all minor thefts, 57 percent of felony thefts, 41 percent of all robberies, and 28 percent of felony assaults committed by the entire sample.

Typological studies involving seriously delinquent youth and female crack-cocaine abusers also revealed that subjects who reported the heaviest levels of cocaine use also had substantially higher rates of property and violent crime than subjects who used crack less frequently. Among a sample of 254 youth identified by Inciardi and coworkers (1993a) as serious delinquents, the 184 CRACK dealers (86% of whom were daily crack users) were responsible for 45,563 property crimes (an average of 231 per user) during the year preceding the interview. In contrast, the seventy subjects who were not crack dealers and who used crack less frequently (approximately three times per week) averaged 135 property crimes per year. In addition, the heavy cocaine users averaged ten robberies per year, compared with one per year for the remaining subjects. Similar results were reported for a sample of 197 female crack abusers (Inciardi et al., 1993b). The average adjusted annual rates for the 58 subjects classified as heavy cocaine users (8 or more doses per day) were 12, 14, and 320 for violent crime, major property offenses, and minor property crimes, respectively. These rates were substantially higher than rates for the 90 subjects classified as "typical" users (4–7.99 doses per day). For those forty-nine users who took less than four doses per day, the average adjusted annual rates for violence and major property crime were less than one, and the rate for minor property offenses was twenty-four.

Increased cocaine use among narcotic addicts has also been associated with increased property and violent-crime rates. Both Nurco et al. (1988b) and Shaffer et al. (1985) found that male narcotic addicts who had higher rates of cocaine use tended to have higher rates of property and violent crime than addicts who did not abuse cocaine.

The 1999 ADAM report found that cocaine use among adult arrestees remained high, with cocaine found in more than one-third of adult arrestees in twenty sites. There was substantial variation in the proportion of those testing positive for cocaine. In three sites (Atlanta, Chicago, New York City), more than 60 percent of adult female arrestees tested positive for cocaine. In six other sites, however, cocaine use was less than 25 percent.

Other Nonnarcotic Drugs. The use of other nonnarcotic drugs appears to be unrelated to increased criminal activity. While there is considerable evidence that frequent users of *multiple* nonnarcotic substances, including amphetamines, BARBITURATES, marijuana, and PCP, typically have high crime rates (although somewhat lower than rates for heroin addicts), such is not the case for users of *single* non-narcotic drugs. Although single use may be related to offenses like disorderly conduct or driving while impaired, it is not generally associated with predatory crime.

Marijuana. Research on the relationship between marijuana use and crime has found that, with the possible exception of the sale of the drug and disorderly conduct or driving while impaired, the use of marijuana is not associated with an increase in crime. Some studies have reported that marijuana use may actually reduce inclinations toward violent crime.

A major problem in studying the association between marijuana use and criminal behavior is that the exclusive use of marijuana is generally short-lived. Further, like other illicit nonnarcotic sub-

stances, marijuana is often used in combination with other drugs. Under such circumstances, it is difficult to isolate the effects of heavy marijuana use from those associated with the use of various drug combinations.

The 1999 ADAM report disclosed that marijuana remains a very popular drug for adult arrestees, particularly among young males between 15 and 20 years old. The median rate of marijuana positives for this group of arrestees was 63 percent compared to the overall adult male arrestee median rate of 39 percent and the overall adult female arrestee median rate of 26 percent.

Amphetamines. Literature reviews published during the late 1970s and early 1980s (Gandossy et al., 1980; Greenberg, 1976) reported that the association between amphetamine use and crime was difficult to determine because, among other factors, of the diversity of amphetamine users. More recent ethnographic studies of drug abusers (Goldstein, 1986) have reported that amphetamine use is related to violent crime in some individuals. In the general population, however, the association between amphetamine use and crime is not readily apparent. Despite assertions of the media in the 1960s and 1970s, the prevalence of amphetamine-related violence among American youth is likely to be quite low.

The 1999 ADAM report indicated that methamphetamine use among ADAM arrestees is concentrated mainly in the Western part of the United States. A large number of sites had virtually no presence of methamphetamine. However, prevalence rates exceeded 10 percent both for adult female arrestees in twelve sites and for adult male arrestees in 9 sites.

Psychedelic Substances. Most studies investigating the relationship between psychedelic-substance abuse and crime have involved PHENCYCLIDINE (PCP). Much of this research has examined the relationship between PCP and violence. As in the coverage of many other nonnarcotic drugs, media reports, principally in the 1970s and early 1980s, emphasized a perceived link between PCP use and violent behavior. However, the actual extent of this link is greatly exaggerated. In his report on the subject, Kinlock (1991) noted that serious methodological problems in some studies and contradictory findings in others disallowed a conclusive answer to the question of whether PCP use increased violent crime. Researchers have suggested, nevertheless, that the inconsistency of study findings may indicate that PCP use facilitates violence in a small proportion of users (Inciardi, 1986; Kinlock, 1991). There is agreement that biological, psychological, situational, and other factors underlying seemingly drug-related aggressive behavior should be examined in future research.

THEORIES ON THE DRUGS-CRIME RELATIONSHIP

Inciardi (1986) has noted that numerous theories have been posited to explain the drugs-crime relationship. Many of these theories have dealt with the etiology of drug use and crime. Early etiological theories tended to be overly simplistic, focusing on what Inciardi termed the "chicken-egg" question: Which came first, drugs or crime? This question polarized the drugs-crime field for over fifty years. It typically reflected two mutually exclusive positions: that addicts were criminals to begin with, and addiction was simply another manifestation of a deviant lifestyle; or that addicts were not criminals but, rather, were forced into committing crime to support their drug habits.

Reflecting a middle-ground position, more recent theories argue for a diversity among narcotic addicts with regard to the predispositional characteristics and motives underlying drug-related criminal behavior. For example, on the basis of their research with narcotic addicts, Nurco and his associates (1991b) concluded that there is considerable variation among addicts in their propensity toward criminal activity. Some addicts had been heavily involved in crime prior to addiction, whereas others were extensively involved in crime only when addicted.

In the late 1970s, drugs-crime theories became increasingly complex, partly because studies tended to have fewer methodological problems that interfered with the measurement of both drug use and crime. With improvements in techniques, researchers gradually become more aware of heterogeneity among drug abusers on many dimensions, including the type and severity of drug-use patterns and related criminal activity. Also, more recent studies have found that drug use and crime, in most instances, do not initially have a causal relationship but, rather, are often the joint result of multiple influences. Among the many factors contributing to drug use and/or crime are those involving the fam-

ily, such as lack of parental supervision, parental rejection, family conflict, lack of discipline, and parental deviance; association with deviant peers; school dropout, failure, and discipline problems; and early antisocial behavior. Consistent with the notion that all drug abusers are not alike, varying combinations of factors probably contribute to different patterns of deviant behavior in individuals at risk.

However, as Inciardi and his associates (1993a) have noted, some limitations of these theories remain. Most theories discuss drug abuse only as one of several manifestations of delinquency. Further, as in earlier years, the primary concern has been with the etiology of deviant behavior. Very little attention has been paid to explaining events that occur after the onset of drug use and criminal behavior, specifically how certain types of drug abuse increase the frequency of criminal activity. Finally, theories have typically focused on adolescence, without incorporating attributes and events that influence behavior during childhood and adulthood.

Among the most prominent theories in the drugs-crime field is that of Paul Goldstein (1986, 1989) regarding the relationship between drugs and violence. Goldstein's theory is based on his numerous ethnographic accounts of violent drug-related acts obtained from both perpetrators and victims in New York City. According to this theory, drugs and violence can be related in three separate ways: psychopharmacologically, economic-compulsively, and systemically. Within the psychopharmacological model, violent crime results from the short- or long-term effects of the ingestion of particular substances, most notably crack-cocaine and heroin. According to the economic-compulsive model, violent crime is committed as a means to obtain money to purchase drugs, primarily expensive addictive drugs such as heroin and cocaine. The systemic model posits that drug-related violence results from the traditionally aggressive patterns of interaction found at various levels within systems of illicit-drug distribution. Examples include killing or assaulting someone for failure to pay debts; for selling "bad," or adulterated, drugs; or for transgression on one's drug-dealing "turf."

Several key studies have analyzed data in the light of Goldstein's concepts. In a study of 578 homicides in Manhattan in 1981, 38 percent of the male and 14 percent of the female victims were murdered as a result of drug-related activity (Tardiff et al., 1986). The investigators contended that these percentages were higher than those previously reported in the United States. In a subsequent study by Goldstein and his coworkers (1989) involving 414 homicides in New York City that occurred over an eight-month period, 53 percent were classified by the police and researchers as being drug-related. In both studies, most of the drug-related homicides were attributed to systemic causes. Interestingly, in the former study, most of the homicides involved heroin, whereas in the latter study, most involved crack-cocaine.

Drug Use and High-Rate, Serious Criminality. As indicated earlier, the onset of illicit drug use typically does not result in the onset of criminal behavior. In most cases, both drug use and crime begin in the early teens. Generally, the less serious the drug or crime, the earlier the age at onset of involvement. For example, among illicit drugs, marijuana is more commonly used at a younger age than are sedatives or tranquilizers, and these drugs, in turn, are typically used at a younger age than are "hard" drugs, such as heroin and cocaine. Similarly, minor forms of crime (e.g., shoplifting, vandalism) have an earlier onset than more serious types of crimes, such as assault, robbery, and drug dealing.

Most marijuana users do not become heroin addicts, and most youths who commit minor property crimes do not subsequently become involved in more serious offenses. In both instances, the salient variable appears to be age of onset—the younger the individual is when first using a "soft" drug or committing a minor crime, the more likely he or she will move on to more serious forms of deviance. In general, the more deviant the environment (family, peers, community), the earlier the onset of deviance.

Since 1980, independent studies have identified several core characteristics of high-rate, serious offenders. According to Chaiken and Chaiken (1990), these studies have consistently found that predatory individuals tend to commit many different types of crime, including violent crime, at high rates, and to abuse many types of drugs, including heroin and cocaine. Also, research findings have consistently reported that among heroin addicts, prisoners, and seriously delinquent youth, the younger one is at onset of heroin and/or cocaine addiction, the more frequent, persistent, and severe

one's criminal activity tends to be. In these studies, individuals with early onsets of addiction (typically before age 16) tended to abuse several types of drugs and to have disproportionately high rates of several types of crime, regardless of addiction status. Such findings have been observed in various geographic locations and are independent of ethnic group. These results are similar for both males and females, with one notable exception: females with early onsets of addiction are more likely to commit prostitution, shoplifting, and other property crimes at high rates, whereas males with early onsets are more likely to commit violent acts.

Chaiken and Chaiken's 1982 study of over two thousand male prisoners in three states was significant for at least two reasons. First, it challenged the long-held perception that drug abusers were less violent than other arrestees. While 65 percent of Chaiken and Chaiken's sample reported having used illicit drugs during the one- to two-year period preceding the arrest leading to the most recent incarceration, an even higher proportion (83%) of high-rate, serious offenders, identified as "violent predators," had used drugs during the same period. Among the offenders studied, violent predators were also most likely to have had histories of "hard" drug use (including heavy multiple-drug use and heroin addiction) and to have had early onsets of several types of drug use and criminal activity. Second, and perhaps more important, the information on an offender's drug history was more likely than official arrest records to be related to the amount and seriousness of self-reported criminal activity. As in the results of drug-crime studies discussed earlier, official arrest data were poor indicators of the type, amount, and severity of crime committed by these respondents.

These findings suggest a potential for using an individual's history of illicit drug use, including age of onset, in identifying high-rate, dangerous offenders. However, this approach has several limitations. First, a general caution is in order whenever findings based on aggregate data are applied to the individual case. Second, although self-reports of drug use and crime are generally valid when obtained from individuals who are either at large in the community, entering a drug-abuse treatment program, or already incarcerated, they tend to be less accurate for individuals being evaluated for initial disposition in the criminal-justice system. Approximately one out of every two new arrestees identified as drug users by urine testing conceal their recent drug use, even in a voluntary, confidential interview having no bearing on their correctional status.

(SEE ALSO: *Antisocial Personality; Conduct Disorder; Crime and Alcohol; Families and Drug Use; Family Violence and Substance Abuse*)

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CROP-CONTROL POLICIES (DRUGS)

Eliminating drug crops at the source through crop eradication and/or crop substitution has been a central, or at least an integral, part of U.S. international narcotics-control policy for the past twenty years. U.S. government policy officials maintain that eradication of illicit narcotics closest to the source of the raw material represents the most cost-effective and efficient approach to narcotics control within the overall supply-reduction strategy. The source of the illicit crop is believed to be the most commercially vulnerable point in the chain from grower to user. Since 1990, however, U.S. government policy officials have shifted away from crop control in favor of enhanced interdiction and targeting major trafficking organizations. Despite the best efforts of the United States and cooperating drug-SOURCE COUNTRIES, controlling the crop has been a difficult, if not impossible, task. Several HEROIN and MARIJUANA crop-control successes have occurred, most notably in MEXICO and COLOMBIA, but these programs had their problems. To date, notwithstanding minor, short-term successes in BOLIVIA, coca crop-control has remained an elusive goal in the Andes. Undertaking a drug crop-control program involves political as well as economic costs for both the source country and the United States.

CROP-ESTIMATING METHODOLOGIES

For more than a decade, the U.S. government has estimated the total acreage under illicit-drug cultivation at home and abroad, applying proven methods similar to those used to estimate the size of legal crops. The government knows with less certainty, however, actual crop yields (the amount of coca leaf or OPIUM gum produced per acre). Soil fertility, weather, farming techniques, and plant diseases can produce wide variations in crop yields. Given the clandestine nature of the drug business and variations from year to year and place to place, the government cannot estimate accurately the quantities harvested and available for processing. Furthermore, wide variations in processing efficiencies (depending on the orgin and quality of raw material, technical processing method, size and sophistication of laboratories, and the skill and experience of workers and chemists) make cocaine and heroin production estimates extremely complicated. Using commonly believed processing effi-

ciencies, the government estimates a range for COCAINE and heroin production. Estimating the amount of this production that enters the United States poses still more difficult challenges.

The government uses two principal methods of estimating illicit-drug acreage under cultivation: (1) photographic-based aerial surveys: and, (2) remote sensing from satellite surveillance. Both methods have validity and reliability problems, but aerial surveys matched by ground truth (the verification of cultivation in the areas photographed) produce the best estimates. Satellite surveillance data are problematic because of weather, instrument calibration, cultivation under foliage, the small size of fields, false positives and negatives that result from color spectrum (signature) inaccuracies, and lack of ground truth.

EFFECTIVE CROP-CONTROL STRATEGIES

Many believe the illicit-drug trade is most susceptible to disruption at the organizational center of gravity—the traffickers' home country of production. Once the product leaves the production area and enters the distribution networks, it becomes more difficult to locate and control. Consequently, the U.S. government's drug-supply reduction programs have historically focused major attention on the drug source, which represents the smallest, most localized point in the grower-to-user chain.

International supply-reduction efforts close to the source of the drug also complement domestic supply-and-demand reduction efforts and give them a better chance of success. The U.S. 1991 *National Drug Control Strategy* states that when it is judged to be feasible politically, particularly when the market price of the raw product has been depressed below the cost of production, cooperative efforts can and should be taken to reduce the net cultivation of the NARCOTIC crops. Such crop control, or eradication, would then occur through manual or herbicidal means, crop substitution, income replacement, and area-development projects that provide income and raise the standard of living. Additionally, efforts would be made to convince the cultivators to plough under or cut down their drug crops voluntarily or not to plant them in the first place.

A Mexican federal policeman prepares to get off a helicopter as it lands near a large marijuana field slated for destruction in Mocorito, Sinaloa, Mexico, August 28, 1995. (AP Photo/Dario Lopez-Mills)

Crop-control strategies are important for cocaine, heroin, and marijuana reduction, although neither the United States nor the source governments have much control over, or much access to, the largest opium-producing reigons of Southwest Asia (the Middle East) and Southeast Asia (especially Myanmar [formerly Burma] and Afghanistan, the world's largest producers of illicit opium). The Peruvian government has exercised only limited sovereignty over the world's largest area of coca cultivation (for cocaine), the insurgent-controlled Upper Huallaga Valley (UHV). When government commitment and ability exists for controlling the source of the illicit product, crop eradication and/or income substitution can be effective ways to achieve a net reduction in the production of the illegal crop.

COCA

Both a legal and an illegal commodity, the COCA PLANT is mainly grown in Andean South America—in Peru, Bolivia, and Colombia; it belongs to the genus *Erythroxylon* within the family Erythroxylaceae. Most cocaine comes from the leaves of 2 of the 250 identified species: *E. coca Lam* and *E. novograntense*. Each of these species, in turn, has two varieties. Agriculturally speaking, coca is a hardy, relatively labor-free, subtropical perennial plant that thrives at high to somewhat lower elevations and in dry to slightly humid climates, depending on variety. Coca plants are shallow-rooted, broad-leaved woody shrubs that grow to heights of

3 to 10 feet (1 to 3 m) and live about 30 years (White, 1989; LaBattAnderson, 1990). It takes 30 to 36 months before the coca bush is mature enough to produce leaves that can be used in the production of cocaine. Once the plant is mature, three to four crops of coca leaves may be harvested per year for an estimated yield of 1 to 3 tons per acre (0.8 to 2.7 metric tons [MT] per hectare) of dry coca leaf cultivated per year. Actual yield depends on microclimate, plant maturity, and species. After harvesting and drying, the leaves are soaked in a mixture of solvents, and the resulting COCA PASTE is precipitated, further refined to coca base, and finally refined to cocaine hydrochloride (HCl), the salt or white powder form of cocaine. Leaves weighing 1.1 tons (1MT) produce approximately 6.6 pounds (3 kg) of paste. These 6.6 pounds (3 kg) of paste, called *pasta*, are then converted to about 3.3 pounds (1.5 kg) of coca base, which is equivalent to 3.3 pounds (1.5 kg) of cocaine HCl. The U.S. government estimates that the Andean countries produced approximately 900 tons (816 MT) of cocaine for world consumption in 1991 (INCSR, 1992).

OPIUM POPPY

Unlike the perennial coca bush, the opium poppy flower is an annual that requires the planting of seeds for each crop. Poppies of many species grow throughout the world, but only *Papaver somniferum* yields opium and its derivatives—the medicinal analgesics CODEINE and MORPHINE as well as the now illicit addictive heroin. Once planted, the life cycle of the labor-intensive poppy lasts for 120 to 150 days, from planting until the petals fall off. Day and night, certain nitrogen-containing compounds, alkaloids, are produced by the plant and stored in its cells. After the petals fall, the seed capsule swells and is incised while still green. A milky alkaloid-rich sap seeps from tiny tubes in the capsule wall, which dries, darkens, and turns gummy—becoming a substance called opium gum. Raw opium gum is converted by crude refineries into morphine base; a few pots, simple chemicals, and a source of fresh water are all that is needed to create the morphine base from which illicit heroin is made. About 22 pounds (10 kg) of opium make 2.2 pounds (1 kg) of morphine base; treating the base with acetic anhydride creates heroin. *Papaver somniferum* is cultivated in dozens of varieties adapted to do well in various soils and climates, ranging from southern Sweden to the Equator. Depending on the soil and climatic conditions, the growers can harvest at least two crops per year (White, 1985). Although the opium poppy seems to flourish at about 3,000 feet (915 m) in low humidity, it also grows and survives in humid lowlands, under foliage, or in full sunlight. With the aid of irrigation and pesticides, the poppy growers have expanded the conditions and acreage in which the plant will produce. The U.S. government estimated that 4,187 tons (3,800 MT) of opium, or approximately 418 tons (380 MT) of heroin—if the total were converted—were produced throughout the source countries of Southeast Asia, the Middle East, Mexico, Guatemala, and Colombia.

CANNABIS

Marijuana, a by-product of the plant *Cannabis sativa*, remains the most commonly used illicit substance in the United States, although its use has been decreasing steadily for the past several years. Both the plant and its psychoactive ingredient TETRAHYDROCANNABINOL (THC) are controlled substances. The U.S. government estimates that Mexico still supplies the majority of the marijuana available in the United States, perhaps as much as 63 percent. Domestic supply accounts for another 18 percent, Colombia for 5 percent, Jamaica for 3 percent, and the remaining 11 percent comes form Belize, Laos, the Philippines, Thailand, Lebanon, Pakistan, and Afghanistan. Brazil and Paraguay also cultivate cannabis, but the majority is consumed locally or exported to neighboring countries, with very little, if any, finding its way to the United States (NNICC, 1990).

The flowering tops and the leaves of the marijuana plant are collected, dried, and used for psychoactive effect—usually smoked as a cigarette or in a pipe but also ingested as an ingredient of food. The plant is an annual; it is planted from seed and harvested traditionally in two seasons of five- to six-month cycles each year. Cannabis can grow almost anywhere outside of the ice-bound frigid zones, if provided with adequate sunshine and water. As a chlorophyll-based green plant, it requires photosynthesis to grow and mature. Coating the plant's leaves with a contact herbicide such as paraquat or glyphosate has been very effective in killing the plant in Mexico, Belize, and Colombia.

Unlike the coca plant, which has a perennial root system, most types of marijuana can be destroyed easily by digging, spraying, or cutting.

CROP ERADICATION

Illicit crops probably constitute the least expense in the narcotics chain. Producers devote few economic resources to prevent detection, although it is easier to locate and destroy crops in the field than to locate the processed drugs once they enter the smuggling routes or on U.S. city streets. Despite this belief, however, few effective crop-control policies have been implemented, and crop control remained a secondary approach, at best, in the U.S. government's 1991 and 1992 *National Drug Control Strategy*. It was easier for U.S. agencies to function at the border or within the United States than to get compliance on command from source countries. DRUG INTERDICTION and immobilizing the trafficking organizations were the preferred policy approaches.

Crop eradication can be effected forcibly or voluntarily (1) by manual plant removal, (2) by biological control through the use of pathogens or predators, or (3) by the use of herbicides. Of the three methods, herbicidal eradication has been the most effective and efficient, though not the most neutral politically. Payment to the growers for the labor of uprooting the plants voluntarily is an important short-term element, while development assistance is a longer-term component of the successful implementation of any eradication effort that invites voluntary reduction.

Because the coca bush is a perennial, destroying the plant can have a devastating effect on the productive capacity of the trafficking organizations. After the plant dies, it would take nearly three years for the grower to be reemployed on that land productively. For the most part, the coca fields of Peru, Bolivia, and Colombia are remote relatively small plots. There is little or no intercropping, where the grower mixes his coca fields with yucca or other agricultural produce, therefore, aerial herbicidal eradication is an efficient option—but one that requires significant amounts of herbicide to kill the hardy coca bush. The opium poppy and the marijuana plant are easier to eradicate than coca bushes but, because they are annuals and planted from seed, with several harvests a year, they require year-round crop-control efforts.

Manual Eradication. Manual eradication involves physical removal or cutting. For coca, removing the plant is more effective than cutting, because coca can sprout new shoots from the base of the stump. The plants, however, are difficult to remove after three years of growth because of their root systems. Coca, poppy, and marijuana plants can be removed only if grown in areas that are easily accessible. Many of the illicit growing areas are in remote corners of the countryside, so transporting personnel and equipment may be expensive and hazardous. More importantly, the manual eradicators place themselves at great personal risk, as for example in coca-growing areas of Peru and Colombia—where violence directed at eradication personnel have forced the suspension of such efforts.

Biological Control. Numerous biological control agents—parasites, predators, pathogens—have been identified that may destroy coca and poppy plants. Too little is known, however, about the effect of these agents on the ecology of the growing regions. Another issue is the possible negative impact on legitimate crops, which, if inadvertently destroyed, could cause famine and/or severe economic losses. Use of biological agents may be a future possibility, but the current state of research on such pests and their potential impact does not make this option feasible yet.

Herbicides. Control of coca by foliar application of herbicides was attempted in Colombia in 1985, with inconsistent results. In 1987 and 1988, further small-scale testing was conducted in Peru, involving both foliar- and soil-applied herbicides, which confirmed the efficacy of soil-applied herbicides. Two herbicides were chosen for further testing: tebuthiuron and hexazinone. Based on further tests in Peru in 1989, researchers learned that aerial application of either pellet tebuthiuron or granular hexazinone, at lower rates than used typically in the United States, can kill a significant percentage of coca plants in a field and force its abandonment. Environmental tests were conducted in Peru for more than two years to measure such ecological effects as translocation of the herbicide into the soil, water, and air; water solubility; effect on the flora and fauna; and ability of the herbicide to leech on the clay molecules in the soil. Tebuthiuron and hexazinone were judged environmentally safe and effective. Other herbicides such as dicamba, imazapyr, picloram, and triclopyr

were also tested and found to be more toxic but less effective. Use of herbicides on poppy and marijuana has been tested and thoroughly documented. Concise environmental reviews (measuring impact on environment) and environmental-impact statements (measuring health consequences for consumers who use the drug product after it has been sprayed with herbicide) have been filed for 2,4-D, glyphosate, and paraquat use on poppy and marijuana fields.

CROP-ERADICATION SUCCESSES

In Mexico, Colombia, Belize, Myanmar (formerly Burma), Bolivia, Jamaica, and Thailand, croperadication efforts continue to have varying degrees of success in reducing illicit crop cultivation. In the mid-1970s, Mexico began an aerial herbicidal-eradication program on both opium and marijuana and reduced the cultivation of these illicit crops significantly. In 1991, Mexico reportedly destroyed some 16,000 of its 25,000 acres (6,500 of its 10,000 hectares) of opium and 27,000 of its 71,500 acres (11,000 of its 29,000 hectares) of cannabis. In the early 1980s, the Colombian government used glyphosate in the north to eradicate most of its marijuana there. In the early 1990s, Colombia planned to use the same herbicide on newly discovered opium in the Cauca and Huila departments. In 1987, the U.S. government supported the government of Belize in an aerial marijuana-eradication program, which resulted in a 90 percent decline in cannabis production.

In the 1987–1988 growing season, Myanmar sprayed the herbicide 2,4-D from fixed-wing agricultural-spray aricraft to destroy about 31,000 acres (12,500 ha) of opium poppy. This program came to a halt late in 1988 when the government moved its limited military resources to attack mounting antigovernment protests. The political balance also changed abruptly when Myanmar's ruling military eliminated the opium-eradication program, abolished bureau rights, and accommodated certain trafficking insurgents (the Wa and Kokang Chinese components of the now-defunct Burmese Communist Party, who were lighting one of Burma's principal enemies, Khung Sa and his Shan United Army).

In addition to these aerial herbicidal-eradication efforts, manual eradication of crops continued in 1991 in a number of countries, to include destruction of nearly 30 percent of Thailand's 10,500 acres (4,200 ha) of opium, about half of Colombia's 6,000 acres (2,500 ha) of opium, approximately 10 percent of Bolivia's 131,000 acres (53,000 ha) of coca, and a little less than half of Jamaica's 4,500 acres (1,800 ha) of cannabis.

CROP-ERADICATION DIFFICULTIES

Conceptual, political, and technical arguments are often raised against drug-crop eradication. Opponents of eradication believe that the reduction of foreign supplies of illicit drugs is probably not achievable, or short-term at best; they say that even if eradication had a longer-term impact in the source country, it would not have a meaningful effect on levels of illicit-drug consumption in the United States, where the consumer would simply switch to other available drugs. Moreover, some fear that inordinate environmental damage will result from herbicide use. Others question whether a global policy of crop control is feasible politically, because many growing areas are far beyond government control, and even when there is government jurisdiction, crop eradication becomes impractical because the grower can continually shift areas of cultivation. Instituting effective eradication efforts in some source countries, such as Peru, might also drive political insurgents (who co-locate with the drug traffickers) into threatening alliances that would undermine the central govenment even further. Finally, some question the value of supply-reduction efforts at the source altogether, since world production and supply of illicit drugs vastly exceed world demand. If the worldwide supply were reduced dramatically, it would not be felt in the United States until the supply had dried up throughout the rest of the world, because U.S. consumers often pay higher prices than those in any other market; moreover, U.S. dollars are the preferred narco-currency (Perl, 1988).

CROP-SUBSTITUTION EFFORTS

Crop substitution—the replacement of opium, coca, or marijuana production with a legal agricultural commodity—can never be successful by itself, because of the immense profits from illicit-drug cultivation. A more broadly defined income-replacement approach (which may include an agricultural crop-substitution component), however,

coupled with strong law enforcement, may succeed in convincing drug growers to stop planting the illicit crop.

In the Malakand District of Pakistan's North-West Frontier Province, the U.S.—Pakistani efforts in the early 1980s to implement a fully integrated, rural development project, which provided roads, water, electrification, and agricultural substitutes (e.g., peanuts, apples), resulted in a net reduction in opium poppy production. Providing project support for the 300,000 inhabitants of the district enabled local residents to earn an income from an extensive road-building and infrastructure development program, thus making cultivation of the opium poppy unnecessary. A side benefit of the development efforts was the creation of valuable infrastructures to raise the standard of living throughout the region and encourage the nomadic populations to establish roots and achieve more stable living conditions.

The Highland Village Project in northern Thailand has provided similar benefits to the culturally diverse, opium-producing hill tribes. This resulted in decreased opium cultivation consistently in the early 1990s. In Laos, the Houaphanh project near the remote border region with Vietnam began in 1990 to provide area development incentives (of improved water and roads and medical and educational benefits) for growers who opted to cease planting opium. In the Western Hemisphere, a principal component of the Andean Strategy to eliminate illicit coca in Peru and Bolivia has an economic-assistance element that provides hard-currency earnings, trade incentives, and local project assistance to entice the growers away from coca cultivation. Some funds have already been expended on agro-research (discovering viable crops), infrastructure development, extension training, and rudimentary marketing.

In the broadest sense of the term, crop substitution worked rather effectively in Turkey in the early 1970s when a government cash subsidy permitted the farmers to harvest the poppy *before* the plant ripened to produce the opium gum. In this way, the traditional cooking and ceremonial uses of poppy could be maintained through the poppy straw process, as it is called, but the seed pod would not be available for the illicit opium gum.

CROP-SUBSTITUTION DIFFICULTIES

Several inherent difficulties exist with crop-substitution approaches.

1. Many of the growing regions are remote inhospitable areas, outside central government control.
2. In a free-market economy, no legitimate crop can compete with either coca or opium as an income-producing agricultural commodity. Even if there were competitive substitutes, with the immense profits from the drug trade, the drug traffickers could continue to raise the price to compete for willing cultivators.
3. Much of the land in the growing zones cannot produce legitimate agricultural products sufficient to support the farming population.
4. The presence of political insurgents and threat of violence in some of the growing areas (Peru, Myanmar, Afghanistan) create an unfavorable climate for crop substitution.
5. There are difficulties in finding international markets to accept the substitute crop; for example, in Bolivia's Chapare region, oranges and coffee are viable agricultural products, but the international coffee cartel and U.S. citrus growers do not allow Bolivian products to compete for shares of existing markets.
6. In some regions, such as Peru's Upper Huallaga Valley and Bolivia's Chapare, the vast majority of coca cultivators are not farmers and know nothing about agriculture. Many were unemployed urban dwellers and laborers who moved to the coca-growing regions to seek a viable living after the collapse of the tin market in the late 1970s and early 1980s in Bolivia. In the long run, regional development efforts in the urban areas may be required to attract the cultivators back to their places of origin.
7. Successful crop substitution takes years of agro-research, infrastructure development, training, and marketing; it may take too long for subsistence-crop and/or cash-crop producers to make a living. In Pakistan's Malakand project, it took more than five years to develop the agricultural component. Some argue that in the absence of strong law enforcement and control, crop substitution becomes only an *additional* income generator, not a true substitute. The growers will accept the substitute and continue to cultivate the illicit-drug crop.

8. Corruption and powerful interest groups in the growing areas pose serious impediments to any crop-control efforts.

(SEE ALSO: *Foreign Policy and Drugs; Golden Triangle; International Drug Supply Systems*)

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CROSS-DEPENDENCE *See* Addiction: Concepts and Definitions; Tolerance and Physical Dependence

CROSS-TOLERANCE *See* Addiction: Concepts and Definitions; Tolerance and Physical Dependence

CULTS AND DRUG USE The relationship between cults and drug use is complex and contradictory. Traditionally, cults are groups that diverge from major religions or that form new philosophical/religious systems, often around a charismatic leader. Consequently, at any given time, it may be difficult to distinguish a cult from a newly formed religion. Some cults last and become new religions; some remain cults, some die. The line is hard to draw and open to interpretation, even by social scientists and the clergy who specialize in this field.

BACKGROUND

Historically, some cults and cultlike groups have sponsored the use of drugs as an integral aspect of ritual. In ancient Greece, for example, the use of ergot (genus Claviceps), a fungus that grows on grains and causes hallucinations, appears to have played a significant role in the rituals of the Eleusinian mysteries, celebrated in worship of the goddesses Demeter and her daughter Persephone. As poets noted, "I have seen the truth within the kernel of wheat"—a foreshadowing of the Countercultural Revolution/Love Generation, when a purified ergot derivative (LYSERGIC ACID DIETHYLAMIDE, LSD) offered a similar experience. Indeed, the word lysergic means "dissolving ergot."

In Islam, alcohol is forbidden, but medieval Islamic sects were formed to use HASHISH (a form of Cannabis sativa, MARIJUANA). It came into use in the Islamic Middle East only centuries after the Prophet Mohammed (lived about 570 to 632) and his followers founded the Moslem religion; hashish was allegedly used to offer a taste of the paradise to come.

In pre-Columbian America, drugs of a wide variety were utilized in religious rituals; the Native American Church still continues to use the HALLUCINOGENS peyote and mescaline (both de-

More than 900 members of the People's Temple died by mass suicide/murder on November 18, 1979, in Jonestown, Guyana. The leader, Jim Jones, had lured many people into the cult by claiming he would cure their drug abuse problems. (© Bettmann/CORBIS)

rived from the small cactus Lophophora williamsii). Recent court decisions have protected and reaffirmed the right of this church to use these drugs in religious ceremonies. As Preston and Hammerschlag (1983) have noted, this use of hallucinogens is rigidly controlled—part of a transcendent experience, accompanied by rituals of purification, and not lending itself to use on a promiscuous basis.

TWENTIETH CENTURY

The 1960s and 1970s were characterized by an extraordinary youth movement of baby-boomers with an intense interest in the cultic and the occult—and by a popularization of drug use within mainstream American society. Some of this interest was fueled by the philosophies and practices of Asia, especially Southeast Asia, where the Vietnam War was being fought; some was inspired by the Shangri-La nature of the lands of the Himalayas, where Buddhism was practiced in secluded monasteries and nirvana was sought. As the "Greening of America" proceeded through these two decades, mind-altering joined ALCOHOL and NICOTINE, becoming available on the street, and were no longer confined to the disenfranchised or marginal. There was an increasing juxtaposition of the so-called transcendent religious experience (the mind-expanding experience) with drug use that often became drug abuse.

This juxtaposition had been anticipated by some earlier poets, such as William Blake (1757–1827), Charles Baudelaire (1821–1867), and Arthur Rimbaud (1854–1891), by the illustrator Aubrey Beardsley (1872–1898), and by cult figures such as Aleister Crowley (born Edward Alexander Crowley, 1875–1947). By combining aspects of their own experimentation with hallucinogenic drugs with elements of Transcendental Meditation/Mahareshi (movements based on Buddhism) in their song "Lucy in the Sky with Diamonds" (1967), the much adored singing group called the Beatles (the members born between 1940 and 1943, active as a group from 1960 to 1969) both mirrored and promoted the use of hallucinogens as providing a readily accessible transcendental experience—although in Buddhism the goal of all existence is the state of complete redemption (nirvana, a state achieved by righteous living, not by drugs). Unlike Aldous Huxley (1894–1963), who combined an interest in Vedanta (an orthodox system of Hindu philosophy) and the use of mescaline, the Beatles and their alleged mentor, the Mahareshi Mohesh Yogi, proclaimed the desirability of enlightening the masses rather than restricting enlightenment to a righteous educated elite.

In literary works of that era, such as Armistead Maupin's *Tales of the City* (1978), characters routinely advocate and use mind-altering substances (especially marijuana) without any apparent appreciation of their darker potential, which was consistent with the general attitude toward TOBACCO and alcohol use at that time as well. In addition, there was no special appreciation that drug use, in and of itself, might encourage cult affiliation, yet this was very much the time of the rapid growth of cults among youth in the United States.

The relationship of such cults to drug use is paradoxical. Deutsch (1983) has noted that prolonged drug use may encourage this type of cult affiliation, and many cult groups offer themselves to the public and to vulnerable persons as quasi-therapeutic contexts where the person will be able to transcend the need for drugs. This aspect of cult-appeal turned thousands of lost and confused free spirits and flower-children into vacant-eyed smiling cultists who signed over to the cult all their worldly goods—to spend their days wandering the streets, airports, and bus or train stations, seeking donations for their cult by shaking bells and tambourines or by offering flowers to passing

strangers. Rigorous training programs, called "brainwashing" by parents of the lost children and by other skeptics, were fashioned to strip cultists of free will and substitute nodding acquiescence.

THE PEOPLE'S TEMPLE

One charismatic cult leader was the Reverend Jim Jones, leader of the People's Temple. His claim of curing drug abuse was only one of the lures. After moving around the United States for a while, he brought his followers to an isolated spot in South America, where one of the former substance abusers mixed for them a massive batch of poisoned Kool-Aid for the cult's final event—a basically unexplained mass suicide.

SCIENTOLOGY

The People's Temple was not unique—organizations such as Narcanon (that is, narcotics anonymous) have stated that their treatment of substance abusers reflects the dianetics-based teachings of L. Ron Hubbard (born Lafayette Ronald Hubbard, 1911–1986), an American science-fiction writer, whose Scientology movement expanded in the 1950s when he moved to England (he was subsequently banned from re-entering England in 1968). Scientology is a quasi-philosophical system that claims to improve mental and physical well-being as followers advance within the cult, by undertaking (and paying well for) a series of courses.

TWELVE-STEP PROGRAMS

Intense religious commitment is a significant aspect of much of the twelve-step recovery movement. Accordingly, there is concern that this level of commitment to a program can lead to a kind of cult affiliation. ALCOHOLICS ANONYMOUS (AA), the oldest, most constructive, and most respected of the TWELVE-STEP programs, is not considered a cult. Still, Rebhun (1983) and many others have noted the danger that drug-treatment programs can turn into cults such as SYNANON. Synanon was not unique; the history of residential drug-treatment centers includes a number of authoritarian and hierarchical organizations. Recovering substance-abusers often find it very difficult to leave the protection of the THERAPEUTIC COMMUNITY to become independent members of mainstream society. Often times program staff really help individual members overcome drug problems and other problems. Yet other times, a false resolution of these problems comes through fusion with an authoritarian and charismatic leader who will ostensibly provide the continuity and structure for which the substance abuser hungers.

SUMMARY

Drugs and other mind-altering substances have formed an integral part of some cultic/religious rituals from very ancient times. In the mid-to-late twentieth century, the structure provided by groups that mobilize intense religious or quasi-religious feelings has sometimes enabled vulnerable individuals to transcend their personal difficulties. However, the very intensity of the substance user's object hunger may enable the transformation of otherwise viable or valuable organizations into cults or cultlike groups.

(SEE ALSO: *Religion and drug use*)

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CUSTOMS SERVICE, U.S. *See* U.S. Government Agencies: U.S. Customs Service

D

DARE *See* Volume 4, Appendix II, Drug Abuse Resistance Education (DARE) Regional Training Centers

DARP *See* Drug Abuse Reporting Program

DAWN *See* Drug Abuse Warning Network

DAYTOP VILLAGE *See* Treatment Programs/Centers/Organizations: An Historical Perspective

DEA *See* U.S. Government: The Organization of U.S. Drug Policy

DECRIMINALIZATION *See* Marihuana Commission: Recommendations on Decriminalization; Policy Alternatives

DELINQUENCY AND DRUG ABUSE *See* Conduct Disorder and Drug Use; Crime and Drugs; Gangs and Drugs

DELIRIUM Delirium has been defined in many ways. Some use the term to refer to an acute, hyperactive, confusional state. Psychiatrists define it more broadly to describe clinical states characterized by a reduced level of consciousness, an inability by the affected individual to sustain or shift attention appropriately, disorganized thinking, disorientation to time, place, or person, and memory impairment. In addressing the affected individual, questions need to be repeated, the individual may perseverate in responses, and speech may be rambling or incoherent. Additional features include an altered sleep-wake cycle, sensory misperceptions, disturbances in the pace of psychological and motor activity, and varying mood states (e.g., apathy, euphoria). Sensory misperceptions—usually visual ones—may include illusions (e.g., specks on the floor are thought to be insects) or hallucinations (one "sees" a relative in the room when there is actually no one there). Delusions may be present (e.g., the person is convinced that medical staff are secret government agents). The individual may respond emotionally (e.g., with anxiety) and behaviorally (e.g., attack those viewed as threatening) to the context of the delusion. There may be elevated blood pressure, a rapid heartbeat, and sweating and dilated pupils. The onset of such a clinical state is relatively rapid (taking an hour to days), the symptoms fluctuate throughout the course of illness, and the duration is usually brief (about one week). It is important to note that the altered level of consciousness exists on a continuum. Hy-

pervigilance can progress to confusion and drowsiness.

The factors that may cause delirium are numerous. They can include head trauma, infections (e.g., meningitis), metabolic disorders, liver and kidney disease, postsurgical states, and psychoactive substance intoxication and withdrawal. The common underlying functional disturbance in delirium is diffuse impairment of brain-cell metabolism and stability. These changes can frequently be seen on an electroencephalogram (EEG). Delirium can occur at any age but is more common in the very young and the very old. It is most often seen in hospital settings. The treatment of delirium consists of maintaining critical bodily functions (i.e., cardiac and respiratory functions and hydration), correcting the precipitating problem, and managing the psychological and behavioral symptoms.

(SEE ALSO: *Delirium Tremens; Withdrawal: Alcohol*)

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DELIRIUM TREMENS (DTS) This clinical disorder is a DELIRIUM that occurs after abrupt cessation of, or reduction in, ALCOHOL consumption in an individual who has been a heavy drinker for many years. It represents the most severe form of the alcohol WITHDRAWAL state and is not very common. It is associated, however, with a significant mortality rate of those who develop it (10–15%), if left untreated.

Typically, the delirium sets in forty-eight to seventy-two hours after the last drink or after reduction in drinking. The course of illness is generally short, lasting, in most cases, two to three days. The disorder becomes significantly more life threatening if there is concurrent physical illness, such as liver failure, infection, or trauma.

Clinical signs and symptoms are the same ones that are characteristic of a delirium and include disorientation, fluctuating levels of consciousness, vivid hallucinations, delusions, agitation, fever, elevated blood pressure, rapid pulse, sweating, and tremor. The delirium may at times be preceded by a withdrawal seizure. Close monitoring and medical treatment in a hospital setting are required.

(SEE ALSO: *Withdrawal: Alcohol*)

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DEMEROL *See* Meperidine

DEPENDENCE *See* Addiction: Concepts and Definitions; Disease Concept of Alcoholism and Drug Abuse

DEPRESSANTS *See* Drug Types

DEPRESSION The term *depression* has been used to refer both to an emotional state and a group of psychiatric disorders. As an emotional state, it is also known by various comparable terms: dejection, despair, sadness, despondency, lowering of spirits. Cognitions (perceptions and judgments) of a negative nature often accompany depressed mood.

Most people experience brief periods of depressed or despondent mood, often in response to a disappointing life event. Each individual utilizes

different COPING skills and relies on available social supports to deal with such episodes, which generally pass within hours to days.

When a dysphoric mood becomes more severe, is persistent, and impairs functioning, a major depression as a clinical syndrome has developed. Concurrent clinical features include a loss of interest or pleasure in usual activities, a sense of hopelessness, poor or alternatively increased sleep, loss of appetite or overeating with resultant changes in weight, fatigue, anxiety, restlessness, obsessive thinking, difficulty concentrating, irritability, feelings of worthlessness, recurring thoughts of death, and suicidal ideation or an actual attempt to end one's life. Suicidal disturbances are of serious concern; approximately 66 percent of depressed patients contemplate suicide, and it is estimated that 10 to 15 percent succeed. In some cases, psychotic features such as hallucinations and delusions may develop.

Depression is one of the most common psychiatric disorders seen in adults. The lifetime prevalence of major depressive disorder (using DSM-III-R criteria) in the United States is estimated to be 12.7 percent in men and 21.3 percent in women. Some individuals suffer from chronically depressed mood of a less intense nature than that experienced in a major depressive episode; this is referred to as dysthymia. A depressive syndrome may occur as part of manic-depressive illness, and depression as a symptom (i.e., a depressed mood) can be found in many other psychiatric disorders.

Depression should be distinguished from the normal despair of bereavement and from the various medical disorders (e.g., Parkinson's disease) and chemical agents (e.g., alcohol or drugs for heart conditions) that can produce symptoms of depressed mood. The cause of depression is unknown. Biological factors (e.g., dysregulation of neurotransmitter systems), genetic factors, and psychosocial factors (e.g., life events, learned behaviors, and cognitions) have been proposed, and it is likely that all interact to varying extents. Depression is a treatable (but not really curable) illness in the vast majority of people. Treatment consists of a number of modalities, depending on the type and severity of the depression. PSYCHOTHERAPY, antidepressant medications, and electroconvulsive therapy are the main interventions used.

(SEE ALSO: *Causes of Substance Abuse; Complications: Mental Disorders*)

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DESIGNER DRUGS Designer drugs are synthesized chemical analogues of known, dangerous drugs; they are designed to produce pharmacological effects similar to the drugs they mimic. In the pharmaceutical industry, the development of new drugs often utilizes principles of basic chemistry, so that the structure of a drug molecule may be slightly altered to change its pharmacological activity. For therapeutic purposes, these strategies have had a long and successful history; for medical pharmaceutics, many useful new drugs or modifications of older drugs have resulted in improved health care. The principle of structure-activity relationships has been applied to many medically approved drugs in the marketplace, especially in the search for painkillers—nonaddicting opioid analgesics.

The clandestine production of new street drugs is, however, intended to avoid federal regulation and control. This practice can often result in the appearance of unknown substances, with wide-ranging degrees of purity, which have the potential to cause dangerous toxicity and serious health consequences for the unwitting drug user (the quality of personnel involved in clandestine drug synthesis can range from cookbook amateurs to highly skilled chemists). The most publicized case regarding the tragic consequences associated with the manufacture and use of designer drugs on the street involves MPTP (1-methyl, 4-phenyl, 1,2,3,6-tetra-

hydropyridine), a substance that was later found to cause a Parkinsonian syndrome in humans.

A controlled substance that has served as a template for the design of new look-alike OPIOID drugs is MEPERIDINE (Demerol). A slight change in its chemical structure yields the drug known as MPPP (1-methyl-4-propionoxy-4-phenylpyridine), a meperidine look-alike drug, which is known on the streets as synthetic heroin. In California in 1982, four young drug abusers developed Parkinsonian symptoms after the illicit intravenous use of street HEROIN. The analysis of their remaining drug samples revealed the presence of both MPPP and MPTP. The dealer involved in this illicit synthesis and sale of MPPP was a bad chemist, since MPTP represents a side product formed through the inadequate control of the temperature and/or acidity of the chemical reaction.

Another opioid that has resulted in serious health hazards on the street is fentanyl (Sublimaze), a potent and extremely fast-acting NARCOTIC ANALGESIC (painkiller) with a high ABUSE LIABILITY. This drug has also served as a template for many look-alike drugs that come out of clandestine chemical laboratories. Very slight modifications in the chemical structure of fentanyl can result in analogues such as para-flouro-, 3-methyl-, or alpha-methyl-fentanyl—with, respectively relative potencies 100, 900, and 1,100 times that of MORPHINE. During the 1980s, the Drug Enforcement Administration (DEA) has reported a steady increase in deaths from drug overdoses associated with fentanyl-like designer drugs. Not every "designer" drug is actually thought up by chemists in illegal labs; some were actually synthesized for legitimate medical uses but were never marketed. HALLUCINOGENIC drugs, such as LSD or MESCALINE, rarely cause death—except as ACCIDENTS related to drug-induced mental aberrations. Adverse reactions to typical hallucinogens are usually treated by support, reassurance, and a quiet environment. Hallucinogenic designer drugs, however, include such substituted AMPHETAMINE ("speed") analogues as methylenedioxyamphetamine (MDA), methylenedioxymethamphetamine (MDMA or "Ecstasy"), and methylenedioxyethamphetamine (MDEA or "Eve"). Both acute and chronic toxicity have been reported following the administration of these drugs. Acute toxicity is usually manifested as restlessness, agitation, sweating, high blood pressure, tachycardia, and other cardiovascular effects, all of which are suggestive of excessive central nervous system stimulation. Following chronic administration in animals, MDA has been demonstrated to produce a degeneration of serotonergic nerve terminals in rats, implying that MDA might induce chronic neurological damage in humans as well. This also suggests that extreme caution should be exercised regarding the manufacture and use of MDA, MDMA, and related drugs—although a few psychotherapists claim that MDMA is a useful adjunct in the treatment of some patients.

The production of many substances such as methamphetamine, PCP, MDMA and methcathinone requires little sophisticated equipment or knowledge of chemistry. Many clandestine labs are small enough to fit on a kitchen table. (Drug Enforcement Administration)

The widespread illicit manufacture and use of designer drugs with unknown chronic toxicity could result in millions of people experimenting with the drug before the toxic effect was recognized; this could potentially produce an epidemic of neurodegenerative disorders.

(SEE ALSO: *Complications; Controlled Substances Act of 1970; MDMA; MPTP*)

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DESOXYN *See* Methamphetamine

DETOXIFICATION: AS ASPECT OF TREATMENT Detoxification is the term commonly used to describe the process or set of procedures involved in readjusting a drug- or alcohol-dependent person to a lower or absent tissue level of the substance (drug) of dependence. With chronic (long-term) use of many drugs, there is adaptation within the nervous system. Readaptation of the nervous system to the absence of a particular substance can cause a WITHDRAWAL syndrome (as a manifestation of PHYSICAL DEPENDENCE). The patient would reasonably be expected to have symptoms (what they tell the health-care provider) and exhibit signs (what the observer sees) of a withdrawal syndrome.

The detoxification process usually occurs in a supportive environment, which might be a hospital or clinic, but not always; it might also involve the use of medications (other drugs) in order to control or suppress symptoms and signs of withdrawal, but not always. The level of care and the use of medications depends on the substance of abuse and the level of physical dependence (severity of withdrawal syndrome), complications, or potential for complications. The more severe complications are seen most frequently in association with alcohol or sedative-hypnotic withdrawal. The goal of detoxification is to provide a safe and comfortable transition to a drug-free state. Detoxification is generally the first step in the process of treatment for rehabilitation.

(SEE ALSO: *Addiction: Concepts and Definitions; Clonidine; Treatment Types: Overview; Treatment Types: Nonmedical Detoxification; Withdrawal: Alcohol*)

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DETOXIFICATION: NONMEDICAL *See* Treatment Types

DEXTROAMPHETAMINE This is the *d*-isomer of AMPHETAMINE. It is classified as a PSYCHOMOTOR STIMULANT drug and is three to four times as potent as the *l*-isomer in eliciting central nervous system (CNS) excitatory effects. It is also more potent than the *l*-isomer in its ANORECTIC (appetite suppressant) activity, but slightly less potent in its cardiovascular actions. It is prescribed in the treatment of narcolepsy and OBESITY, although care must be taken in such prescribing because of the substantial ABUSE LIABILITY.

High-dose chronic use of dextroamphetamine can lead to the development of a toxic psychosis as well as to other physiological and behavioral problems. This toxicity became a problem in the United States in the 1960s, when substantial amounts of the drug were being taken for nonmedical reasons. Although still abused by some, dextroamphetamine is no longer the stimulant of choice for most psychomotor stimulant abusers.

(SEE ALSO: *Amphetamine Epidemics; Cocaine*)

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DIAGNOSIS OF DRUG ABUSE: DIAGNOSTIC CRITERIA Diagnosis is the process of identifying and labeling specific disease conditions. The signs and symptoms used to classify a sick person as having a disease are called diagnostic criteria. Diagnostic criteria and classification systems are useful for making clinical decisions, estimating disease prevalence, understanding the

causes of disease, and facilitating scientific communication.

Diagnostic classification provides the treating clinician with a basis for retrieving information about a patient's probable symptoms, the likely course of an illness, and the biological or psychological process that underlies the disorder. For example, the DIAGNOSTIC AND STATISTICAL MANUAL (DSM) of the American Psychiatric Association (1987) is a classification of mental disorders that provides the clinician with a systematic description of each disorder in terms of essential features, age of onset, probable course, predisposing factors, associated features and differential diagnosis. Mental health professionals can use this system to diagnose substance use disorders in terms of the following categories: acute INTOXICATION, ABUSE, DEPENDENCE, WITHDRAWAL, DELIRIUM, and other disorders. In contrast to screening, diagnosis typically involves a broader evaluation of signs, symptoms, and laboratory data as these relate to the patient's illness. The purpose of diagnosis is to provide the clinician with a logical basis for planning TREATMENT and estimating prognosis.

Another purpose of classification is the collection of statistical information on a national and international scale. The primary purpose of the INTERNATIONAL CLASSIFICATION OF DISEASES (ICD), for example, is the enumeration of morbidity and mortality data for public health planning (World Health Organization, 1992). In addition, a good classification will facilitate communication among scientists and provide the basic concepts needed for theory development. Both ICD and DSM have been used extensively to classify persons for scientific research. Classification provides a common frame of reference in communicating scientific findings.

Diagnosis also may serve a variety of administrative purposes. When a patient is suspected of having a substance use disorder, diagnostic procedures are needed to exclude "false positives" (i.e., people who appear to have the disorder but who really do not) and borderline cases. Insurance reimbursement for medical treatment increasingly demands that a formal diagnosis be confirmed according to standard procedures or criteria. The need for uniform reporting of statistical data, as well as the generation of prevalence estimates for epidemiological research, often requires a diagnostic classification of the patient.

CLASSIFICATION SYSTEMS

ALCOHOLISM and drug ADDICTION have been variously defined as medical diseases, mental disorders, social problems, and behavioral conditions. In some cases, they are considered the symptom of an underlying mental disorder (Babor, 1992). Some of these definitions permit the classification of alcoholism and drug dependence within standard nomenclatures such as the *International Classification of Diseases* and the *Diagnostic and Statistical Manual of Mental Disorders*. The recent revisions of both of these diagnostic systems has resulted in a high degree of compatibility between the classification criteria used in the United States and those used internationally. Both systems now diagnose dependence according to the elements first proposed by Edwards and Gross (1976). They also include a residual category (harmful alcohol use [ICD-10]; alcohol abuse [DSM-III-R]) that allows classification of psychological, social, and medical consequences directly related to substance use.

Some diagnostic classification systems are used primarily for epidemiological and clinical research. These include the Feighner Criteria (Feighner et al., 1972) and the Research Diagnostic Criteria (Robins, 1981). Other classifications are intended primarily for clinical care (DSM-III-R; see American Psychiatric Association, 1987) or statistical reporting (ICD-10; see World Health Organization, 1992).

HISTORY TAKING

Obtaining accurate information from patients with alcohol and drug problems is often difficult because of the stigma associated with substance abuse and the fear of legal consequences. At times they want help for the medical complications of substance use (such as injuries, or depression) but are ambivalent about giving up alcohol or drug use entirely. It is often the case that these patients are evasive and attempt to conceal or minimize the extent of their alcohol or drug use. Acquiring accurate information about the presence, severity, duration and effects of alcohol and drug use therefore requires a considerable amount of clinical skill.

The medical model for history taking is the most widely used approach to diagnostic evaluation. This consists of identifying the chief complaint, evaluating the present illness, reviewing past his-

tory, conducting a review of biological systems (e.g., gastrointestinal, cardiovascular), asking about family history of similar disorders, and discussing the patient's psychological and social functioning. A history of the present illness begins with questions on use of alcohol, drugs, and TOBACCO. The questions should cover PRESCRIPTION DRUGS as well as illicit drugs, with additional elaboration of the kind of drugs, the amount used, and the mode of administration (e.g., smoking, injection). Questions about alcohol use should refer specifically to the amount and frequency of use of major beverage types (wine, spirits, BEER). A thorough physical examination is important because each substance has specific pathological effects on certain organs and body systems. For example, alcohol affects the liver, stomach, and cardiovascular system. Drugs often produce abnormalities in "vital signs" such as temperature, pulse, and blood pressure. A mental status examination frequently gives evidence of substance use disorders because of poor personal hygiene, inappropriate affect (sad, euphoric, irritable, ANXIOUS), illogical or delusional thought processes, and memory problems. The physical examination can be supplemented by laboratory tests, which sometimes aid in early diagnosis before severe or irreversible damage has taken place. Laboratory tests are useful in two ways (1) alcohol and drugs can be measured directly in blood, urine, and exhaled air; (2) biochemical and psychological functions known to be affected by substance use can be assessed. Many drugs can be detected in the urine for twelve to forty-eight hours after their consumption. An estimate of BLOOD ALCOHOL CONCENTRATION (BAC) can be made directly by blood test or indirectly by means of a breath or saliva test. Elevated gamma glutamyl transpeptidase (GGTP), a liver enzyme, is a sensitive indicator of chronic, heavy alcohol intake.

In addition to the physical examination and laboratory tests, a variety of diagnostic interview procedures have been developed to provide objective, empirically based, reliable diagnoses of substance use disorders in various clinical populations. One type, exemplified by the DIAGNOSTIC INTERVIEW SCHEDULE (DIS; see Robins et al., 1981) and the Composite International Diagnostic Interview (CIDI; see Robins et al., 1988), is highly structured and requires a minimum of clinical judgment by the interviewer. These interviews provide information not only about substance use disorders, but also about physical conditions and psychiatric disorders that are commonly associated with substance abuse. A second type of diagnostic interview is exemplified by the STRUCTURED CLINICAL INTERVIEW for DSM-III-R (SCID), which is designed for use by mental health professionals (Spitzer et al., 1992). The SCID assesses thirty-three of the more commonly occurring psychiatric disorders described in DSM-III-R. Among these are depression, schizophrenia, and the substance use disorders. A similar clinical interview, which has been designed for international use, is the Schedules for Clinical Assessment in Neuropsychiatry (SCAN; see Wing et al., 1990). The SCID and SCAN interviews allow the experienced clinician to tailor questions to fit the patient's understanding, to ask additional questions that clarify ambiguities, to challenge inconsistencies, and to make clinical judgments about the seriousness of symptoms. They are both modeled on the standard medical history practiced by many mental health professionals. Questions about the chief complaint, past episodes of psychiatric disturbance, treatment history, and current functioning all contribute to a thorough and orderly psychiatric history that is extremely useful for diagnosing substance use disorders.

In recent years there has been interest in researching and developing a system of self-reporting to aid in the diagnosing of drug use severity. There has been resistance to this kind of diagnostic tool because of clinical suspicion that individuals with substance-use disorders often are not capable of reporting their symptoms accurately. The result of which is a reliance on clinicians or trained interviewers over self-reporting, paper/pencil measures to determine a patient's drug use severity. However, patient-reported data on outcomes and effectiveness of substance abuse treatments is becoming an increasing necessity. Additionally, according to a recent investigation of methodological studies, self-report measures appear to be neither inherently reliable nor unreliable. Certainly the information reported can be imprecise because of memory loss and under- or overreporting, among other variables. Also a variety of conditions can render self-report measurements susceptible to measurement error and systematic response bias but there is no empirical evidence to definitively show that self-reported data is more problematic than interviewer formats. Research has shown that format can create systematic bias but this can be accounted for by

combining the data from alternate forms (Heithoff & Wiseman, 1996).

DIAGNOSIS OF ABUSE AND HARMFUL USE

A major diagnostic category that has received increasing attention in research and clinical practice is substance *abuse* in contrast to *dependence*. This category permits the classification of maladaptive patterns of alcohol or drug use that do not meet criteria for dependence. The diagnosis of *abuse* is designed primarily for persons who have recently begun to experience ALCOHOL or drug problems, and for chronic users whose substance-related consequences develop in the absence of marked dependence symptoms. Examples of situations in which this category would be appropriate include (1) a pregnant woman who keeps drinking alcohol even though her physician has told her that it could be responsible for FETAL damage; (2) a college student whose weekend binges result in missed classes, poor grades, and alcohol-related traffic ACCIDENTS; (3) a middle-aged beer drinker regularly consuming a six-pack each day who develops high blood pressure and fatty liver in the absence of alcohol-dependence symptoms, and (4) an occasional MARIJUANA smoker who has an accidental injury while intoxicated.

In the latest version of the *Diagnostic and Statistical Manual* (DSM IV; see American Psychiatric Association, 1994), *substance abuse* is defined as a maladaptive pattern of alcohol or drug use leading to clinically significant impairment or distress, as manifested by one or more of the symptoms listed in Table 1. For comparative purposes, the table also lists the criteria for harmful use in ICD-10 and for alcohol abuse in DSM-III-R. To assure that the diagnosis is based on clinically meaningful symptoms rather than the results of an occasional excess, the duration criterion specifies how long the symptoms must be present to qualify for a diagnosis.

In ICD-10, the term *harmful use* refers to a pattern of using one or more PSYCHOACTIVE substances that causes damage to health. The damage may be (1) physical (physiological)—such as fatty liver, pancreatitis from alcohol, or hepatitis from needle-injected drugs; or (2) mental (psychological)—such as depression related to heavy drinking or drug use. Adverse social consequences often accompany substance use, but are not in themselves sufficient to result in a diagnosis of harmful use. The key issue in the definition of this term is the distinction between *perceptions* of adverse effects (e.g., wife complaining about husband's drinking) and *actual* health consequences (e.g., trauma due to accidents during drug intoxication). Since the purpose of ICD is to classify diseases, injuries, and causes of death, harmful use is defined as a pattern of use already causing damage to health.

Harmful patterns of use are often criticized by others and sometimes legally prohibited by governments. The fact that alcohol or drug intoxication is disapproved by another person or by the user's culture is not in itself evidence of harmful use—unless socially negative consequences have actually occurred at dosage levels that also result in psychological and physical consequences. This is the major difference that distinguishes ICD-10's harmful use from DSM-IV's substance abuse—the latter category includes social consequences in the diagnosis of abuse.

THE DEPENDENCE SYNDROME CONCEPT

The diagnosis of *substance use disorders* in ICD-10 and DSM-IV is based on the concept of a *dependence syndrome*, which is distinguished from disabilities caused by substance use (Edwards, Arif, & Hodgson, 1981). An important diagnostic issue is the extent to which dependence is sufficiently distinct from abuse or harmful use to be considered a separate condition. In DSM-IV, *substance abuse* is a residual category that allows the clinician to classify clinically meaningful aspects of a patient's behavior when that behavior is not clearly associated with a dependence syndrome. In ICD-10, *harmful substance use* implies identifiable substance-induced medical or psychiatric consequences that occur in the absence of a dependence syndrome. In both classification systems, dependence is conceived as an underlying condition that has much greater clinical significance because of its implications for understanding etiology, predicting course, and planning treatment. This will become clear in the following discussion of the assumptions behind the dependence-syndrome concept.

The dependence syndrome is seen as an interrelated cluster of cognitive, behavioral, and physiological symptoms. Table 2 summarizes the criteria used to diagnose dependence in ICD-10, DSM-

TABLE 1
Diagnostic Criteria for Harmful Use (ICD-10) and Substance Abuse (DSM-III-R, DSM-IV)

	ICD-10 Criteria for Harmful Use	*DSM-III-R Criteria for Abuse*	*DSM-IV Criteria for Abuse*
Symptom Criteria	Clear evidence that alcohol or drug use is responsible for causing actual psychological or physical harm to the user	A maladaptive pattern of alcohol or drug use indicated by at least one of the following: (1) continued use despite knowledge of having a persistent or recurrent social, occupational, psychological, or physical problem that is caused or exacerbated by substance use or (2) recurrent use in situations in which substance use is physically hazardous (e.g., driving while intoxicated)	A maladaptive pattern of alcohol or drug use indicated by at least one of the following: (1) failure to fulfill major role obligations at work, school, or home (e.g., neglect of children or household); (2) use in situations in which it is physically hazardous (e.g., driving an automobile); (3) recurrent substance-related legal problems (e.g., arrests for substance-related disorderly conduct); (4) continued substance use despite having recurrent social or interpersonal problems
Duration Criterion	The pattern of use has persisted for at least 1 month or has occurred repeatedly over the previous 12 months	Some symptoms of the disturbance have persisted for at least 1 month or have occurred repeatedly over a longer period of time	One or more symptoms has occurred during the same 12-month period

III-R, and DSM-IV. A diagnosis of dependence in all systems is made if three or more of the criteria have been experienced at some time in the previous twelve months.

The dependence syndrome may be present for a specific substance (e.g., tobacco, alcohol, or diazepam), for a class of substances (e.g., opioid drugs), or for a wider range of various substances. A diagnosis of *dependence* does not necessarily imply the presence of physical, psychological, or social consequences, although some form of harm is usually present. There are some differences among these classification systems, but the criteria are very similar, making it unlikely that a patient diagnosed in one system would be diagnosed differently in the other.

The syndrome concept implicit in the diagnosis of alcohol and drug dependence in ICD and DSM is a way of describing the nature and severity of addiction (Babor, 1992). Table 2 describes four dependence syndrome elements in relation to the criteria for DSM-III-R, DSM-IV, and ICD-10. The same elements apply to the diagnosis of dependence on all psychoactive substances, including alcohol, marijuana, opiates, cocaine, sedatives, phencyclidine, other hallucinogens, and tobacco. The elements represent biological, psychological (cognitive), and behavioral processes. This helps to explain the linkages and interrelationships that account for the coherence of signs and symptoms. The co-occurrence of signs and symptoms is the essential feature of a syndrome. If three or more criteria do occur repeatedly during the same period, it is likely that dependence is responsible for the amount, frequency, and pattern of the person's substance use.

Salience. Salience means that drinking or drug use is given a higher priority than other activities in spite of its negative consequences. This is reflected in the emergence of substance use as the

TABLE 2
ICD and DSM Diagnostic Criteria for Dependence (labeled according to diagnostic level—physiological, cognitive, and behavioral—and underlying dependence elements)

Dependence Element	*Diagnostic Level*	*ICD-10 Symptoms*	*DSM-III-R Symptoms*	*DSM-IV Symptoms*
Salience	Cognitive, behavioral	Progressive neglect of alternative activities in favor of substance use	Important social, occupational, or recreational activities given up	Important social, occupational, or recreational activities given up
	Behavioral	Persistence with substance use despite harmful consequences	Continued use despite social, psychological, or physical problems	Continued use despite psychological or physical problems
	Behavioral, physiological		Great amount of time devoted to substance use, intoxication, or withdrawal; failure to fulfill major role obligations	
Impaired control	Behavioral, cognitive	A strong desire or sense of compulsion to drink or use drugs	Persistent desire or one or more unsuccessful efforts to cut down or control substance use	
	Behavioral	Evidence of impaired capacity to control substance use in terms of its onset, termination, or levels of use	Substance often taken in larger amounts or over longer period than the person intended	Substance often taken in larger amounts or over a longer period than intended Any unsuccessful effort or a persistent desire to cut down or control substance use
Tolerance	Biological, behavioral	Increased doses of substance are required to achieve effects originally produced by lower doses	Marked tolerance; need for markedly increased amounts of substance to achieve intoxication or desired effect or markedly diminished effect with continued use of the same amount	Either (a) increased amounts needed to achieve desired effect; or (b) markedly diminished effect with continued use
Withdrawal and withdrawal relief	Behavioral, biological, cognitive	A physiological withdrawal state Use to relieve or avoid withdrawal symptoms and subjective awareness that this strategy is effective	Characteristic withdrawal symptoms Substance often taken to relieve or avoid withdrawal symptoms	Either (a) characteristic withdrawal syndrome for substance; or (b) the same substance taken to relieve or avoid symptoms

preferred activity from a set of available alternative activities. In addition, the individual does not respond well to the normal processes of social control. For example, when drinking to intoxication goes against the tacit social rules governing the time, place, or amount typically expected by the user's family or friends, this may indicate increased salience.

A characteristic of salience is that drinking or drug use persists in spite of its negative consequences. This implies that substance use has become the preferred activity in the person's life. One indication of this is the amount of time or effort devoted to obtaining, using or recovering from substance use. For example, people who spend a great deal of time at parties, bars, or business lunches give evidence of the increased salience of drinking over nondrinking activities.

Chronic drinking and drug intoxication interfere with the person's ability to conform to tacit social rules governing daily activities, such as keeping appointments, caring for children, or performing a job properly, that are typically expected by the person's reference group. Substance use also results in mental and medical consequences. Thus, a key aspect of the dependence syndrome is the persistence of substance use in spite of social, psychological, or physical harm, such as loss of employment, marital problems, depressive symptoms, accidents, and liver disease. This indicates that substance use is given a higher priority than other activities, in spite of its negative consequences.

One explanation for the salience of drug and alcohol-seeking behavior despite negative consequences is the relative reinforcement value of immediate and long-term consequences. For many alcoholics and drug abusers, the immediate positive reinforcing effects of the substance, such as euphoria or stimulation, far outweigh the delayed negative consequences, which may occur either infrequently or inconsistently.

Impaired Control. The main characteristic of impaired control is the lack of success in limiting the amount or frequency of substance use. For example, the alcoholic wants to stop drinking, but repeated attempts have been unsuccessful. Typically, rules and other stratagems are used to avoid alcohol entirely or to limit the frequency of drinking. Resumption of heavy drinking after receiving professional help for a drinking problem is evidence of lack of success. The symptom is considered present if the drinker has repeatedly failed to abstain or has only been able to control drinking with the help of treatment, mutual-help groups, or removal to a controlled environment (e.g., prison).

In addition to an inability to abstain, impaired control is also reflected in the failure to regulate the amount of alcohol or drug consumed on a given occasion. The cocaine addict vows to snort only a small amount but then continues until the entire supply is used up. For the alcoholic, impaired control includes inability to prevent spontaneous onset of drinking bouts as well as failure to stop drinking before intoxication. This behavior should be distinguished from situations in which the drinker's "control" over the onset or amount of drinking is regulated by social or cultural factors, such as occur during college beer parties or fiesta drinking occasions. One way to judge the degree of impaired control is to determine whether the drinker or drug user has made repeated attempts to limit the quantity of substance use by making rules or imposing limits on access to alcohol or drugs. The more these attempts have failed, the more the impaired control is present.

Tolerance. TOLERANCE is a decrease in response to a psychoactive substance that occurs with continued use. For example, increased doses of heroin are required to achieve effects originally produced by lower doses. Tolerance may be physical, behavioral, or psychological. Physical tolerance is a change in cellular functioning. The effects of a dependence-producing substance are reduced, even though the cells normally affected by the substance are subjected to the same concentration. A clear example is the finding that alcoholics can drink amounts of alcohol (e.g., a quart of vodka) that would be sufficient to incapacitate or kill nontolerant drinkers. Tolerance may also develop at the psychological and behavioral levels, independent of the biological adaptation that takes place. Psychological tolerance occurs when the marijuana smoker or heroin user no longer experiences a "high" after the initial dose of the substance. Behavioral tolerance is a change in the effect of a substance because the person has learned to compensate for the impairment caused by a substance. Some alcoholics, for example, can operate machinery at moderate doses of alcohol without impairment.

Withdrawal Signs and Symptoms. A withdrawal state is a group of symptoms occurring after

TABLE 3
Withdrawal Symptoms Associated with Different Psychoactive Substances

	Alcohol	*Amphetamine*	*Caffeine*	*Cocaine*	*Opioids*	*Nicotine*
Craving					X	X
Tremor	X					
Sweating, fever	X				X	
Nausea or vomiting	X				X	
Malaise, fatigue	X	X		X		
Hyperactivity, restlessness	X	X	X	X		X
Headache	X					
Insomnia	X	X	X		X	
Hallucinations	X					
Convulsions	X					
Delirium	X					
Irritability	X	X		X		X
Anxiety	X		X	X		X
Depression	X			X		
Difficulty concentrating						X
Gastrointestinal disturbance			X			
Increased appetite						X
Diarrhea					X	

cessation of substance use. It usually occurs after repeated, and usually prolonged drinking or drug use. Onset and course of the withdrawal symptoms are related to type of substance and dose being used immediately prior to abstinence. Table 3 lists some common withdrawal symptoms associated with different psychoactive substances. Some drugs, such as *Cannabis* (MARIJUANA) and HALLUCINOGENS do not typically produce a withdrawal syndrome after cessation of use.

Alcohol withdrawal symptoms follow the cessation or reduction of prolonged heavy drinking within hours. These include tremors, hyperactive reflexes, rapid heartbeat, hypertension, general malaise, nausea, and vomiting. Seizures and convulsions may occur, particularly in people with a preexisting seizure disorder. Patients may have HALLUCINATIONS, illusions, or vivid nightmares. Sleep is usually disturbed. In addition to physical withdrawal symptoms, anxiety and depression are also common. Some chronic drinkers never have a long enough period of abstinence to permit withdrawal to occur.

The use of a substance with the intention of relieving withdrawal symptoms and with an awareness that this strategy is effective are cardinal symptoms of dependence. Morning drinking to relieve nausea or the "shakes" is one of the most common manifestations of physical dependence in alcoholics.

Other Features of Dependence. To be labeled dependence, symptoms must have persisted for at least one month or must have occurred repeatedly (two or more times) over a longer period of time. The patient does not need to be using the substance continually to have recurrent or persistent problems. Some symptoms (e.g., the desire to cut down) may occur repeatedly whether the person is using the substance or not.

Many patients with a history of dependence experience rapid reinstatement of the syndrome following resumption of substance use after a period of abstinence. Rapid reinstatement is a powerful diagnostic indicator of dependence. It points to the impairment of control over substance use, the rapid development of tolerance, and (frequently) physical withdrawal symptoms.

Patients who receive OPIATES or other drugs for PAIN relief following surgery (or for a malignant disease like cancer) sometimes show signs of a

withdrawal state when these drugs are ended. The great majority have no desire to continue taking such drugs and therefore do not fulfill the criteria for dependence. The presence of a physical withdrawal syndrome does not necessarily indicate dependence but rather a state of neuroadaptation to the drug that was being administered.

It is commonly assumed that severe dependence is not reversible—an assumption indicated by the rapid reinstatement of dependence symptoms when drinking or drug use is resumed after a period of detoxification.

(SEE ALSO: *Addiction: Concepts and Definitions; Alcoholism: Origin of the Term; Causes of Substance Abuse; Disease Concept of Alcoholism and Drug Abuse; Tolerance and Physical Dependence; Wikler's Pharmacologic Theory of Drug Addiction*)

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DIAGNOSTIC AND STATISTICAL MANUAL (DSM) The *Diagnostic and Statistical Manual of Mental Disorders* is the most widely accepted diagnostic system in the United States. First published by the American Psychaiatric Association (APA) in 1952, the DSM is used by medical professionals, insurance companies, and the court system to diagnose and define mental illnesses and disorders, including substance abuse and dependence. In fact, the diagnosis code assigned to a case often determines insurance reimbursement for treatment. The book is also an important indicator of societal mores: until 1973 homosexuality was defined as a mental disorder.

The first tabulation of mental illness in the United States appeared in the 1840 census, when the category "idiots" and the category "insane" were first counted. By the 1880 census, seven types of mental illness were recognized, including epi-

lepsy. In 1917 the American Medico-Psychological Association (now the APA), in conjunction with the National Commission on Mental Hygiene, further enlarged its categories of mental illness. This broader list, while certainly of greater clinical use, was still chiefly designed to count the numbers and types of patients in mental hospitals. Several years after this tabulation, the newly renamed APA released a compendium of nationally recognized psychiatric terms—most of which applied to psychotic disorders and severe neurological impairments—that would become part of the American Medical Association's standard classified nomenclature of disease.

After the end of World War II, the Veterans Administration (VA) added many more diagnoses to the APA inventory, incorporating the various psychological manifestations exhibited by servicemen. This expanded compilation proved to be influential, for shortly after its publication the World Health Organization (WHO) published the sixth edition of its *International Classification of Diseases* (ICD), which for the first time included information on mental disorders, much of it based on by the VA classifications.

The first edition of the DSM (DSM-I), published in 1952, was little more than a pamphlet. Its importance, however, lay in its description and definition of the approximately 100 diagnostic categories then recognized by clinicians. DSM-I, like its successor, DSM-II, was heavily influenced by the seventh and eighth editions of ICD. In fact, until the publication of of DSM-III, the American system for classifying psychiatric disorders was virtually identical to the ICD.

During the 1970s, however, researchers affiliated with the Washington University School of Medicine (Feighner et al., 1972) developed the "research diagnostic" approach to psychiatric diagnosis, which emphasized clearly formulated and observable signs and symptoms that could be used for both research and clinical practice. DSM-III, published in 1980, incorporated this approach, adding clear diagnostic standards and objective descriptions of symptoms and behaviors.

DSM-III also introduced a multiaxial system for diagnostic evaluation to ensure that all relevant clinical information was considered. Axis I describes syndromes, such as major DEPRESSION, SCHIZOPHRENIA, and substance use disorders. Axis II covers childhood and personality disorders that often persist into adult life. Axis III refers to physical disorders or conditions that are potentially relevant to the understanding or management of the patient. Axis IV rates the severity of psychosocial stressors that have occurred in the year preceding the current evaluation and that may have contributed to the patient's symptoms. Axis V is a global assessment of psychological, social and occupational functioning, which should be taken into account in treatment planning.

For the first time DSM-III listed substance use disorders as a separate diagnosis category, distinguishing them from personality disorders, which they had previously been considered. In addition, the term *dependence* replaced the more generic *alcoholism* or *addiction*, and was distinguished from *abuse* by the presence of the symptoms of TOLERANCE or WITHDRAWAL. Alcohol and drug abuse were assigned to separate subcategories, permitting a greater differentiation and range of severity for each.

Another important change to the substance use disorders section in DSM-III (Rounsaville, Spitzer, & Williams, 1986) was the adoption of a new dependence syndrome concept (Edwards, Arif, & Hodgson, 1981), in which dependence was defined as an interrelated cluster of psychological symptoms: a strong desire or CRAVING for the substance; physiological signs, especially tolerance and withdrawal; and behavioral indicators, particularly using the substance to relieve withdrawal discomfort. Significantly, the medical and social consequences of both acute intoxication and chronic substance use, such as ACCIDENTS and liver damage are not among the primary diagnostic criteria of dependence. They do, however, play a prominent role in defining the *substance abuse* category.

After the publication of a revised third edition in 1987 (DSM-III-R), a fourth edition (DSM-IV) was published in 1994. This version contained further changes in the diagnosis of substance-related disorders that were designed to assure compatibility between DSM and ICD. Both publications now define substance dependence as a maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by three or more of the following symptoms occurring in the same twelve-month period:

Tolerance—the need for markedly increased amounts of the substance to achieve intoxication or the desired effect

Withdrawal—behavioral changes that occur when blood or tissue levels of the substance decline after a period of prolonged or heavy use; often accompanied by use of the substance to relieve withdrawal symptoms.

Increased use—taking the substance in larger amounts or over a longer period.

Unsuccessful attempts to cut down or control substance use.

Much time spent in activities related to procuring or using the substance.

Ignoring or reducing important social, occupational, or recreational activities because of substance use.

Continued use despite physical or psychological problems caused by the substance.

Patients can become dependent on any of the following: ALCOHOL, TOBACCO, SEDATIVES–HYPNOTICS–ANXIOLYTICS, CANNABIS (MARIJUANA), STIMULANTS, OPIOIDS, COCAINE, HALLUCINOGENS, PCP (PHENCYCLIDINE), or a combination of drugs, which is known as POLYSUBSTANCE ABUSE. The most important factor in determining dependence, according to the DSM-IV, is not simply abusing alcohol or drugs, but the patient's refusal to stop using the substance(s) despite recognizing the serious problems this causes.

(SEE ALSO: *Addiction: Concepts and Definitions; Alcoholism: Origin of the Term; Disease Concept of Alcoholism and Drug Abuse*)

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DIAGNOSTIC INTERVIEW SCHEDULE (DIS) Developed in the late 1970s for use in large-scale studies of the prevalence of mental disorders in the U.S. population (Regier et al., 1984), the Diagnostic Interview Schedule (DIS) is a highly structured psychiatric interview that carefully specifies the questions that the interviewer must ask to make a DIAGNOSIS. Another version is the DISC, or Diagnostic Interview Schedule for Children. Unlike the DIS, this version allows the re-ordering of questions or sections. Because the DIS requires a minimum of clinical judgment, it can be administered by nonprofessional or nonclinician interviewers who have received a week of intensive training. In addition to alcohol and other substance-use disorders, the DIS provides diagnostic information about DEPRESSION, SCHIZOPHRENIA, and ANXIETY disorders; eating disorders; ANTI-SOCIAL PERSONALITY; and a variety of other psychiatric conditions. The DIS has been the subject of a number of validation studies showing that nonclinician interviewers diagnose patients as accurately as trained clinicians using criteria from DSM-III (*DIAGNOSTIC AND STATISTICAL MANUAL of Mental Disorders*, third edition). With the American Psychiatric Association's publication of the revised versions of DSM, major changes were made to the DIS as well.

In June 2000 a study of 349 individuals who were given the DIS was published, then examined by psychiatrists using the Schedules for Clinical Assessment in Neuropsychiatry (SCAN). The DIS missed many cases of major depressive disorders (as determined by SCAN), but there was correlation in the symptom groups. The researchers concluded that DIS may be too conservative with risk factors.

The DIS was first used in The Epidemiologic Catchment Area (ECA) study, which was a survey of mental disorders in the United States. This survey's results led to worldwide testing, which in turn led to comparative analyses among the nations.

The DIS also has been widely used in research on substance-use disorders (Helzer & Canino, 1992), in part because it can be administered by nonclinician interviewers in population surveys. Interviewers read questions aloud to the subject exactly as they are written in the interview booklet. No deviation from the written format is allowed, except to repeat questions that may have been misunderstood. A set of standard probes is used to determine whether a given symptom was caused by the effects of physical illness. The interviewer also asks for the age of onset and the recency of most symptoms.

A series of thirty questions constitutes the ALCOHOL DEPENDENCE/abuse section of the DIS. The section begins with questions about alcohol consumption and intoxication (e.g., "Have you ever gone on binges or benders where you kept drinking for a couple of days or more without sobering up?"). Additional questions are asked to diagnose the symptoms of dependence (e.g., "Did you ever get tolerant to alcohol, that is, you needed to drink a lot more in order to get an effect, or found that you could no longer get high on the amount you used to drink?"). A third type of question pertains to the symptoms of alcohol abuse (e.g., "Have you ever had trouble driving because of drinking—like having an ACCIDENT or being arrested for drunk driving?").

The drug dependence section of the DIS (version III-B) consists of twenty-four questions that conform to the DSM-III-R criteria for drug use disorders. This section begins by asking if the patient has used any of the following types of drugs "to get high or for other mental effects": MARIJUANA, STIMULANTS (e.g., AMPHETAMINES), SEDATIVES (e.g., BARBITURATES), prescribed drugs (e.g., TRANQUILIZERS), COCAINE, HEROIN, other OPIATES, PSYCHEDELICS, PCP, INHALANTS, and other drugs not previously specified. If the person has used any of these substances more than five times, additional questions are asked to evaluate the mode of ingestion for each drug (e.g., by mouth, smoking, snorting, or injecting).

The remaining questions ask about DSM-III-R symptoms of dependence and abuse. For example, patients are asked if they have had difficulty abstaining from drugs ("Have you ever tried to cut down on any of these drugs but found you couldn't?"); experienced WITHDRAWAL symptoms ("Has stopping or cutting down on any of these drugs made you sick?"); or experienced other physical complications ("Did you have any health problems like an accidental OVERDOSE, a persistent cough, a seizure [fit], an infection, a cut, sprain, burn, or other injury as a result of taking any of these drugs?"). The DIS can be scored manually or by computer to obtain specific drug and alcohol diagnoses in DSM-III-R.

In 1994, the American Psychiatric Association released the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV). This version is applicable to both children and adults, which has made it an integral part of school and child psychology, especially when dealing with attention deficit hyperactivity disorder (ADHD). The DSM-IV functions as a way of organizing and recognizing cognitive and personality disorders, as well as addictive and disruptive behaviors. The DSM-IV was also used in the late 1990s (in conjunction with part of the DIS) to help determine substance abuse treatment needs for prisoners and to screen veterans for post-traumatic stress disorder (PTSD).

(SEE ALSO: *Addiction: Concepts and Definitions; Diagnosis of Drug Abuse; Disease Concept of Alcoholism and Drug Abuse*)

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REVISED BY REBECCA MARLOW-FERGUSON

DIAZEPAM *See* Benzodiazepines

DIET PILL *See* Amphetamine; Anoretic

DIHYDROMORPHINE Dihydromorphine is a semisynthetic OPIOID ANALGESIC (painkiller), derived from MORPHINE. Structurally, it is very similar to morphine—the only difference being the reduction of the double bond between positions 7 and 8 in morphine to a single bond. Although slightly more potent than morphine in relieving PAIN, it is not widely used clinically. At standard analgesic doses, it has a side-effect profile very similar to that of morphine. These include constipation and respiratory depression. Chronic use will produce TOLERANCE AND PHYSICAL DEPENDENCE.

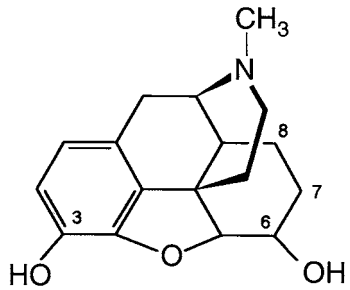

Figure 1
Dihydromorphine

(SEE ALSO: *Addiction: Concepts and Definitions; Opiates/Opioids; Opioids: Complications and Withdrawal*)

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GAVRIL W. PASTERNAK

DILAUDID *See* Hydromorphone

DIMETHYLTRYPTAMINE (DMT) This drug is a member of the HALLUCINOGENIC substances known as indoleamines. These are compounds that are structurally similar to the neurotransmitter SEROTONIN. Although found in certain plants and, according to some evidence, can be formed in the brain, DMT is synthesized for use. Its effects are similar to those produced by LYSERGIC ACID DIETHYLAMIDE (LSD), but unlike LSD, DMT is inactive after oral administration. It must be injected, sniffed, or smoked.

DMT has a rapid onset, usually within one minute, but the effects last for a much shorter period than those produced by LSD—with the user feeling "normal" within thirty to sixty minutes. This is because DMT is very rapidly destroyed by the enzyme monoamine oxidase, which metabolizes structurally related compounds, such as serotonin.

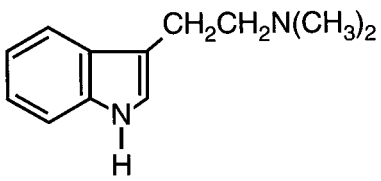

Figure 1
DMT

The dose amount of DMT is critical, since larger doses produce slightly longer, much more intense, and sometimes very uncomfortable "trips" than do lower doses. The sudden and rapid onset of a period of altered perceptions that soon terminates is also disconcerting to some users. DMT was known briefly as the "businessman's LSD"—one could have a PSYCHEDELIC experience during the lunch hour and be back at work in the afternoon. It has, however, in fact never been a widely available, steadily obtainable, or popular drug on the street.

(SEE ALSO: *DOM; MDMA*)

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DIS *See* Diagnostic Interview Schedule

DISEASE CONCEPT OF ALCOHOLISM AND DRUG ABUSE Throughout most of recorded history, excessive use of ALCOHOL was viewed as a willful act leading to intoxication and other sinful behaviors. The Bible warns against drunkenness; Islam bans alcohol use entirely. Since the early nineteenth century, the moral perspective has competed with a conceptualization of excessive use of alcohol as a disease or disorder, not necessarily a moral failing. The disease (or disorder) concept has, in turn, been evolving with considerable controversy since then, and has itself been challenged by other conceptual models. Because this article is concerned primarily with the disease concept, the other models will be mentioned only briefly.

Among the first to propose that excessive alcohol use might be a disorder, rather than willful or sinful behavior, were the physicians Benjamin Rush, in the United States, and Thomas Trotter, in Great Britain. Both Rush and Trotter believed that some individuals developed a pernicious "habit" of drinking and that it was necessary to undo the habit to restore those individuals to health. Words such as *habit* and *disease* were used to convey interwoven notions. Trotter saw "the habit of drunkenness" as "a disease of the will," while Rush saw drunkenness as a disease in which alcohol was the causal agent, loss of control over drinking behavior the characteristic symptom, and total abstinence the only effective cure. In 1849, a Swedish physician, Magnus Huss, introduced the term *alcoholism* ["alcoholismus"] to designate not only the disorder of excessive use but an entire syndrome, including the multiple somatic consequences of excessive use.

Late-nineteenth-century physicians, although not the first to see habitual use of other drugs (such as OPIATES, TOBACCO, COFFEE) as disorders, are credited with stressing the idea that each was but a subtype of a more *generic disorder of inebriety*. However, they also minimized Trotter's and Rush's notions of learned behavior as a central feature of a generic disorder of inebriety and emphasized instead the idea of a disorder rooted in acquired or inherited biological malfunction or VULNERABILITY. This more biologically based view of inebriety was used in Britain and the United States by advocates of publicly funded treatment facilities—inebriate asylums. Many temperance leaders also supported the establishment of treatment facilities. However, while physicians advocated treatment, temperance leaders, still convinced that alcohol itself was the root of the problem, pushed for its control and, eventually, for its prohibition.

In the United States, the ratification in 1920 of the Eighteenth Amendment, which prohibited the production, sale, and distribution of alcohol, temporarily dampened scientific inquiry into the nature of alcoholism. But concern about the problematic and excessive use of other drugs, such as OPIOIDS, COCAINE, and BARBITURATES, continued to stimulate writings both in the United States and

abroad. Was excessive drug use a disease, a moral failure, or something else—perhaps something in between?

By the mid-twentieth century, the rise of ALCOHOLICS ANONYMOUS (AA), the publications of E. M. Jellinek, and the establishment of the Yale Center for Alcohol Studies revived interest in exploring the nature of ALCOHOLISM. In the early 1960s, the idea reemerged that, for certain "vulnerable" people, alcohol use leads to physical addiction—a true disease.

EARLY MODELS OF THE DISEASE CONCEPT

Central to the disease concept of alcoholism put forward by Jellinek were the roles of TOLERANCE AND PHYSICAL DEPENDENCE, usually considered hallmarks of ADDICTION. *Tolerance* indicates that increased doses of a drug are required to produce effects previously attained at lower doses. *Physical dependence* refers to the occurrence of WITHDRAWAL symptoms following cessation of alcohol or other drug use. Although Jellinek recognized that alcohol problems could occur without alcohol addiction, addiction to alcohol moved to the center of scientific focus.

Despite being couched in the language of science, the reemergence of the disease concept of alcoholism was not a result of new scientific findings. Jellinek believed it was necessary to see alcoholism as a disease in order to increase the availability of services for alcoholics within established medical facilities. He also recognized that efforts to prevent alcoholism would still have to address the complex cultural, demographic, political and economic issues contributing to the problem. Although he sometimes appeared to take a broad view of the disease concept of alcoholism, he reserved the disease category for those individuals manifesting tolerance, withdrawal symptoms, and either "loss of control" or "inability to abstain" from alcohol. These individuals could not drink in moderation; with continued drinking, their disease was progressive. Others who drank merely in response to psychological stress ("alpha alcoholism") and those who sustained toxic consequences from alcohol but were not physically dependent ("beta alcoholism") did not qualify for his more explicit and restrictive definition of disease. Jellinek's view of alcoholism as a progressive disease is sometimes referred to as the "classic" disease model to distinguish it from later perspectives of a disorder or syndrome more powerfully influenced by learning and social factors.

Alcohol researcher and theorist Thomas Babor has pointed out that when definitions specify alcohol addiction or dependence as a disease entity, it can be argued more convincingly that "dependence is an organically based entity which produces a characteristic set of signs and symptoms . . . and increases the probability of repetitive drinking behavior."

The American Psychiatric Association included alcoholism in the first edition (1952) of the DIAGNOSTIC AND STATISTICAL MANUAL *of Mental Disorders*. In the second edition (*DSM-II*), published in 1968, the group followed a precedent set by the World Health Organization's INTERNATIONAL CLASSIFICATION OF DISEASES (*ICD-8*) and included three subcategories of alcohol-related disorders: alcohol addiction, episodic excessive drinking, and habitual excessive drinking. Both of these publications included alcoholism among the personality disorders and certain other nonpsychotic disorders, implying that the alcohol use was either secondary to an underlying personality problem or a response to extreme internal distress. This view of excessive drug use as a symptom of some other psychiatric disorder is sometimes referred to as the symptomatic model. According to this concept, drug or alcohol dependence is not really a disorder in and of itself.

Meanwhile, from the late 1950s and throughout the 1960s, the Expert Committee on Addiction-Producing Drugs of the WORLD HEALTH ORGANIZATION (WHO) continued to formulate and refine definitions of addiction and HABITUATION that could facilitate WHO's responsibility (required by international treaties) for control of NARCOTICS, cocaine, and CANNABIS. In the 1950s, the presence of physical dependence was emphasized in the definition of drug dependence, and the WHO Expert Committee was still concerned with differentiating between psychic dependence and physical dependence. At one level, the concept of psychic dependence was compatible with the psychodynamic view that these disorders were a response to psychic distress (such as negative mood states). According to the psychodynamic model, excessive alcohol or drug consumption was merely a response to underlying psychopathology. This model was also consis-

tent with Jellinek's view of one of the "species" of alcoholism, in which individuals drink to relieve emotional pain (alpha alcoholism). In 1969, the committee abandoned the effort to differentiate *habits* from *addictions* and adopted terminology first proposed by Nathan Eddy and colleagues in 1965, in which the term *drug dependence* designates "those syndromes in which drugs come to control behavior." The committee recognized that dependencies on different classes of drugs (such as alcohol, opiates, cocaine) can differ significantly and that withdrawal symptoms are not always present or necessary aspects of dependence (see Table 1).

In 1972, alcoholism was included in a listing of diagnostic criteria for use in psychiatric research published by Feighner and coworkers. The defining criteria for alcoholism included withdrawal symptoms, loss of control, severe medical consequences, and social problems. In the same year the NATIONAL COUNCIL ON ALCOHOLISM also outlined criteria for diagnosing alcoholism, which emphasized tolerance and physical dependence and incorporated certain concepts developed by ALCOHOLICS ANONYMOUS. This definition, and one issued jointly with the American Medical Society on Alcoholism in 1976 (see Table 1), represented an attempt to emphasize the seriousness of the disorder, the experience of clinicians and of recovering alcoholics, and the view that alcoholism is a primary or independent disorder, not merely a manifestation of an underlying personality problem. These statements come close to being current definitions of the classic disease model.

PROBLEM DRINKING AS A DISTINCT DIMENSION

The importance of what can now be called the classic "disease model" of alcoholism as a primary focus for health programs was challenged in 1977 by a report of a WHO Expert Committee on alcohol-related disabilities. This report stressed that not everyone who develops a disability related to alcohol use exhibits alcohol dependence or addiction, nor would such an individual necessarily develop a dependence in the future. The report asserted that some alcohol-related disabilities represent a dimension of *problem drinking* distinct from the disease of alcoholism or *alcohol dependence syndrome*. This perspective provided support for policies aimed at reducing overall alcohol consumption, not just at promoting abstinence among vulnerable individuals. The report described the alcohol dependence syndrome itself as a learned phenomenon, not a disease state, which is either present or absent, but "a condition which exists in degrees of severity." It is important to recognize that this syndrome perspective does not take a position on whether alcoholism should be considered a disease.

TABLE 1
Some Recent Attempts to Define Alcoholism and/or Drug Dependence

Drug dependence. A state, psychic and sometimes also physical, resulting from the interaction between a living organism and a drug, characterized by behavioural and other responses that always include a compulsion to take the drug on a continuous or periodic basis in order to experience its psychic effects, and sometimes to avoid the discomfort of its absence. Tolerance may or may not be present. A person may be dependent on more than one drug. (*World Health Organization Technical Report Series*, 1969, no. 407, p. 6.) This definition was reaffirmed in the WHO Expert Committee on Drug Dependence Nineteenth Report, *World Health Organization Technical Report Series*, 1973, no. 526, p. 16.

Alcoholism is a chronic, progressive, and potentially fatal disease. It is characterized by tolerance and physical dependency or pathologic organ changes or both, all of which are the direct or indirect consequences of the alcohol ingested. (National Council on Alcoholism/American Medical Society on Alcoholism, 1976.) (See Flavin & Morse, 1991.)

The 1976 definition was revised and broadened in 1991 to include the concept of *denial:*

Alcoholism is a primary, chronic disease with genetic, psychosocial, and environmental factors influencing its development and manifestations. The disease is often progressive and fatal. It is characterized by continuous or periodic impaired control over drinking, preoccupation with the drug alcohol, use of alcohol despite adverse consequences, and distortions in thinking, most notably denial. (National Council on Alcoholism and Drug Dependence/American Medical Society on Alcoholism, 1976) (See Flavin & Morse, 1991.)

The concept of dependence as a syndrome is quite similar to that put forward in 1965 by drug-abuse researcher Jerome Jaffe, who viewed addiction as standing at one end of a continuum of involvement in drug use: "In most instances it will not be possible to state with precision at what point [along the continuum] compulsive use should be considered addiction," Jaffe observed. He emphasized that "the term addiction cannot be used interchangeably with physical dependence. It is possible to be physically dependent on drugs without being addicted and . . . to be addicted without being physically dependent." In this view, the behavioral disorder, not physical dependence, is the syndrome. Jaffe defined addiction as "*a behavioral pattern of drug use, characterized by overwhelming involvement with the use of a drug (compulsive use), the securing of its supply, and a high tendency to relapse after withdrawal.*" This proposed generic notion of dependence is applicable to STIMULANTS and HALLUCINOGENS (for which physical dependence is not a significant factor), as well as to alcohol, opiates, and SEDATIVE-HYPNOTIC drugs (for which physical dependence is a factor). The *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, revised (DSM-III-R), published by the American Psychiatric Association more than twenty years later, in 1987, also used such a generic definition.

FROM PSYCHIC AND PHYSICAL DEPENDENCE TO DEPENDENCE SYNDROME

The changing perspectives on the general concept of drug dependence, given momentum by the 1977 WHO report on alcohol and by other research, were ultimately reflected in changes in the definitions and other positions of the World Health Organization and in its 1980 *International Classification of Diseases*, 9th edition (ICD-9). With its publication, the concept of an alcohol dependence syndrome formally emerged at an international level. The ICD-9 concept of dependence was based on a 1976 proposal by researchers Griffith Edwards and Milton Gross, who defined seven characteristics of the alcohol dependence syndrome and proposed that there are certain implicit assumptions to the syndrome: First, it is a symptom complex involving both biological processes and learning. Second, it should be defined along a continuum of severity, rather than as a discrete category. Third, dependence should be differentiated from alcohol-related disabilities. Both dependence and disabilities exist in degrees, rather than on an all-or-none basis. There is some evidence that people with more severe degrees of alcohol dependence who seek treatment have a different clinical course from those with less severe dependence.

By the late 1970s, the American Psychiatric Association's *Diagnostic and Statistical Manual*, 3rd edition (DSM-III), moved away from more descriptive and psychodynamic orientation toward a nomenclature in which specific diagnostic criteria were laid out for specific syndromes. In the case of alcohol and drug dependence, the original drafts of DSM-III considered inclusion of a dependence syndrome that varied in degree of severity and in which tolerance and physical dependence were important, but not essential, criteria for diagnosis. At the last moment, however, it was decided that tolerance and physical dependence were both necessary and sufficient for a diagnosis of drug dependence; the presence of other criteria listed were by themselves insufficient without tolerance and physical dependence. Nevertheless, by distinguishing drug (or alcohol) *dependence* from drug (or alcohol) *abuse*, DSM-III recognized the two-dimensional conceptualization previously put forth in the WHO report of 1977 and in ICD-9.

In 1980, during the short interval between the publication of DSM-III and the beginning of work on DSM-III-R, a WHO working group met to further refine terminology. One result of the meeting was the publication of a WHO memorandum on nomenclature and classification of drug- and alcohol-related problems that endorsed the concept that drug dependence is a syndrome that exists in degrees and that can be inferred from the way in which drug use takes priority over a drug user's once-held VALUES. The criteria for making this inference included many of those mentioned by Edwards and Gross in their 1976 paper and some that had been developed for DSM-III. The WHO memorandum, while recognizing the importance of tolerance and physical dependence, did not view these phenomena as always essential and required. It endorsed again the two-dimensional perspective—not all drug or alcohol problems are manifestations of dependence; and harmful or hazardous use can occur independently of the decreased flexibility and constricted choice that are the hallmarks of the

dependence syndrome. This perspective was underscored by pointing out that the presence of physical dependence per se (as in the case of patients taking drugs for pain) was not in itself sufficient for the diagnosis of dependence. The memorandum also presented a model of dependence emphasizing that the dependence phenomenon is not a property of the individual but resides in the relationships among the elements in the model—social, psychological, and biological. This view has been called the *biopsychosocial model*.

CRITERIA FOR DIAGNOSIS OF A GENERIC DEPENDENCE DISORDER

The American Psychiatric Association's DSM-III-R, published in 1987, built on both DSM-III and the WHO memorandum. It presented nine criteria for diagnosing a *generic dependence syndrome*, applied to a wide variety of drugs. The user must have experienced at least three criteria in order for the practitioner to consider any degree of dependence to be present. Neither tolerance nor physical dependence was a required criterion. The presence of more than three criteria would indicate a more severe degree of dependence. *Drug abuse* was a residual category used for designating drug-related problems when dependence was not present.

The DSM-III-R conceptualization of dependence was controversial. Because for many years physical dependence and tolerance had been considered evidence of "true disease," many clinicians believed that changing these criteria from the necessary and required status they had had in DSM-III was a mistake that erroneously broadened the category of drug dependence. Much of the focus in the development of DSM-IV, published in 1994, was on how to restore the primacy of these phenomena in the diagnosis of drug and alcohol dependence. DSM-IV defines seven generic criteria for alcohol and other drug dependence. Three are required for a diagnosis of alcohol or other drug dependence. Although tolerance and withdrawal are listed first, they are not required—but the clinician must specify whether either is present.

Despite these concerns, there was little argument about the importance of psychological and sociological factors in the development and perpetuation of the syndrome—that is, there was still consensus about the biopsychosocial model.

At the same time, at the international level, the framers of ICD-10 continued the evolution begun in ICD-9 and adhered closely to the concepts of dependence outlined in the 1977 WHO report and 1981 WHO memorandum. Published in 1992, ICD-10 includes a generic model of drug dependence with similar criteria for alcohol, tobacco, opioids, and other drugs that affect the brain. Like DSM-IV, ICD-10 presents a number of criteria (six) for determining the presence of the alcohol (or drug) *dependence* syndrome; at least three of these must be present for the clinician to judge that the syndrome exists to some degree.

ICD-10 does not include a diagnostic category of alcohol or drug *abuse* but instead includes a category of *harmful use*—a pattern of use that is causing damage to mental or physical health. Unlike DSM-IV, which defines drug or alcohol (substance) abuse as "a maladaptive pattern of use" causing significant impairment or distress and interpersonal, family, and legal problems (e.g., arrests), ICD-10 does not consider such patterns of use and consequences necessarily to be evidence of harmful use.

ICD-10 and DSM-IV share important characteristics that represent a further evolution in understanding drug and alcohol dependence syndromes. In contrast to some disease-oriented defintions that see alcoholism as uniformly progressive, in ICD-10 and DSM-IV the course of the disorder is not one of uniform progression or predictable cure, but there are a variety of significant states of remission. For example, DSM-IV distinguishes early remission (within the first 12 months) from sustained remission (at least 12 months); within each of these it differentiates full remission from partial remission (i.e., all criteria for dependence have not been met, although at least one has been met intermittently or continuously). DSM-IV also recognizes the circumstances supporting remission and allows for distinctions such as remission while the user is in a controlled environment (where substances are highly restricted) or remission from drug of dependence when the user is maintained on a similar agonist. The categorization of states of remission (abstinence) in ICD-10 is somewhat similar, although the distinction between early and sustained remission is not made.

CHALLENGES TO THE DISEASE CONCEPT

The classic disease model of alcoholism and drug dependence has served as a challenge to some behavioral researchers and social scientists; they have raised a number of questions about biologically based theories of such behaviors. Critics of the disease concept point to studies showing that some former alcoholics could apparently return to normal drinking. Such findings challenged the concept of alcoholism as a progressive disease. The concept of inevitable "loss of control" over drinking was also challenged by Merry's study (1966) in which alcoholics were given drinks containing either vodka or a placebo (no alcohol) on alternate days and reported having no more desire to drink after consuming the vodka than after the placebo. The results suggested that if "loss of control" did occur in alcoholics, it was not triggered as a biological response to alcohol but rather as a learned response with associated EXPECTANCIES concerning drinking behavior. Researchers Nancy Mello and Jack Mendelson also reported, in 1971, that alcoholics did not manifest "loss of control" in their drinking behavior and did not drink to avoid withdrawal symptoms. The work of Mello and Mendelson and of other researchers led to the conclusion that drinking behavior could be shaped like any other operant in a behavioral paradigm. Other researchers challenged the notion of alcoholism as a distinct entity (with clear differentiations between alcoholics and nonalcoholics), as well as the concepts of inevitable progression to loss of control and of alcoholism as a permanent and irreversible condition precluding the possibility of moderate drinking. (For these and other references, see Meyer, 1992.)

These findings by behavioral researchers in the laboratory had counterparts in large surveys of drinking practices conducted by the RAND Corporation. Evidence in the general population indicated that some alcoholics might be able to drink moderately without relapsing to excessive drinking.

These and other such challenges to the disease concept of alcoholism sharpened the debate and clarified the construct. Efforts to replicate some of these earlier studies sometimes led to conflicting results, calling into question the conclusions they had drawn or leading to refinements. RAND Corporation found at later follow-up that severely dependent alcoholics had to remain abstinent in order to maintain improvement. Several studies appeared to confirm that severely dependent alcoholics might be different from those who were less dependent. Some researchers, such as Hodgson, reported that small doses of alcohol had a "priming" effect (i.e., stimulated a strong urge to drink more), the magnitude of which correlated with the severity of alcohol dependence. Other researchers criticized the methodology used in previous studies. (For references, see Meyer, 1992.)

These findings help to explain why, beginning in the late 1970s, the classic disease concept was being reexamined and redefined as a symptom complex called "dependence" or "dependence syndrome." However, this shift has not satisfied some critics who object to any conceptualization that comes close to viewing compulsive alcohol or other drug use as a disease or disorder. The debate over the disease concept continues to be more heated in the alcohol field than in other areas of addictive disorders, such as compulsive use of opioids. In the early 1990s, however, an analogous and equally heated debate has developed about the conceptualization of tobacco smoking.

While health professionals throughout the world now generally agree that some forms of drug and alcohol use should be seen as disorders (at least for record-keeping and some public policy purposes), dissent from this view persists. The most compelling arguments against the disease concept have come from social and behavioral scientists. This may be partly because behavioral clinicians tend to work with less seriously impaired individuals, while physicians usually deal with people whose dependence has become more severe; and also because the physician's primary-care office may be where early identification of substance-abuse problems and effective behavioral interventions is most likely to take place.

ALTERNATIVE MODELS

Swedish researcher Lars Lindström's summary of current perspectives on the nature of alcoholism is equally applicable to the divergent views about other forms of excessive and/or compulsive drug use. Each of these models attempts to explain why people use alcohol or drugs, why use escalates to excessive and/or harmful levels, why some people continue drug use despite the harmful conse-

quences, how and why they stop using drugs, and why they relapse after a period of abstinence. The perspectives include the *moral model*, which holds that individuals have choice and are accountable for their behavior; the *disease model* (both the classic and its variants); the *symptomatic model*, which views excessive drug or alcohol use as a symptom of underlying psychiatric disorder; the learning model (drug addiction and alcoholism are learned behaviors); the *social model*, which emphasizes the primacy of environmental factors, such as availability, social controls, interpersonal relationships; and the *biopsychosocial model*, which attempts (in several variants) to synthesize elements of other models, taking into account biology, vulnerability, psychopathology, and cultural, social, economic, and pharmacological factors. The dependence syndrome model is probably best viewed as a variant of the biopsychosocial model.

Lindström points out that these models are now rarely encountered in pure form: each commonly incorporates elements from other perspectives. Furthermore, proponents of a particular model may, in practice, give greater emphasis to the central features of another. For example, ALCOHOLICS ANONYMOUS (AA) generally espouses the disease model. Yet because AA holds people accountable for the consequences of their drug use and emphasizes the central role of spiritual alienation in the perpetuation of alcoholism, AA's approach may also be seen as a variant of the moral model.

Although the term *disease concept* is often used synonymously with *biological* or *medical model*, these terms do not always convey the same ideas, especially with respect to implications for treatment. For example, the medical model of treatment is frequently contrasted with the social or *social recovery model*, now widely used and advocated in California. Medical-model programs are generally characterized not only by a philosophy about the problem but also by hospital-based detoxification, often pharmacologically assisted, and outpatient components in which there are formal treatment plans. Attention is paid to careful record keeping and professional credentials of the treatment staff. Physicians retain medical and legal responsibility for the overall program. In contrast, social-model recovery programs reject the involvement of professional staff and many of the activities of the medical model, such as the data gathering, licensing, and record keeping that link funding to units of service for specific patients. Instead, these programs emphasize the experience and knowledge that staff derive from the recovery process built on TWELVE-STEP mutual-help principles. There are no patients—only participants—and the role of staff is to manage the environment. Yet social models, in emphasizing the critical role that people "in recovery" play in the helping process, are employing a term—*recovery*—that is itself derived from the classic disease concept, which views alcoholism as a permanent disease state for which the only cure is total abstinence and the twelve-step AA program as the best route to such abstinence.

PERSISTENCE OF THE MORAL PERSPECTIVE

Despite the preponderance of medical opinion that some drug and alcohol users have a disorder—a diminished capacity to choose freely whether or not to use a particular substance—the moral models retain some vitality. In 1882, when the disease concept was first gaining momentum, the Reverend J. E. Todd wrote an essay entitled "Drunkenness a Vice, Not a Disease." In the late 1980s, the disease concept critics Fingarette and Peele put forth almost precisely the same thesis. Peele has argued that the disease concept exculpates the individual from responsibility, runs counter to scientific facts, and is perpetuated for the benefit of the treatment industry. However, his thesis has been criticized for using the classic disease model as a "straw man" because it does not take into account the more recent adoption of the biopsychosocial model.

Some sociologists in the United States have noted that the term *alcoholic* is still commonly used as a synonym for *drunkard* rather than as a designation for someone with an illness or disorder. The word *addict* is similarly used in a pejorative way, even when it is used more loosely to refer to a wide range of relatively benign behaviors, such as running or watching television. In the minds of most people, the concept of alcoholism or drug addiction as a disorder or disease can coexist quite comfortably with the concept of drunkenness or drug use as a vice. Since the nature of drug dependence is so closely linked to questions about the nature of free will and human volition—issues that have fascinated philosophers and scientists through the ages—it is likely that the disease concept of addic-

tion will continue to be debated for a long time to come.

(SEE ALSO: *Addiction: Concepts and Definitions; Alcoholism; Causes of Substance Abuse; Tolerance and Physical Dependence; Treatment, History of, in the United States*)

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DISTILLATION Distillation is the process of purifying liquid compounds on the basis of dif-

ferent boiling points or the process of separating liquids from compounds that do not vaporize. Since the actual process causes liquids to precipitate in a wet mist or drops that concentrate and drip, the word derives from the Latin *de* (from, down, away) + *stillare* (to drip).

In the simplest form of distillation, saltwater can be purified to yield freshwater by steam distillation, leaving a residue of salt. Distillation is also the process by which alcohol (ethanol, also called ethyl alcohol) as liquors or spirits, are separated from fermenting mashes of grains, fruits, or vegetables. When this process is used to distill alcohol, it is based on the following: Ethyl alcohol (C_2H_6O) has a lower boiling point than does water (78.5°C versus 100°C), so alcohol vapors rise first into the condenser, where cool water circulates around the outside of the condenser, causing the alcohol vapors to return to liquid form and drop into the collection flask. The purity of the distillate can be increased by repeating the process several times.

About 800 A.D., the process of distillation was evolved by the Arabian alchemist Jabir (or Geber) ibn Hayyah. He may also have named the distillate *alcohol*, since the word derives from an Arabic root, *al-kuhul*, which refers to powdered antimony (kohl) used as an eye cosmetic in the Mediterranean region; with time and use it came to mean any finely ground substance, then the "essence," and eventually, the essence of wine—its spirit, or alcohol. It came into English from Old Spanish, from the Arabic spoken by the Moors of the Iberian peninsula during their rule there (750–1492 A.D.).

(SEE ALSO: *Beers and Brews; Distilled Spirits, Types of; Fermentation*)

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DISTILLED SPIRITS, TYPES OF Distilled spirits (or, simply, spirits or liquors) are the ALCOHOL-containing fluids (ethanol, also called ethyl alcohol) obtained via DISTILLATION of fermented juices from plants. These juices include wines, distillates of which are termed brandies. The most commonly used plants are sugarcane, potatoes, sugar beets, corn, rye, rice, and barley; various fruits such as grapes, peaches, and apples are also used. Flavors may be added to provide distinctive character.

All distilled spirits begin as a colorless liquid, pure ethyl alcohol (as it was called by 1869)—C_2H_6O. This had been called *aqua vitae* (Latin, water of life) by medieval alchemists; today it is often called grain alcohol, and the amount contained in distilled spirits ranges from 30 to 100 percent (60 to 200 proof)—the rest being mainly water.

Examples of distilled spirits include brandy, whiskey, rum, gin, and vodka. Brandy was called *brandewijn* by the Dutch of the 1600s—burned, or distilled, wine. It was originally produced as a means of saving space on trade ships, to increase the value of a cargo. The intent was to add water to the condensate to turn it back into wine, but customers soon preferred the strong brandy to the acidic wines it replaced. Cognac is a special brandy produced in the district around the Charente river towns of Cognac and Jarnac, in France, where wine is usually distilled twice, then put into oak barrels to age. The spirits draw color and flavor (tannins) from the wood during the required five-year aging process.

Beer and wine were the most popular drinks of the New World colonists. By the mid-1700s, whiskey (from *uisce beathadh* in Irish Gaelic; *uisge beatha* in Scots Gaelic) was introduced into the American colonies by Scottish and Irish settlers to Pennsylvania. Whiskey is distilled off grains—usually corn or rye, but millet, sorghum, and barley are also used. Traditional American whiskeys are bourbons (named after Bourbon county in Kentucky), which are made from a sour mash of rye and corn. Bourbons typically contain 40 to 50 percent ethyl alcohol (called 80 to 100 proof, doubled by the liquor industry). Canadian whiskey is very similar to bourbon and to rye whiskey, while Irish whiskey is dry (has less sugars), with a distinctive austere flavor gained by filtration. All these whiskeys lack the smoky taste of Scotch whiskeys, which get their unique flavor by using malt that had been heated over peat fires. By using less malt and by aging for only a few years in used sherry casks (traditionally), a light flavor is produced; by using more malt and long aging, heavy peaty smoky flavors are produced. Today, some scotches

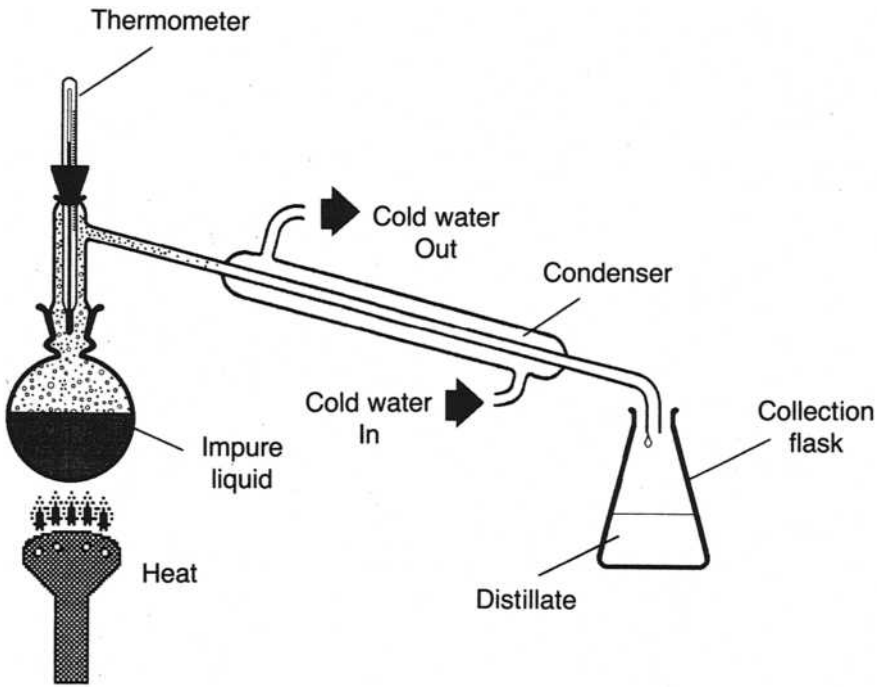
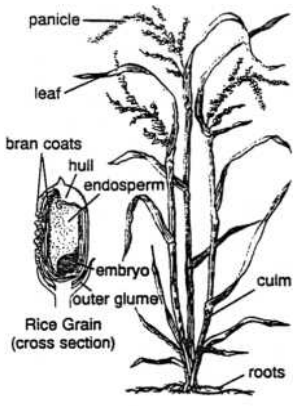


Figure 1
Simple Distillation Apparatus

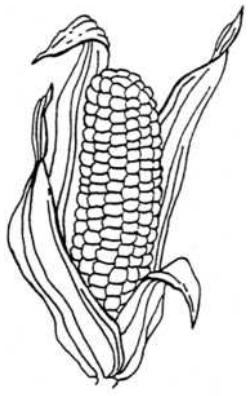
and other whiskeys are blended to achieve uniform taste from batch to batch.

The distillation of fermented sugarcane (*Saccarum officinarum*) results in rum. Of all distilled spirits, rum best retains the natural taste of its base, because (1) the step of turning starch into sugar is unnecessary; (2) it can be distilled at a lower proof; (3) chemical treatment is minimized; and (4) maturing can be done with used casks. The amount of added (sugar-based) caramel gives rum its distinctive flavor and color—which can vary from clear to amber to mahogany. The New England colonists made rum from molasses, which is the thick syrup separated from raw sugar during crystal sugar manufacture. Caribbean colonists grew sugarcane and shipped barrels of molasses to New England. New Englanders shipped back barrels of rum. Both substances were originally ballast for the barrels, which were made from New England's local forests to hold the sugar shipped from the Caribbean to the mother country, England.

Gin is a clear distillate of a grain (or beer) base that is then reprocessed; juniper berries and other herbs are added to give it its traditional taste. Vodka is also clear liquor, often the same as gin without the juniper flavor. Traditional vodkas,



Rice



Corn

Juniper

Wheat

made in Russia, Ukraine, Poland, and other Eastern European countries, are made from grain or potatoes at a very high proof; typical ranges are 65 to 95 proof, or about 33 to 43 percent ethyl alcohol. Vodka has no special taste or aroma, although some are slightly flavored with immersed grasses, herbs, flowers, or fruits. The Scandinavian aquavit is clear, like vodka, distilled from either grain or potatoes, and flavored with caraway seed; it is similar to Germany's kümmelvasser (*kümmel* means caraway in German). When any clear liquor is added to fruit syrups, the product is called a cordial or a liqueur. Swiss kirschwasser is, however, a clear high-proof cherry-based brandy (*Kirsche* means cherry in German); and slivovitz is a clear high-proof Slavic plum-based brandy.

The raw grain alcohol distilled in the American South and in Appalachia has been called *white lightning* since the early 1900s; this is also known as moonshine, corn whiskey, or corn liquor—illegally produced in private nonlicensed stills, in very high proofs, to avoid state and federal controls or taxation. The term *firewater* was used along the American frontier after about 1815, to indicate any strong alcoholic beverage; this was often traded, given, or sold to Native Americans, causing cultural disruptions and social problems that continue even today. These include a high rate of ALCOHOLISM and children born with FETAL ALCOHOL SYNDROME.

(SEE ALSO: *Alcohol: History of Drinking; Beer and Brews*)

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Oat

DISTILLED SPIRITS COUNCIL In 1974, the Distilled Spirits Council of the United States, Inc. (DISCUS) was formed by the merger of three organizations—the industrywide Licensed Beverage Industries, Inc. (LBI), the Distilled Spirits Institute (DSI), and the Bourbon Institute. DISCUS, headquartered in Washington, DC, is supported by the distilled spirits producers, representing 90 percent of the liquor sold in the United States. In all major respects DISCUS is a trade association representing producers and marketers of distilled spirits sold in the United States.

DISCUS's primary functions are to maintain legislative relations with state and federal governments (lobbying); to conduct or support economic and statistical research; to promote export and standards of identity for American-made liquors; to maintain a voluntary code of ADVERTISING practices; and to represent the distilling industry on social issues of concern, such as teenage drinking, DRUNK DRIVING, and other forms of ALCOHOL abuse. State government relations activities are conducted by DISCUS regional representatives.

As a trade association, DISCUS seeks to inform the public about the importance of distilled spirits to the U.S. economy. By 1999, the distilled spirits industry generated $95 billion in U.S. economic activity annually and over 1.3 million people were employed in the United States through the manufacture, distribution, and sale of distilled spirits. Jobs within the distilled spirits industry account for more than $28 billion in U.S. wages.

As had its predecessor LBI, DISCUS has supported programs of alcohol abuse PREVENTION and research conducted by independent groups and experts in education, traffic safety, and alcoholism. These projects have included the Grand Rapids, Michigan, study of drunk driving (1961–1965) and the research led by Harburg and Gomburg (1978–1984) on how drinking may affect the offspring of different types of drinkers.

In addition to supporting the Harvard Medical School course for DIAGNOSIS and TREATMENT of alcoholism, now adopted by eighty medical schools, DISCUS has provided extensive support to national organizations in the alcoholism field since 1970. Its approach is based on the knowledge that alcoholism is an identifiable illness and can respond to intervention and treatment.

In 1978, DISCUS endorsed the "responsible decisions on alcohol" approach developed by the Education Commission of the States, and in 1982, it supported the National Association of State Boards of Education in its nationwide project based on this concept (which includes abstinence). In 1980, DISCUS cooperated with the U.S. Department of Health and Human Services and other sponsors in supporting the Friends of the Family parenting education program.

In 1979, DISCUS became a charter member of the Licensed Beverage Information Council (LBIC), an industrywide consortium (beer, wine, and spirits at the producer, wholesaler, and retailer levels), whose membership includes nine other associations. LBIC has supported varied prevention groups and specialists in conducting medical and public education programs devoted to alcoholism as a treatable illness; FETAL ALCOHOL SYNDROME (FAS); teenage drinking; and drunk driving. The consortium has conducted the nationwide Friends Don't Let Friends Drive Drunk campaign.

DISCUS member companies are the principal supporters of the Century Council, a nonprofit organization dedicated to reducing alcohol abuse across the United States. Through public/private partnerships, Century Council investigates, funds, and implements innovative approaches to address the problems of drunk driving and underage drinking.

In 1994, DISCUS developed and initiated the Drunk Driving Prevention Act (DDPA), model legislation that strengthens drunk driving laws. Its provisions, many of which are being considered and adopted by state legislatures around the country, include mandatory alcohol and drug education for drivers; a ban of open containers in motor vehicles; Administrative License Revocation (ALR) authorizing a police officer to confiscate the license of any driver who either fails a chemical test or refuses to submit to it; zero tolerance for drivers under age 21; mandatory license revocation for persons under age 21 who attempt to purchase, consume, or misrepresent their age for the purpose of buying or consuming beverage alcohol; and mandatory alcohol and drug testing in fatal crashes.

DISCUS has also developed BACCHUS (Boosting Alcohol Consciousness Concerning Health of University Students). BACCHUS is a college-based peer education program to reduce alcohol abuse. Its educational materials include Open Doors, a Guide to Alcohol and Residence Life for Resident Administrators; Community College Guide to Peer Education; Gamma Guide (Greeks Advocating Mature Management of Alcohol); Certified Peer Educator Training Program; and Student Athletes as Peer Educators.

DISCUS has discouraged drinking by underage youth; encouraged adults who choose to drink to do so responsibly; and emphasized significant distinctions between normal social drinking and alcohol abuse.

The organization's economic research includes annual compilations of "apparent consumption" data (i.e., distilled spirits entering channels of

trade) and an assessment of the liquor industry's contributions to the economy. Total U.S. distilled spirits consumption has declined in recent years, a fact noted by DISCUS as one of the many refutations of the "control of alcohol availability" hypothesis.

The DISCUS Code of Good Practice provides for self-regulation of advertising practices by distillers. An unusually high degree of compliance has been achieved even with nonmembers. The code was applied to radio in 1936, when that was a major medium; it has voluntarily excluded the use of television as an advertising medium by distillers since 1947. Contrary to a widely held impression, spirits advertising on television is not prohibited by law.

Distilled spirits have been the most heavily taxed consumer commodity in the United States. DISCUS and its predecessors have claimed over the years that such taxes are discriminatory and excessive, and they do not reduce chronic alcohol-abuse problems. DISCUS has consistently argued that the tax structure imposed on distilled spirits is unjust because the government taxes spirits at a higher rate than beer and wine. It contends that standard servings of beer, wine, and distilled spirits contain the same amount of alcohol, yet the federal tax rate on distilled spirits is almost three times the rate on wine and over two times the rate on beer.

In 1999, DISCUS continued to lobby Congress for a reduction in excise taxes. DISCUS pointed out that more than half of the price that consumers spend on a typical bottle of distilled spirits is taxes. Federal, state, and local governments receive more than $18 billion per year in tax revenue from the beverage alcohol industry and tax revenues from the distilled spirits industry alone account for more than $7.5 billion. DISCUS pointed out that federal, state and local governments combined realize fourteen times more in spirits tax revenues than the distillers make in profits. However, Congress has remained unresponsive to the attempt by DISCUS to reduce excise taxes.

As a long-standing policy, DISCUS and its members do not encourage people to start drinking or to drink too much. DISCUS's review of the research literature indicates that there is no scientific evidence that brand advertising either influences or shapes those behaviors. The marketing purpose of product advertising is to build consumer acceptance of specific brands, according to DISCUS. In the late 1990s, DISCUS began publicizing the health benefits of alcohol consumption. It noted a growing body of scientific evidence reporting that moderate beverage alcohol consumption may reduce the risk of cardiovascular disease, the leading cause of death in the United States. This potential benefit is equally available from moderate consumption of any form of beverage alcohol—distilled spirits, beer, or wine. However, DISCUS does not promote the use of alcohol consumption for health reasons.

(SEE ALSO: *Advertising and the Alcohol Industry; Alcohol: History of Drinking; Legal Regulation of Drugs and Alcohol; Minimum Drinking Age Laws; Prevention; Social Costs of Alcohol and Drug Abuse; Tax Laws and Alcohol*)

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DISULFIRAM The registered trademark name for disulfiram is Antabuse—it is the most commonly used medication for the treatment of ALCOHOLISM and the only one of two medications (the other being naltrexone) approved for this use in the United States, as of 2000. It is not intended as a substitute for the counseling alcoholics receive while in treatment; it is meant to be an aid in keeping alcoholics sober, so that they may benefit from counseling. Although disulfiram has been in clinical use since the late 1940s, only since the 1980s has its efficacy been studied by appropriate scientific methodology.

Disulfiram is used to deter drinking by causing an unpleasant reaction if a medicated person drinks ALCOHOL (ethanol). This reaction is called

the disulfiram–ethanol reaction (DER); the symptoms include flushing, dizziness, rapid heartbeat, nausea, vomiting, and headache. The DER is of varying severity, and the degree of severity often depends on the dose of disulfiram being taken plus the amount of alcohol that was consumed. A DER can cause hypotension (low blood pressure) and can be so severe that death occurrs, although with adjusted dosage regimens this is very rare.

Disulfiram blocks the action of several of the body's enzymes, including aldehyde dehydrogenase (ALDH). The inhibition of ALDH is responsible for the DER; this occurs because ethanol (drinking alcohol) is metabolized in the liver to acetaldehyde. Acetaldehyde, in turn, is converted to acetic acid, which is catabolized to water and carbon dioxide.

Aldehyde dehydrogenase is the enzyme that facilitates the catabolism of acetaldehyde to acetate acid. When the action of ALDH is inhibited by disulfiram, acetaldehyde is not converted to acetate but accumulates in the blood. Most of the symptoms of the DER are due to the increased circulating acetaldehyde. Since the inhibition of ALDH by disulfiram is irreversible, a person taking disulfiram cannot stop taking it one day and begin drinking the next—several days (usually 4 to 7) must go by, because this is the amount of time necessary for the body to produce new enzyme.

OTHER MEDICATIONS

Certain other medications cause a mild DER, including such antibiotics as metronidazole (Flagyl). A medication available in Canada but not in the United States is citrated CALCIUM CARBIMIDE (Temposil), which inhibits ALDH in a mixed reversible–irreversible fashion. When citrated calcium carbimide is discontinued, 80 percent of ALDH activity is restored within 24 hours. Hence, one can drink alcohol as soon as a day after stopping the use of citrated calcium carbimide without having a reaction.

In addition to disulfiram, there are other medications with different mechanisms of action that are now approved for use in helping recovering alcoholics maintain sobriety. The most promising of these newer drugs is naltrexone hydrochloride (ReVia), an opioid agonist that was found in 1992 to reduce the incidence of relapse in alcoholics in outpatient treatment programs. Since approval of the drug by the U.S. Food and Drug Administration (FDA), a number of additional studies of naltrexone have been published. In addition, a large number of studies are underway, with support from the National Institute on Alcohol Abuse & Alcoholism (NIAAA). Although, most studies of naltrexone have shown it to be helpful for relapse prevention, not all studies have been positive.

ADMINISTRATION AND DOSAGE

Disulfiram should be administered only by a physician and is given by mouth in tablet form. It should never be given until the patient has abstained from alcohol for at least 12 hours, and preferably for 48 hours. The dose is usually 250 or 500 milligrams daily. Some patients report not experiencing a DER with smaller doses, so larger doses may be required. Clinical experience indicates, however, that doses larger than 500 milligrams are accompanied by a greater risk of serious side effects. A problem that limits the effectiveness of disulfiram is that patients frequently stop taking the medication. To prevent this, disulfiram tablets sometimes have been implanted just below the skin of the abdominal wall. This technique, however, has been shown to be ineffective (Johnsen et al., 1987) because the absorption of the implanted disulfiram is erratic and poor, resulting in very low blood levels of disulfiram and a weak or no DER.

Patients should take disulfiram only under careful medical and nursing supervision. They should be warned that as long as they are taking the drug, ingesting alcohol in any form will make them sick and may endanger their life. Patients should be taught to recognize and avoid disguised forms of alcohol such as cough syrups, mouthwashes, some sauces, fermented vinegar, and even aftershave lotion or rubbing alcohol. In addition, patients should be taught to recognize the signs of disturbed liver function (jaundiced eyeballs or skin, nausea or pain in the upper right quadrant of the abdomen, dark urine, clay-colored stool) and report them at once to their doctor.

SIDE EFFECTS

The use of disulfiram may be accompanied by side effects. The most common one is drowsiness; for this reason, the medication is usually taken at bedtime. Timing is usually sufficient to take care of

this problem, but if not, the medication may have to be discontinued, especially for those who drive or work in hazardous environments. Idiosyncratic liver toxicity can occur from taking disulfiram. For this reason, liver function must be monitored closely during the first several months of treatment, and if blood tests indicate possible liver damage, disulfiram must be discontinued immediately.

A 1986 Swedish study found that disulfiram enhances the absorption and toxicity of lead in rats. Recovering alcoholics who must work in environments containing lead or lead products are advised not to use disulfiram to maintain sobriety.

In addition, serious psychotic reactions and depressive episodes have occurred in patients taking disulfiram. In a multisite study of 605 men, admissions for psychiatric problems were uncommon; as many admissions of this type occurred in men taking the placebo or not receiving disulfiram as in those receiving a 250-milligram dose (Branchey et al., 1987). The risk of serious psychoses or of major affective illnesses occurring appears to be worse with higher doses.

INTERACTIONS WITH OTHER DRUGS

Disulfiram should not be given to patients who are taking metronidazole (Flagyl) or paraldehyde (Paral), as these drugs will produce a DER. Patients taking isoniazid (INH, Laniazid) may develop neurological symptoms if given disulfiram. Lastly, disulfiram may increase the blood levels and toxicity of warfarin (Coumadin), barbiturates, and phenytoin (Dilantin).

(SEE ALSO: *Naltrexone; Relapse; Treatment: Alcohol; Treatment Types: Aversion Therapy*)

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DIVERSION *See* Treatment Alternatives to Street Crime

DMT *See* Dimethyltryptamine

DOGS IN DRUG DETECTION In 1970, the U.S. CUSTOMS SERVICE faced a shrinking inspectional staff, a flood of illegal NARCOTICS, and an increasing load of vehicles and passengers entering the United States. In that same year a manager in the U.S. Customs Service thought that dogs could be used to detect illegal narcotics. The manager's name has been lost in the corporate history of the Customs Service, yet years later not only are dogs used to detect narcotics but also currency, weapons, explosives, fruits, and meats. Dogs could be trained to detect anything that produces an odor. Although the idea of narcotic detector dogs originated in the U.S. Customs Service, Customs' managers had to go to the U.S. Air Force for the technical expertise—not in narcotic detection, because it did not exist, but dog training in general. The air force loaned the Customs Service five instructors to develop the program. Those instructors, using the age-old method of trial and error, developed a training method for narcotic detection that was still used in the 1990s. Through the years, several key aspects of the training program were identified and became the basis for a very successful program—dog selection; development of a conditioned response; and odor integrity.

It became evident that to make the training program successful the instructors had to start with a

An airport security officer guides a dog along a row of luggage, checking for narcotics. In the United States, the Customs Service's Canine Enforcement Program has accounted for more than 120,000 drug seizures. (© Galen Rowell/ CORBIS)

dog that displayed certain natural traits. Those traits were retrieval motivation and self-confidence. The instructors soon realized that a dog displaying a natural desire to retrieve was the easiest to condition for response to the narcotic odor. They used the retrieval method just as the Russian physiologist Ivan Pavlov (1849–1936) used a bell: In Pavlov's experiments, he had observed that dogs salivate when food is placed in their mouths. He would give the dogs food while providing another stimulus such as a bell ringing. After a few repetitions, the dogs would salivate when they heard the bell ring—even without the food being present. The dogs had learned to associate the bell ringing with food. This response was called a *conditioned response*.

The Customs' instructors used a similar method to create a conditioned response to a drug odor. A dog was subjected to a series of retrieval exercises with a specific drug's odor. After each retrieval, the dog played a game of tug-of-war with its handler and would receive physical praise. The dog soon associated the specific drug odor with the game and the physical praise.

Using the dogs' natural desire to retrieve as a selection criterion limited the number of breeds that could be considered for this type of training. It was obvious that most of the sporting breeds fit this criterion—golden retrievers; Labrador retrievers; German short-hair retrievers; and mixed breeds of these types. They have had the retrieval drive bred into them over the centuries. In addition, these breeds predominated in the dog shelters and humane societies used by U.S. Customs as the primary source for its dog procurement, which has not only benefited the Customs' program but the local dog shelters too. Local shelters must by law destroy stray dogs after a certain time period if no one selects or adopts the dog. The Customs' instructors select dogs scheduled to be put to sleep.

The Customs' training method is based on the natural behavior of these retriever breeds of dogs. By using a dog's natural behavior, the instructors can adjust certain aspects of their training program to deal with the individual personality of each dog. Although each dog that entered the training program possessed the same basic qualifications, each then displayed them in varying degrees of intensity because of personality differences.

During the training process, the other aspect of the program that ensured success has been maintaining the integrity of the narcotic odor. During the development of the training program there were several incidents when the detector dog would respond to nondrug odors. In those incidents a common factor was identified: The nondrug odor was present in the training program. To the dog, the materials that were used in the scent-association process (a process by which the dog identifies the narcotic odor with the tug-of-war game) combined with the drug odor represented a completely different odor picture (a combination of odors that the dog associates with a positive reward). This situation became apparent when the drug was separated from the other materials; the dog would not respond to it alone or would display a considerable amount of confusion when confronted with it. This problem was eliminated by ensuring that all materials used in the training process smelled like the specific drug in question.

In summary, the key factors in narcotic detector-dog training is (1) dog selection; (2) development of a conditioned response; and (3) the integrity of the narcotic odor. This information is a very small segment of the overall training methodology. Further information about this type of dog training can be obtained through the U.S. Customs Service's Office of Canine Enforcement Programs, Washington, D.C.

(SEE ALSO: *Drug Interdiction*)

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DOLOPHINE *See* Methadone

DOM This drug's street name is STP. During the hippie drug culture of the VIETNAM war period, its name referred to "serenity, tranquility, and peace." This was also a taunt and a spoof, since the initials were the same as a widely available oil additive that made an automobile engine run smoothly. The drug DOM is a member of a family of HALLUCINOGENIC substances based on molecular additions to phenethylamine. This is a group of compounds that have structural similarities to the catecholamine-type NEUROTRANSMITTERS, such as NOREPINEPHRINE, epinephrine, and DOPAMINE. While our bodies make these catecholamines from dietary amino acids, they do not make the chemical substitutions that produce a PSYCHEDELIC compound. MESCALINE is the best and longest known of this family of HALLUCINOGENS.

DOM is a synthesized compound that produces effects similar to mescaline and LYSERGIC ACID DIETHYLAMIDE (LSD), but the effects of DOM can last for fourteen to twenty hours, much longer than those of LSD. In addition, the effects of DOM have a very slow onset. Some of the initial street users of DOM had previous experience with LSD, a drug with a much more rapid onset. When the typical LSD-type effects were not found soon after taking DOM, some users took more drug, which led to a very intense and long-lasting psychedelic experience. Ironically, DOM was originally manufactured in the hope of producing a shorter, less-intense trip than LSD, which, it was thought, might be more useful and manageable in producing a period of insight and self-reflection in psychotherapy. This hope was never achieved.

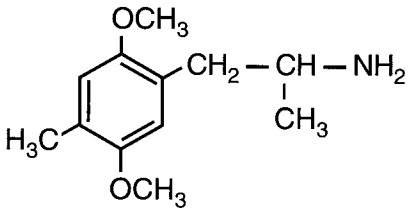

Figure 1
DOM

(SEE ALSO: *Designer Drugs; Dimethyltryptamine*)

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DOPAMINE Dopamine (DA) is a catecholamine according to its chemical structure and a neurotransmitter of special importance for drug addiction. DA is a decarboxylated form of dopa (an amino acid) found especially in the basal ganglia. Chemically known as 3,4 dihydroxyphenylethylamine, DA arises from dihydroxphenylacetic acid (dopa) by the action of the enzyme dopa decarboxylase. Dopamine-containing NEURONS (nerve cells) are widespread in the brain and the body. Small interneurons are found in the autonomic ganglia, retina, hypothalamus, and medulla. Long axon neurons are found in two extensive circuits: (1) the nigrostriatal pathway links the substantia nigra neurons to the basal ganglia neurons and regulates locomotor events; (2) the mesocortical and mesolimbic circuits arise in the ventral tegmental area and project to the neocortex, limbic cortices, nucleus accumbens, and amygdala, where they regulate emotional events, including several forms of drug addiction, reinforcement, or reward. DA is also found in minute amounts in other catecholamine neurons as a precursor to norepinephrine. The DA transporter, which transports DA from outside the nerve terminal to inside the nerve terminal, functions to retrieve released DA and help terminate its action at receptors. The transporter is the target of psychostimulant drugs that produce their effects, at least in part, by blocking the transporter and preventing its removal from receptors. A consistent observation, for example, is the efflux of DA from nerve terminal regions in the nucleus accumbens in response to giving animals a psychostimulant such as cocaine or amphetamine. DA is

also thought to be involved in schizophrenia and psychosis since DA-receptor-blocking drugs are clinically useful antipsychotic agents. Another disease, in which DA is lost due to the degeneration of DA-containing neurons, is Parkinson's disease, which can be treated by replacing DA with its precursor, dopa.

FLOYD BLOOM

DOPE/DOPE FIEND *See* Slang and Jargon

DORIDEN *See* Glutethimide

DOSE-RESPONSE RELATIONSHIP The relationship between the dose (amount) of a drug and the response observed can often be extremely complex, depending on a variety of factors including the absorption, metabolism, and elimination of the drug; the site of action of the drug in the body; and the presence of other drugs or disease. In general, however, at relatively low doses, the response to a drug generally increases in direct proportion to increases in the dose. At higher doses of the drug, the amount of change in response to an increase in the dose gradually decreases until a dose is reached that produces no further increase in the observed response (i.e., a plateau). The relationship between the concentration of the drug and the observed effect can therefore be graphically represented as a hyperbolic curve (see Figure 1).

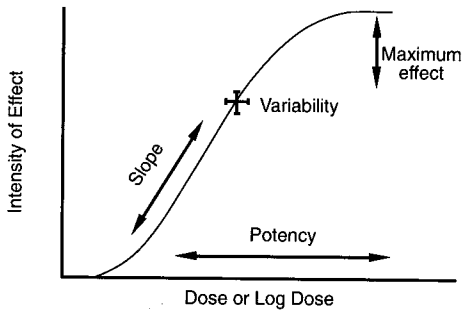

Figure 1
Representative Dose-Effect Curve, with Its Four Characteristics

Often, however, the response (ordinate) is plotted against the logarithm of the drug concentration (abscissa) to transform the dose-response relationship into a sigmoidal curve. This transformation makes it easier to compare different dose-response curves—since the scale of the drug concentration axis is expanded at low concentrations where the effect is rapidly changing, while compressing the scale at higher doses where the effect is changing more slowly (see Figure 2).

Finally, there are two basic types of dose-response relationships. A graded dose-response curve plots the degree of a given response against the concentration of the drug as described above. The second type of dose-response curve is the quantal dose-effect curve. In this case, a given quantal effect is chosen (e.g., a certain degree of cough suppression), and the concentration of the drug is plotted against the percentage of a specific population in which the drug produces the effect. The median effective dose (ED50 or the dose at which 50% of the individuals exhibit the specified quantal effect) and the median lethal dose (LD50 or the dose at which death is produced in 50% of the experimental animals in preclinical studies) can be estimated from quantal dose-effect curves. With this type of curve, the relative effectiveness of various drugs for producing a desired or undesired effect, as well as the relative safety between various drugs, can be determined. The ratio of the LD50 to the ED50 for a given effect indicates the therapeutic index of a drug for that effect and suggests how selective the drug is in producing its desired effects. In clinical studies, the concentration of the drug required to produce toxic effects can be compared to the con-

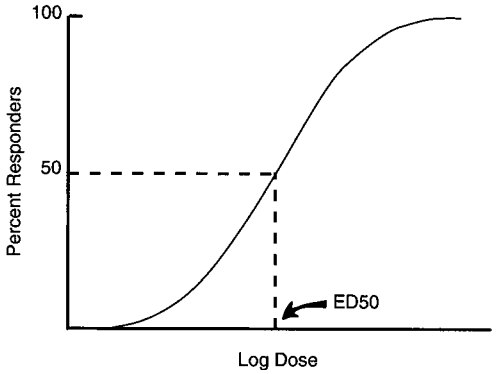

Figure 2
Representative Dose-Effect Curve, Showing a Median Effective Dose (ED50)

centration required for a specific therapeutic effect in the population to estimate the clinical therapeutic index.

(SEE ALSO: *Drug Metabolism*)

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NICK E. GOEDERS

DOVER'S POWDER *Dover's Powder*, developed and described by the British physician Thomas Dover in 1732, was one of the more popular and enduring of the opium-based medications that were widely used in the United States and Europe prior to the twentieth century. The medication combined OPIUM with what we know today as ipecac (ipecacuanha), a substance that induces vomiting. The result was a pain-reducing potion that might induce a sense of euphoria but could not be ingested in large quantities because of its emetic properties. Taken as a nonprescription medicine by the general public for over 200 years, it was also prescribed by physicians for home and hospital use. Versions of the preparation are still listed in pharmaceutical formularies in which "Dover's Powder" commonly denotes any opium-based mixture that includes ipecacuanha. The wide use of Dover's Powder declined in the early 1900s largely because of the addiction that resulted from the prolonged use of OPIATES, because of the introduction of other nonaddicting ANALGESICS (painkillers), mainly aspirin, and because of laws regulating sales of opium products.

Thomas Dover (1662–1742) studied medicine at Oxford University in the 1680s. He claimed to have served an apprenticeship with Dr. Thomas Sydenham, the illustrious seventeenth-century practitioner and teacher, who originated the formula for LAUDANUM, another early and popular opium-based medicine. Dover practiced medicine for over fifty years, although during his lifetime he was more famous for his exploits as an adventurer and privateer. His involvement in the early slave trade and in the plundering of Spanish settlements off the coast of South America brought him fortune and fame. On one of his voyages he found the shipwrecked Alexander Selkirk, who, on being returned to London, created a sensation and was to become the inspiration for Daniel Defoe's *Robinson Crusoe*. Dover retired from his merchant sailing career a wealthy man, but poor investments led him to resume his medical career first in Gloucestershire and later in London.

In 1732, probably to attract patients to his new practice in London, Dover published *An Ancient Physician's Legacy to His Country*, one of the earliest medical treatises written for the general public. The book listed forty-two ailments with successful treatments used by Dover, and included the testimonial letters of many "cured" patients. The book enjoyed popular success and was reprinted eight times, the last in 1771, nearly thirty years after his death. One remedy described in the book, the use of mercury, earned him the nickname during his lifetime of the Quicksilver Doctor, but the formula for Dover's Powder, which appears unchanged in all eight editions, has proven to be his most enduring legacy. Appearing on page 18 of the original edition as a treatment for gout, the directions read:

> Take Opium one ounce, Salt-Petre and Tartar vitriolated each four ounces, Ipecacuana one ounce, Liquorish one ounce. Put the Salt Petre and Tartar into a red hot mortar, stirring them with a spoon until they have done flaming. Then powder them very fine; after that slice your opium, grind them into powder, and then mix the other powders with these. Dose from forty to sixty or seventy grains in a glass of white wine Posset going to bed, covering up warm and drinking a quart or three pints of the Posset—Drink while sweating.

Dover's familiarity with opium most probably resulted from his association with Thomas Sydenham and thereby his acquaintance with the benefits of laudanum (an alcoholic tincture of opium). Dover's ingenious use of opium with ipecacuanha seems to have been original. His unique formula, Pulvis Ipecacuanha Compositus, with its specificity of ingredients, produced a relatively reliable and consistent potion in an era when there was no regulation of medications and little standardization in their preparation. Medications could be purchased at apothecary shops with or without doctors' prescriptions or at back-street stores that sold drugs along with food, clothing, and other necessities of life. The major issue at the time in the use of

opiate-based medications was not that they contained what we now know to be a NARCOTIC, but whether the consistency of the formula or the misuse by the patient caused overdoses of what could be poisonous ingredients. Dover's Powder provided a stable product that, because of the ipecacuanha, could not be taken in excess at any one time. The powder came to be trusted by the general public and widely prescribed by physicians. It was considered such a safe remedy that it was even prescribed for children.

Although Dover originated his powder as treatment for gout, it was used throughout the eighteenth and nineteenth centuries along with many other opium-based patent and official preparations by large numbers of people for a wide variety of disorders. Opium, used as a healing plant for over 6,000 years, was an ingredient in countless formulas that were openly available and credited with curing the most common disorders of the time. Mixed in a tincture, it was found in laudanum; in a camphorated formula it became PAREGORIC; and it was also included in preparations for lozenges, plasters, enemas, liniments, and other general medications.

Opium-based medicines were used for many disorders, including insomnia, diarrhea, bronchitis, tuberculosis, chronic headache, insanity, menstrual disorders, pain, malaria, syphilis, and smallpox. Often both physicians and patients mistook its narcotic properties, which relieved pain and created a sense of well-being, as curative rather than palliative, and little was understood of the darker side of opiate medications—the destructive nature of addiction—until well into the nineteenth century. By this time, it was common for middle- and upper-class people, especially women and those with chronic diseases, to be addicted to opiates that were frequently seen in innocuous health elixirs or in remedies that had been originally prescribed by physicians. Widespread prescribing by physicians and easy availability of the opiate medications made addiction a frequent result of medical therapeutics.

By the middle of the nineteenth century, the issue of opium addiction began to appear with more frequency in the medical literature, and in both the United States and England there were pressures to regulate both the pharmacy trade and the use of narcotic medications, especially the patent medicines containing opiates. Even then, it was not until the end of the century—as a result of better education of physicians and pharmacists, advances in diagnosis and therapeutics, and a growing understanding of the nature of addiction—that opium-based medications were supplanted by other curative treatments and by nonaddictive salicylate analgesics such as aspirin. Opium-based medicines used today, such as MORPHINE and CODEINE, are government-regulated and can be purchased legally only by prescription.

Dover's Powder in its original form is now an obsolete medication. It should be recognized for its place in the history of pharmacology as a relatively reputable medicine used from 1732 until the 1930s, an era in which opium-based medications were one of the few remedies that brought relief to suffering patients. Many of these medications came to be misused by both patients and physicians who had little understanding of addiction and few options for PAIN relief. Thomas Dover, seen as an adventurer and opportunist by many during his lifetime, developed a preparation that allowed patients to use a narcotic while limiting its ingestion. More precise knowledge of the healing as well as the addictive properties of narcotics allows modern physicians and pharmacologists to deal specifically with the dosage of narcotic medications. Nevertheless, Dover's Powder, an ingenious and effective solution to a thorny problem, became a household name long after its originator's medical career had ended and his medical treatise had been published.

(SEE ALSO: *Addiction: Concepts and Definitions; Britain, Drug Use in; Disease Concept of Alcoholism and Drug Abuse; Opioids and Opioid Control: History*)

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VERNER STILLNER

DRAMSHOP LIABILITY LAWS Dramshops are taverns, saloons, bars, and drinking establishments. All states impose fines and other punishments when alcohol is sold to "visibly intoxicated" customers or "habitual drunkards." Although historically these laws aimed to preserve public order and morality, today they are perceived primarily as tools to curtail drunk driving. Their effectiveness is a direct function of compliance and enforcement. Although compliance has rarely been studied, one study in Michigan found that an increase in police enforcement (through visits and warnings) resulted in a three-fold increase in the rates of service refusal to intoxicated patrons. In addition, service intervention training has been voluntarily implemented in many states and is required by law in some. Although the evidence is mixed, recent research indicates that sustained server training can reduce the risk of drunk driving.

In addition to these statutory penalties, more than half the states also impose tort liability on tavern keepers for injuries caused by intoxicated patrons. Liability in such situations serves both compensatory and deterrent purposes. Although it is difficult to isolate the effects of the threat of so-called "dramshop tort liability" or server behavior, one study attributed a decline in traffic crash injuries in Texas to the filing of two major liability suits in that state (Wagenaar & Holder, 1991). Courts in a few states have extended the dramshop principle to private "social hosts" who fail to take adequate precautions to prevent obviously intoxicated guests from getting behind the wheel.

Whether or not the threat of liability for servers of alcohol exerts a clear-cut deterrent effect, it is clear that dramshop liability serves an important pedagogical effect and, together with other legal and cultural factors, helps to shape social norms against driving while intoxicated.

(SEE ALSO: *Alcohol: History of Drinking; Driving, Alcohol, and Drugs; Driving Under the Influence; Drug Interactions and Alcohol; Drunk Driving; Legal Regulation of Drugs and Alcohol; Mothers Against Drunk Driving; Students Against Destructive Decisions*)

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CHRISTOPHER B. ANTHONY
REVISED BY RICHARD J. BONNIE

DRIVING, ALCOHOL, AND DRUGS Injuries, especially from motor vehicle collisions, are the leading cause of death for individuals under age 44. The presence of alcohol is the factor most frequently associated with fatalities in vehicles, drownings, falls, and fire (U.S. Department of Health and Human Services, 1987). In the first report to Congress on traffic safety and alcohol (U.S. Department of Transportation, 1968), it was concluded that more than 50 percent of fatal traffic collisions and 33 percent of serious injury traffic collisions were alcohol-related.

Although the association between alcohol consumption and traffic accidents had been recognized by the beginning of the twentieth century, the magnitude of the problem did not capture public attention until the 1970s. Public tolerance of DRIVING UNDER THE INFLUENCE of alcohol decreased sharply—a shift in attitude that, combined with increased legal countermeasures, resulted in a significant decline in alcohol-related fatalities from a high of 57 percent in 1982 to 38 percent in 1998.

Voas et al. (1998) compared the relative frequency of driving under the influence of alcohol in three U.S. nationwide surveys, done in 1973, 1986, and 1996 on weekend nights. Drivers were stopped

at random and asked to provide breath samples for alcohol testing. The blood alcohol concentration (BAC) levels were compared for the three surveys as a function of time, day, gender, age, ethnicity, geographical region, etc. Across nearly all population subgroups, the presence of alcohol in nighttime weekend drivers dropped from 36 percent in 1973 to 26 percent in 1986 to 17 percent in 1996. However, although the percent of decline for drivers with BACs below 0.10 percent was 54 percent from 1973 to 1996, there was only a 45 percent decline in drivers with over 0.10 percent BAC. Despite this significant drop in the number of alcohol impaired drivers in the last two and a half decades, alcohol still remains the single largest factor in traffic fatalities and serious injuries.

Epidemiological studies have compared the BAC levels in collision-involved drivers with those of randomly selected drivers passing the collision site at similar times. These studies have demonstrated that the probability of a crash increases with any departure from zero BAC and increases exponentially with increasing BAC levels. By the time BAC levels exceed 0.2 grams alcohol per deciliter of blood (200 mg/100 mL.), the probability of a collision increases more than 100 times (i.e., 10.000%).

Most areas of human behavior are impaired eventually by increasing alcohol levels. However, the examination of alcohol-related collision data from governmental investigations and police collision reports suggests that information-processing errors are common in the majority of alcohol-related traffic collisions. Information-processing deficits include impairment of attention, visual search, and perception. The second largest category of human factor errors involves judgment, such as speed selection. Failure to control a car because of decreasing motor skills remains a distant third crash category, despite the popular assumption that links driving impairment with the appearance of intoxication and motor incapacitation.

The results observed in epidemiological survey studies are supported by numerous experimental studies in which driver behavior was examined under controlled conditions. Such laboratory studies either examine one or two behaviors relevant to driving at a time or in more complex studies of driving-related behavior use driving simulators and a closed-course driving situation that preserve the safety of the driver.

Moskowitz and Robinson (1988) reviewed 177 experimental studies of alcohol's effects on driving-related behaviors that met criteria of scientific merit. The behavior found to be most affected by alcohol was divided attention, with impairment even seen at alcohol levels below 0.02 percent (20mg/100mL). Divided-attention tasks involve simultaneously monitoring and responding to more than one source of information, which is characteristic of many complex man-machine interactions such as driving and flying. While operating a vehicle, drivers under the influence of alcohol frequently fail to detect significant potential threats in the environment.

Similarly, studies have indicated that information processing and perception are affected at low BAC levels. Tracking, which is analogous to car control functions such as maintaining heading and lane position, has been shown to be impaired at low BACs when performed simultaneously with other functions in divided-attention situations, but less impaired when the tracking task is performed by itself. Similarly, complex reaction-time tasks involving several competing stimuli and responses are impaired at low BACs, whereas simple reaction time requiring little information processing was more resistant to the effects of alcohol.

Studies of psychomotor skills performance in driving simulators and closed-course driving studies have shown considerable variation in the BAC levels at which impairment appears. These variations are likely explained by the differences in information-processing requirements among these varied tasks. The review concluded that no minimum threshold for alcohol's impairment of complex human-machine tasks exists. Any reliable measure of alcohol in the human system produces some impairment.

Other areas that have been suggested as leading to alcohol-related accidents, such as poor judgment and violent and aggressive behavior, have been infrequently examined by researchers—mainly because of the difficulty of developing laboratory techniques to measure them.

The low BAC levels at which laboratory studies have indicated significant impairment and epidemiological studies have shown increased crash frequency are below the levels at which the majority of the population would exhibit symptoms of intoxication such as slurred speech and unsteady gait. Thus, the absence of signs of intoxication is not

evidence that a driver is capable of operating a motor vehicle or other machinery safely.

Moskowitz and Fiorentino (2000) updated Moskowitz and Robinson's 1988 report with a review of an additional 112 studies published from 1981 to 1997. Although the main conclusions of the 1988 report remained confirmed, the most recent publications more frequently report impairments at very low alcohol levels, reflecting improvements in the sensitivity and reliability of scientific investigation. Moreover, new behavioral areas are being explored, such as the tendency to fall asleep at the wheel, which increases significantly at low BAC levels.

OTHER DRUGS

The major involvement of alcohol in traffic accidents and other injuries is well documented. What conclusions can we draw about the role of drugs other than alcohol in traffic safety? Although laboratory studies on the effects of many drugs and alcohol are similar in demonstrating the impairment of performance skills, there are difficulties in executing epidemiological studies on the effects of drugs in driving. For example, few non-crash-involved drivers volunteer to provide blood samples so their drug levels might be compared with those in blood samples obtained from collision victims.

Although several studies have been completed in hospitals with drug levels in trauma patients involved in driving collisions with blood samples from volunteers who were in the hospital for other reasons, serious questions arise regarding the representativeness of the control group.

Another problem in relating drug use to vehicle crashes has been difficulty of evaluating the behavioral significance of drug blood levels. Unlike alcohol, where levels in venous blood samples or breath samples are essentially equivalent to those from blood in the brain, the site of drug action, for nearly all other drugs have a complex relationship between blood plasma level and the degree of resulting behavioral impairment. Many drugs remain present in the plasma for weeks beyond any period in which behavioral effects may be observed. In other cases, drug levels in plasma drop extremely rapidly and become difficult to detect while behavioral impairment remains. Thus, most epidemiological studies of drugs and driving report the presence of the drug rather than the level of concentration.

One technique to circumvent control-group problems has been to assign responsibility or nonresponsibility to crash-involved drivers and then correlate the presence of drugs with the frequency of crash responsibility. Within the constraints of these epidemiological studies, researchers have often concluded that tranquilizers, antihistamines, and antidepressants are overrepresented in crash-involved drivers.

Terhune and colleagues (1992) examined the presence of drugs in blood specimens from 1,882 fatally injured drivers. Drugs, both illicit and prescription, were found in 18 percent of the fatalities. MARIJUANA was found in 6.7 percent, COCAINE in 5.3 percent, tranquilizers in 2.9 percent, and AMPHETAMINES in 1.9 percent of these fatally injured drivers.

When crash responsibility was assigned and correlated with drug use, the small number of individuals in each separate drug classification made statistical significance difficult to obtain despite the fact that several drug categories were associated with increased crash responsibility. Crash-responsibility rates did increase significantly as the number of drugs in the driver increased. Many drug users used several drugs simultaneously and these drivers had the highest collision rates. Alcohol was found in 52 percent of the fatalities, with more than 90 percent of the drivers with BACs over 0.08 percent considered responsible for the crash.

The most frequently used illicit drug in the United States of America for the last half-century is marijuana and epidemiological studies have demonstrated that is the most frequent drug consumed by drivers. Bates and Blakely (1999) have reviewed the epidemiological literature for marijuana's role in motor vehicle crashes. They concluded that there is no evidence marijuana alone increased either fatal or serious injury crashes. However, the evidence is inconclusive whether the presence of marijuana in combination with alcohol increases fatalities or serious injuries over that produced by alcohol alone. Nor was it possible to determine whether marijuana increases the rate of less serious vehicle crashes.

In contrast to the lack of scientific information available from epidemiological sources about the role of drugs in causing collisions, numerous experimental research has been performed to evaluate

the effects of drugs on skills performance. Regulatory agencies in many countries have frequently required an evaluation of the side effects of prescription drugs on skills performance. Also, numerous governments have supported studies of the effects of illicit and abused drugs on skills performance in the laboratory.

Thus, the evaluation of the effects of drugs on driving and other human-machine interactions has depended primarily on experimental studies where changes in behavior can be observed as a function of differences in administered doses and the time after administration. However, no other drug has been evaluated in as extensive a range of behaviors as has alcohol. Nevertheless, many drugs have been studied with respect to some important variables required for driving.

The emphasis in these drug studies has tended to be on the evaluation of vision, attention, vigilance, and psychomotor skills. Driving-simulator studies have also been done on occasion. The psychomotor skill most often examined has been some form of tracking.

Reviewing this literature presents considerable difficulties since there are so many differences between classes of drugs, as well as between individual drugs within the same drug classification. For example, many minor tranquilizers, especially BENZODIAZEPINES, have been shown to impair attention and tracking in a wide variety of studies. However, recently introduced tranquilizers, such as buspirone, exhibit little evidence of impairment.

Conclusions about impairments in a drug category are likely to change because of the pressures exerted by the drug regulatory agencies on drug companies to develop medicines that do not impair skills performance. For example, hypnotics often exhibit residual skills impairment the day following use. New drugs have been introduced whose duration of effects is shorter so there will be less residual impairment after awakening.

Another class of psychoactive drugs, the ANTIDEPRESSANTS, especially amitriptyline, have been long known to impair performance in a variety of skills. Again, recently introduced types of antidepressants do not produce the same degree of impairment.

Although narcotic ANALGESICS derived from OPIUM (OPIATES) have been shown experimentally to lead to decreased alertness, there have been reports that chronic use produces considerable tolerance to some of these side effects, which may explain why epidemiological studies have not found differences in crash rates between NARCOTIC users and control groups. Moreover, patients maintained on a stabilized dosage of METHADONE, a synthesized narcotic, have shown little evidence of impairment in a wide variety of experimental and epidemiological studies.

Another category of drug that shows evidence of impairing skills performance in laboratory studies is the antihistamines, many of which produce impairment of performance accompanied by complaints of drowsiness and lack of alertness. Again, recent pharmacological advancements have produced antihistamine drugs, like loratadine (Claritin), which maintain antihistamine actions but have difficulty crossing the blood-brain barrier and thus produce little impairment.

Of all illicit drugs, marijuana has had the largest number of experimental studies performed to examine its effects. Many of these studies indicate that marijuana impairs coordination, tracking, perception, and vigilance, as well as performance in driving simulators and on-the-road studies.

Although there has been concern over increased driver use of STIMULANTS, such as amphatamines and cocaine, there is little experimental evidence demonstrating driving impairment by these drugs. On the contrary, most studies of these stimulants, as well as of CAFFEINE, indicate an improvement in skills performance. However, with the chronic (long-term) use of stimulants, an increased dose must be taken as tolerance develops. Thus, the dose levels examined in the laboratory may not reflect those found among drivers. Second, after the stimulation phase, a subsequent depressed phase occurs (the "crash") with increased drowsiness and lack of alertness. The stimulant drugs have not been well studied in relation to driving and should be. Further study is needed—both for acute (one-time) use and chronic use.

(SEE ALSO: *Dramshop Liability Laws: Drunk Driving; Minimum Drinking Age Laws; Mothers Against Drunk Driving; Psychomotor Effects of Alcohol and Drugs; Social Class of Alcohol and Drug Abuse; Students Against Destructive Decisions*).

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HERBERT MOSKOWITZ

DRIVING UNDER THE INFLUENCE (DUI) DRIVING UNDER THE INFLUENCE is a term that refers to the operation of a motor vehicle after consuming alcohol and being affected by it in some way. It may be used as a legal term denoting a lesser offense than DRIVING WHILE INTOXICATED (DWI). Specific blood-alcohol concentration (BAC) limits are associated with a DUI offense. These vary among states and countries but are often between .05 percent and .10 percent (50 milligrams per deciliter [mg/dl] and 100 mg/dl). In the United States, most states place the limit at .010 percent to be classified as driving under the influence. Some states have reduced the legal limit to 0.08 percent, but Congress rejected legislation in 1998 that would have required all states to lower the drunken driving arrest threshold to .08 percent.

There is a strong correlation between a BAC greater than 0.05 percent and risk of serious injury or death while operating a motor vehicle. After the BAC reaches .08 percent or more, the probability of a crash climbs rapidly. The National Highway Traffic Safety Administration (NHTSA) estimates that in 1998, alcohol was involved in 39 percent of all fatal crashes (almost 16,000 fatalities) and 7 percent of all crashes. NHTSA estimates that three out of ten Americans will be involved in an alcohol-related crash sometime during their lives.

Four of the 40 officers at a roadblock in Cary, North Carolina, stop cars to check for drunk drivers on December 13, 1997. During the Saturday night sweep, officers statewide charged 69 people with DWI and seized six cars. (AP Photo/Karen Tam)

(SEE ALSO: *Breathalyzer; Dramshop Liability Laws; Driving, Alcohol, and Drugs; Drug Interactions and Alcohol; Drunk Driving; Mothers Against Drunk Driving; Students Against Destructive Decisions*)

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DROPOUTS AND SUBSTANCE USE The Monitoring the Future project (HIGH SCHOOL SENIOR SURVEY) and other studies of school-age youths have helped us to understand the substance-use patterns of ADOLESCENTS who remain in and graduate from high school. In contrast, not nearly

as much is known about the substance use of those who become high school dropouts. Nonetheless, by putting together evidence from a variety of sources, including the NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE and the Epidemiologic Catchment Area surveys sponsored by the U.S. government, it is possible to say that high school dropouts are much more likely to have started using TOBACCO, ALCOHOL, and other drugs, as compared with their peers who remained in school. There also is some evidence that dropping out of high school is associated with an increased risk of adult-onset alcohol-dependence syndromes, even among persons whose dropping out could not have been caused by the consequences of starting to drink during the adolescent years. Whether this conclusion also holds for adult-onset DEPENDENCE on other drugs such as COCAINE or MARIJUANA is not yet clear but is under study.

In trying to understand how it might happen that dropouts are more likely to be substance users, the possibility should be considered that substance use has caused some people to drop out of school, as well as the possibility that some schools suspend or expel students for smoking tobacco, drinking alcohol, or using other drugs. By themselves, these circumstances could be enough to explain why high school dropouts are more likely to have taken illicit drugs or started underage smoking or drinking.

In addition, when students drop out before graduating from high school, they often begin spending more time with older youths and adults, some of whom serve as role models for substance use and who may give the dropouts cigarettes or offer them opportunities to try alcohol or other drugs for the first time. As a result, not only is there the possibility that substance use may lead to dropping out, but it is also possible that dropping out may lead to substance use.

More complicated possibilities must also be taken into account. A developmental perspective makes it possible to imagine that the greater frequency of substance use among high school dropouts might have its origins in the earlier years of childhood, so that it is not a simple matter of substance use leading to dropping out, or dropping out leading to substance use. For example, it has been found that youngsters who frequently broke rules, got into fights, and had trouble adapting to elementary school were more likely to become heavy drug users ten or more years later. CONDUCT problems at school are other signs that help to predict who will drop out before completing high school. This provides some evidence that sometimes there can be an underlying common cause in earlier stages of growth and development and that it is essential to consider these earlier stages in observing a link between later substance use and dropping out of school.

Working along these lines, Judith Brook and her research team have looked into school grades and poor school achievement with the idea that students who did not do well in elementary school might be at greater risk for later drug use, in the same way that not doing well in school was a sign of greater risk for dropping out. Her research group also sought to determine whether later improvements in academic performance might modify the risk profiles of low achievers in primary school.

In studying a large sample of school-age youth from primary school through high school, this team of researchers has found a moderately strong linkage between early poor school achievement and later drug use, but also discovered that the linkage was substantially weaker when low achievers in primary school did much better in later school years. This type of developmental evidence is important and needs to be replicated by others before strong conclusions can be drawn. It does suggest that it might be possible to use achievement-strengthening programs not only to reduce school dropout rates but also to reduce rates and levels of teenage drug use.

Several research groups are carrying out rigorous field experiments to see whether intervention programs directed at entire classrooms of first- and second-graders might change their risks for later drug use, conduct problems, and dropout rates. These very early interventions are not drug-education classes. The first- and second-grade teachers are working with the students to help promote their learning and school achievement in new ways and to help them behave themselves and adapt better to the rules of the elementary school classroom.

Other research groups are trying to reduce dropout rates and substance use by targeting the more vulnerable or higher-risk elementary school and middle school students, and giving them and their FAMILIES special programs to promote learning and a sense of mastery over schoolwork; sometimes this is being done in connection with social-influences intervention programs. In contrast with interven-

tions directed at all students in the first- and second-grade classrooms, these involve "pull-out" programs for the specially targeted higher-risk students.

Future research will provide more definitive evidence on the underlying mechanisms that account for observed associations between dropping out of high school and substance use, as well as for the newer suspected associations between dropping out of school and the risk for adult-onset alcohol-dependence syndromes (Crum et al., 1993). If the intervention programs are found to reduce dropout rates and also levels of alcohol, tobacco, and other drug involvement, this will provide some powerful evidence on the causal significance of the early developmental antecedents and will help to explain why dropouts are more likely to be drug users.

In the meantime, the broad range of unfortunate effects of dropping out of school makes it important to sustain and increase the vigor of stay-in-school programs as well as outreach programs for youths who are chronically absent from school or who actually have dropped out before graduation. These programs may help the individual youths, their families, and society in many ways; they may not only confer benefits in relation to schooling and better preparation for adult life, but also reduce the amount of substance use in the teenage years, prevent the occurrence of alcohol and drug problems in adulthood, and possibly prevent other psychiatric disorders such as major DEPRESSION.

(SEE ALSO: *Attention Deficit Disorder; Coping and Drug Use; Education and Prevention; Epidemiology of Drug Abuse; Vulnerability As Cause of Substance Abuse*)

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DRUG As a therapeutic agent, a drug is any substance, other than food, used in the prevention, diagnosis, alleviation, treatment, or cure of disease. It is also a general term for any substance, stimulating or depressing, that can be habituating. According to the U.S. Food, Drug, and Cosmetic Act, a drug is (1) a substance recognized in an official pharmacopoeia or formulary; (2) a substance intended for use in the diagnosis, cure, mitigation, treatment, or prevention of disease; (3) a substance other than food intended to affect the structure or function of the body; (4) a substance intended for use as a component of a medicine but not a device or a component, part, or accessory of a device.

Pharmacologists consider a drug to be any molecule that, when introduced into the body, affects living processes through interactions at the molecular level. Hormones can be considered to be drugs, whether they are administered from outside the body or their release is stimulated endogenously. Although drug molecules vary in size, the molecular weight of most drugs falls within the range of

100–1,000, since to be a drug it must be absorbed and distributed to a target organ. Efficient absorption and distribution may be more difficult when drugs have a molecular weight greater than 1,000. The drug's molecular shape is also important, since most drugs interact with specific RECEPTORS to produce their biological effects. The shape of the receptor determines which drug molecules are capable of binding. The shape of the drug molecule must be complementary to that of the receptor to produce an optimal fit and, therefore, a physiological response.

Within this general definition, most POISONS would be considered to be drugs. Although water and oxygen technically fit this general definition and are used therapeutically and discussed in pharmacology textbooks, they are rarely considered to be drugs. Efforts have been made to develop a more restricted definition, but because so many molecules and substances can affect living tissue, it is difficult to draw a sharp line.

(SEE ALSO: *Inhalants; Plants, Drugs from; Vitamins*)

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DRUG ABUSE *See* Addiction: Concepts and Definitions

DRUG ABUSE REPORTING PROGRAM (DARP) The Drug Abuse Reporting Program began in 1969 as a comprehensive data system that included intake and during-treatment information on individuals entering drug treatment programs funded by the U.S. government. Over time, it was the basis for carrying out the first national evaluation study of community-based treatment programs. It was conducted at Texas Christian University over a period of twenty years and included four distinct phases of research: (1) describing major treatment modalities and the characteristics of drug abusers entering them in the early 1970s; (2) describing during-treatment performance measures and how they related to differences in treatments and clients; (3) describing post-treatment outcomes and how they related to differences in treatments and clients; and (4) describing important elements of long-term addiction careers.

The DARP data system contained records on almost 44,000 admissions to fifty-two federally supported treatment agencies from 1969 to 1973—the years during which the current community treatment delivery system first emerged in the United States. The study population consisted of clients from major TREATMENT modalities—METHADONE MAINTENANCE PROGRAMS, THERAPEUTIC COMMUNITY, outpatient drug-free, and DETOXIFICATION—as well as a comparison intake-only group.

THE EFFECTIVENESS OF DRUG-ABUSE TREATMENT

Initial research in this 20-year project focused on ways of measuring characteristics of treatments, clients, and behavioral outcomes (see Sells & Associates, 1975). It was found that drug use and criminal activities decreased significantly during treatment, including outpatient as well as residential programs. More important, the effects continued after treatment was ended. A sample of 6,402 clients located across the United States were selected for follow-up an average of three years after leaving DARP treatment (and 83% were located). Methadone maintenance, therapeutic communities, and outpatient drug-free programs were associated with more favorable outcomes among opioid addicts than outpatient detoxification and intake-only comparison groups; however, only clients who remained in treatment three months or longer showed significant improvements after treatment. Numerous studies of these data helped establish that treatment "works" and that the longer clients stay in treatment, the better they function after treatment (see Simpson & Sells, 1982).

LONG-TERM OPIOID ADDICTION CAREERS

To study long-term addiction careers, a sample of 697 daily OPIOID (primarily HEROIN) users were followed up with again, at twelve years after enter-

ing treatment (and 80% were located). It was found that about 25 percent of the sample was still addicted to daily opioid use in year twelve. Length of addiction (defined as the time between first and last daily opioid use) ranged from one to thirty-four years. Of the total sample, 50 percent was addicted nine-and-a-half years or longer, yet 59 percent never had a period of continuous daily use that exceeded two years. Only 27 percent reported continuous addiction periods that lasted more than three years.

Three-fourths of the addicts studied had experienced at least one "relapse" to daily opioid use after they had temporarily quit. Among those who had ever temporarily quit daily opioid use at least once, 85 percent had done so while in a drug-abuse treatment, 78 percent had quit while in a jail or prison, 69 percent had temporarily quit "on their own" (without treatment), and 41 percent had quit while in a hospital for medical treatment. The most frequent reasons cited for quitting addiction the last time involved psychological and emotional problems. Ex-addicts reported they had "become tired of the hustle" (rated as being important by 83% of the sample) and needed a change after "hitting bottom" (considered important by 80%). Other reasons cited as being important were "personal or special" events such as a marriage or the death of a friend (64%), fear of being sent to jail (56%), and the need to meet family responsibilities (54%) (See Simpson & Sells, 1990, for further details).

SUMMARY

The DARP findings have been widely used to support continued public funding of drug-abuse treatments and to influence federal drug policy in the United States. Other similar national treatment evaluation studies have been planned and undertaken at the beginning of each decade since the 1970s. Current research efforts focus on increasing understanding of the particular elements of treatment that are most effective and how they can be improved.

(SEE ALSO: *Drug Abuse Treatment Outcome Studies; Narcotic Addict Rehabilitation Act*)

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DRUG ABUSE TREATMENT OUTCOME STUDIES (DATOS) This family of studies is designed to provide comprehensive information on continuing and new questions about the effectiveness of the drug-abuse treatment that is available in a variety of publicly funded and private programs. These data update and augment the information available from earlier national studies, such as the DRUG ABUSE REPORTING PROGRAM (DARP) study, which began in the late 1960s, and the TREATMENT OUTCOME PROSPECTIVE STUDY (TOPS) of clients entering treatment in the 1970s. The work was sponsored by the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) and conducted by the National Development and Research Institute, Texas Christian University, and the University of California, Los Angeles.

The major objective of DATOS is to examine the effectiveness of drug-abuse treatment by conducting a multisite, prospective clinical and epidemiological longitudinal study of drug-abuse treatment. Effectiveness was examined by using data from 10,010 client interviews conducted from 1991 to 1993 at entry to treatment each three months, during treatment, and one year after leaving treatment. Interviews of clients at admission were supplemented with comprehensive clinical assessments of psychological, social, and physical impairments in addition to drug and alcohol dependence. Treatment outcomes were compared for clients who entered treatment with varied patterns of drug abuse and levels of psychosocial impairment.

A secondary objective was to investigate the process of drug-abuse treatment. A detailed examination was being conducted of the treatments and services available and provided to each client, how

these treatments and services are delivered to the client, and how the client responds to treatment in terms of cognitive and behavioral changes.

The study population includes 1,540 clients from twenty-nine outpatient methadone programs; 2,774 clients from twenty-one long-term residential or THERAPEUTIC COMMUNITY programs; 2,574 clients from thirty-two outpatient drug-free programs; and 3,122 clients from fourteen short-term, inpatient, or chemical dependency programs. In addition, treatment programs from DATOS were compared with those for TOPS a decade earlier and for DARP two decades earlier in order to determine how drug-abuse treatment programs have changed and what the changes imply for the provision of effective treatment approaches and services.

The DATOS research builds and expands the knowledge generated from previous research on the effectiveness of treatment. Several events, however, have necessitated a continuing nationally based multisite study of drug-abuse treatment and treatment effectiveness. Major changes have occurred in the nation's drug-abusing population and treatment system. The OMNIBUS Recommendation Act of 1981 shifted the administration of treatment programs from the federal government to the states. The AIDS epidemic has intensified interest in drug-abuse treatment as a strategy to reduce exposure to the HUMAN IMMUNODEFICIENCY VIRUS (HIV), which causes AIDS. COCAINE use rather than OPIOID use was the major drug problem of the late 1980s and early 1990s. Efforts to contain health-care costs may dramatically transform both the public and private treatment systems. It has therefore become necessary to update information so as to reexamine what we have learned about treatment effectiveness, and so as to augment the types of data that are available for exploring new issues about the nature, effectiveness, and costs of treatment approaches. The research based on DATOS has been organized into four areas: (1) treatment selection, access, and utilization, (2) treatment engagement and retention, (3) addiction and treatment, and (4) applications and policy development. Details of these studies can be found in the following references as well as at the URL http://www.DATOS.org.

The initial comparison of data from the DATOS and from the TOPS shows the following about the clients in DATOS: They are older, a greater percentage of them are women, they have more years of education, more of them are married, fewer are fully employed, and a lower percentage of them report that they have considered or attempted suicide. A higher proportion of the clients on METHADONE are entering treatment for the first time, and the proportion of criminal-justice referrals is higher in long-term residential and outpatient drug-free programs than they are in short-term inpatient programs.

In all types of programs, cocaine abuse predominated in the early 1990s, compared to heroin abuse in the past, but the cocaine use was usually combined with extensive use of ALCOHOL. Multiple abuse of psychotherapeutic agents has decreased, so less than 10 percent of clients report that they regularly use these agents as opposed to treatment services. Outpatient programs have fewer early dropouts, but this may reflect better screening and longer, more extensive intake processes. The influence of cost-containment measures and managed care became evident with shortened durations of treatment for short-term inpatients. Short-term inpatient programs also admit more public sector patients than in the 1980s. Early analyses from the DATOS indicate that rates of drug use toward the start of outpatient treatment are two to three times higher than those that were found in TOPS.

The early results of DATOS also show that clients entering drug treatment are a diverse group who have multiple problems. Two-thirds of the clients are men, and approximately half have previously been in treatment. Those who have health insurance that covers treatment are, by far, in the minority. Although the clients entering short-term inpatient chemical-dependency programs appear to have a higher rate of private insurance coverage (40 percent) than any other classification of clients, their rate is considerably lower than that observed among the same type of clients in the 1980s. Depending on the type of treatment, 25 to 50 percent of clients reported predatory criminal activity in the previous year, and less than 20 percent were fully employed. The clients have a variety of health problems, and many report significant psychiatric impairment. Few have received mental health services, however, and about one in every three clients report that they use emergency rooms as their primary health-care provider. Taken together, these data indicate that most clients have deficits in many areas of their lives and have multiple needs in

addition to those directly related to their drug abuse (e.g., medical services and vocational needs).

Patterns of drug use vary markedly by type of treatment in DATOS clients. Although cocaine is the most frequently cited drug of abuse, most clients abuse multiple drugs and exhibit complex patterns of drug use. Frequent alcohol use is also common among many of the clients, as is weekly use of marijuana. Multiple abuse of psychotherapeutic agents is reported by less than 10 percent of clients.

Drug-treatment programs are focusing on providing drug-counseling services to meet the multiple problems of clients, but fewer specialized services, such as medical or psychological services, are being provided to meet clients' other needs. Only a third to a half of clients who report a need for medical services are receiving them, and the situation is much worse in regard to psychological, family, legal, employment, and financial services. Less than 10 percent of clients who report a need actually receive the service while they are in methadone and outpatient drug-free programs. The percentage of clients who receive specialized services other than drug counseling (e.g., medical or psychological attention) has declined dramatically since the mid-1980s. The impact of cost-containment measures and managed care is evident in the shorter stays of clients, particularly those enrolled in short-term inpatient programs.

Limited information on treatment outcomes has provided a mixed indication of the outcomes of treatment. The combination of more severe impairment and less extensive services suggests the potential for poorer outcomes. On the positive side, clients in treatment are being retained in treatment. However, compared to earlier findings, findings from the early 1990s indicated that a higher percentage of clients were actively using drugs during the first months of treatment.

Two other studies have been included under the DATOS program of research. The Drug Abuse Treatment Outcome Study–Adolescent (DATOS–Adolescent) research study is designed to examine the effectiveness of drug-abuse treatment for adolescents through a multisite prospective longitudinal study of youth entering treatment programs that focus on adolescents. Effectiveness will be examined by using interview data from youth under eighteen supplemented by interviews with parents or guardians conducted at entry to treatment, during treatment, and one year after leaving treatment. Treatment outcome will also be assessed by using such measures as changes in the use of the primary problem drug; the use of other drugs; antisocial, delinquent, or criminal behavior; school attendance and achievement; vocational training and employment; family and social functioning; and treatment retention. A secondary objective of DATOS–Adolescent is to investigate the drug-abuse treatment that adolescents receive. A sample of thirty long-term residential, outpatient drug-free, and short-term inpatient programs will be used to accomplish these objectives. The proposed sample design will include three thousand clients.

The Early Retrospective Study of Cocaine Treatment Outcomes is an accelerated retrospective study of clients with a primary diagnosis of cocaine dependence who had been admitted to DATOS–Adult programs prior to or in the early stages of DATOS. The research will provide data about outcomes for cocaine abusers during the first year of treatment, describe the treatment received by these clients through a study of treatment process, and establish a client data base for future follow-up studies. A sample of 2,000 records for cocaine-abusing clients discharged from residential, hospital-based, and outpatient nonmethadone programs will be abstracted to obtain baseline data. A sample of 1,200 face-to-face, follow-up interviews will be completed, after twelve months of treatment, with the discharged clients whose records were reviewed during the record-abstraction phase of the project.

Data analyses of the project will be targeted at describing the posttreatment outcomes for cocaine abusers by detailing their treatment experiences and investigating their posttreatment experiences. This description will include the type, intensity, and duration of services received and an examination of the interrelationships between client and treatment characteristics and posttreatment outcomes. Along with cocaine use, other outcomes that will be considered include the use of drugs other than cocaine, economic functioning, illegal activities, and psychological status. Analytic methods will include univariate and descriptive statistics as well as multivariate methods. The collection of follow-up data for this retrospective study will be conducted simultaneously with the collection of data for the twelve-month follow-up of DATOS clients. A close coordination with the DATOS–Adult is designed to permit comparison across studies.

(SEE ALSO: *Drug Abuse Reporting Program; Methadone Maintenance Programs; Opioid Dependence; Treatment, History of in the United States; Treatment: Outcome Prospective Study; Treatment Types*)

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DRUG ABUSE WARNING NETWORK (DAWN) This is a voluntary national data collection system that gathers information on substance abuse that results in visits to hospital emergency departments (ED) in the contiguous United States. Hospitals tracked in the DAWN system include nonfederal, short-stay, general hospitals that have twenty-four-hour ED—representative of the coterminous United States as a whole—plus a sample of hospitals in twenty-one major metropolitan areas. The system also collects data on drug-related deaths from a nonrandom sample of medical examiners. Such data are published annually in separate reports titled DAWN Medical Examiner Data.

The data collected by the DAWN system represent one of the most widely used national indicators of drug abuse—frequently used by researchers and policymakers to determine the nature and extent of medical consequences of drug use nationally and in the participating metropolitan areas. Although the data are widely used to monitor patterns and trends of drug abuse, DAWN should be used with caution. The DAWN data represent information only on individuals who enter an emergency room because of their drug use. Therefore, the data reflect only the most serious cases of drug abuse. Consequences of drug use that are less severe are not represented in the data. In addition, analysis of the data requires familiarity with the types of cases reportable to DAWN. For example, the emergency-room system contains data not only on OVERDOSE cases, but on individuals seeking DETOXIFICATION in an emergency room or those suffering from chronic effects of drug use—situations that do not necessarily require emergency treatment. Therefore, DAWN cases may reflect other phenomena than medical consequences (e.g., changes in nonhospital medical treatment availability).

HISTORY

DAWN was created in 1972 by the U.S. Department of Justice, DRUG ENFORCEMENT ADMINISTRATION (DEA), as a surveillance system for new drugs of abuse. Since 1980, the DAWN system has been managed by the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) and, more recently, the Office of Applied Studies of the SUBSTANCE ABUSE AND MENTAL HEALTH SERVICES ADMINISTRATION (SAMHSA), which currently oversees it.

The Emergency-Room System. At its outset, the DAWN emergency-room system consisted of a sample of hospitals in twenty-one metropolitan areas and a panel of hospitals representative of the nation as a whole. In subsequent years, however, changes in hospital participation led to a sample that was no longer representative of all hospital EDs in the coterminous United States. Thus, data collected by the DAWN system before 1988 measure drug-related cases only for those hospitals included in the sample; actual estimates of the number of drug deaths and hospital emergencies in the metropolitan areas or across the nation are not available for those years (1972–1987). The primary utility of the data prior to 1988, then, is for examining trends in drug emergencies and deaths in the participating hospitals over time, rather than deriving estimates of actual numbers of cases in the United States.

DAWN data have been collected from a representative sample of eligible hospitals in the coterminous United States. It includes an oversampling from twenty-one metropolitan areas and a National Panel of hospitals sampled from locations outside of these areas. This sample design corrects the limitations of the pre-1988 sample. It also allows for inferences about the actual number of drug-abuse episodes in the contiguous United

States and for the separate DAWN metropolitan areas.

DATA COLLECTION

In each participating facility, a reporter, usually a member of the hospital ED or medical records staff, is assigned to conduct data collection. The reporter reviews records for each case appropriate for inclusion in the DAWN system and records demographic information and information about the circumstances of the episode, including the date and time of the ED visit and the reason the person came to the ED. For each drug mentioned, the DAWN report includes the form in which the drug was acquired, its source, and its route of administration. Only one reason for taking substances is recorded. A report for each case is then submitted to SAMHSA for data entry.

The following criteria are used in determining whether a case is reportable to DAWN. For each record, the patient must be between six and ninety-seven years of age and must meet all of the following criteria:

1. The patient was treated in the hospital's emergency department.
2. The patient's presenting problem(s) had been induced by or related to drug use, regardless of whether the drug ingestion occurred minutes or hours before the visit.
3. The case involved the nonmedical use of a legal drug or any use of an illegal drug.
4. The patient's reason for taking the substance(s) included one of the following: (1) DEPENDENCE, (2) SUICIDE attempt or gesture, or (3) psychic effects.

DAWN cases do not include accidental ingestion or inhalation of a substance, nor do they include adverse reactions to prescription or over-the-counter drugs. Up to four substances can be reported for any drug-abuse death or emergency-room case. Alcohol is reportable only when it is used in combination with a reportable substance.

TRENDS IN DRUG-RELATED EMERGENCIES AND DEATHS

Drug-related Episodes in 1999. In 1999, there were an estimated 554,932 drug-related ED episodes in the coterminous United States. Episodes from males and females occurred in about equal numbers.

Dependence (37% of episodes) and suicide (32%) were the most frequently cited motives for drug use. When analyzed by reason for contact in the emergency room, the data show that overdose (OD) was the most frequently cited reason for the majority of episodes (42%), while the remainder of episodes were due to another cause.

In 1999, the largest number of episodes (196,277, or 35% of all episodes) were due to use of ALCOHOL in combination with other drugs. The other drugs mentioned most frequently were COCAINE (168,763, or 30%), HEROIN/MORPHINE (84,409, or 15%), amphetamine (11,954, or 2%), and methamphetamine/speed (10,447, or 2%). In 1999, marijuana/hashish mentions exceeded heroin/morphine mentions, changing a rank ordering of illicit drug mentions that had been constant since 1990.

Long-term Trends, 1990–1999. The number of drug-related episodes rose 49 percent from 1990 to 1999, from 371,208 to 554,932. Although males consistently outnumber females in illicit drug mentions, their long-term patterns of growth are similar. From 1990 to 1999, mentions of cocaine and heroin/morphine more than doubled for both males and females. ED mentions of marijuana/hashish in 1999 were five and six times their 1990 levels for males and females, respectively. Mentions of the four major illicit drugs increased from 1990 to 1999 as follows: marijuana/hashish (15,706 to 87,150, or 455%), methamphetamine/speed (5,236 to 10,447, or 100%), heroin/morphine (33,884 to 84,409, or 149%), and cocaine (80,355 to 168,763, or 110%). Emergencies for those over age thirty-five rose significantly over the time period (124%), while the number of episodes for those twenty-five and under increased less than 20 percent.

Among adolescents age twelve to seventeen, mentions of marijuana/hashish increased 489 percent (from 2,170 to 12,784) between 1990 and 1999. This is increase is significant, considering that the number of total drug-related episodes among patients in this age group increased only 7 percent (from 49,109 to 52,783) between 1990 and 1999. In addition, the long-term trends for methamphetamine/speed, cocaine, and heroin/morphine among youth aged twelve to seventeen

show increasing use among individuals in this group.

(SEE ALSO: *Abuse Liability of Drugs; Complications; Drug Interactions and Alcohol; Epidemiology of Drug Abuse; National Household Survey on Drug Abuse*)

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DRUG ADDICTION *See* Addiction: Concepts and Definitions; Disease Concept of Alcoholism and Drug Abuse

DRUG CARTEL *See* Colombia as Drug Source

DRUG CONTROLS *See* Controls: Scheduled Drugs/Drug Schedules, U.S.

DRUG COUNSELOR *See* Professional Credentialing

DRUG COURTS Drug courts emerged as a method for responding to America's drug problems at a time when health, treatment and justice systems were overwhelmed by the drug epidemics of the 1980s. The dramatic increase in the availability of cocaine and, later, crack cocaine, particularly in America's cities, translated into a new challenge for the criminal justice system that was already at its limits. The volume of court cases exploded, pushing the judicial process to its limits and threatening traditional modes of managing the criminal caseload. Worse, the huge wave of arrests of drug offenders beginning in and accelerating during the 1980s found a correctional system of local jails and state prisons in many locations in the nation that were already chronically overcrowded. With little room in prisons for the new arrestees, institutional crowding was exacerbated and the processing of criminal cases was slowed, causing backlogs in the courts and a wide range of problems for the justice system as a whole.

In Miami, at the gateway of major drug trafficking routes from South America, the drug crisis was particularly acute. A study had shown that approximately 90 percent of felony defendants entering the judicial process in Miami (Metropolitan Dade County) tested positively for drugs (excluding alcohol) at the time of their arrest. With historical hindsight, the innovation of the Miami Drug Court by its justice leaders (including its chief judge, Gerald Wetherington, its prosecutor, Janet Reno, its public defender, Bennet Brummer, and its control drug control administrator, Timothy Murray) seems obvious and common-sensical. The court system and government leaders reasoned that, if Dade County could not arrest and punish its way out of the drug problem, perhaps it should try providing treatment as a reasonable alternative to prosecution and confinement.

The first drug court in the United States went into operation in Miami under the supervision of Judge Stanley Goldstein, the nation's first drug court judge, in the summer of 1989. Since the breakthrough efforts of the Miami justice leaders, by all measures, the growth of treatment drug courts in the United States has been extraordinary, with upwards of 400 courts reportedly in operation in the year 2000 and others in some stage of planning or preparation. The drug court model has also been adapted in other countries, from Canada and Australia to Great Britain and Ireland.

Taken at its most challenging and as envisioned by its most ardent proponents, the drug court model potentially represents the first stages of a fundamental paradigm shift in justice away from a predominantly punitive orientation (a.k.a. "justice

as usual'') toward an approach that seeks to confront and meliorate the problems associated with persons who appear in the criminal caseload. The challenges implicit in this approach are fundamental and draw into the criminal court setting expertise from health and behavioral sciences as well as linkages with a variety of social services in relationships and configurations that produce a new mix of values, aspirations and methods to guide the judicial process. To their proponents, drug courts represent a major and promising departure from what had become an unrewarding routine of processing, punishing and re-punishing drug offenders to little avail. Instead, the drug court model takes on ''root causes'' of crime more easily ignored or viewed as someone else's responsibility.

The foundation of the drug court model lies in its underlying values, philosophical outlook, and the central role it assigns to the judge, as it incorporates a mix of values with a decided shift toward treatment and restoration of offenders to the community. The mix also includes deterrent and desert values that realistically circumscribe the arena of the drug court modality: it is transacted in a criminal (usually felony) courtroom. The therapeutic activities associated with the treatment-oriented drug court occur in a ''theater in the square,'' the square representing not only the architectural features of the physical courtroom but also the boundaries imposed by the criminal process. Led and closely supervised by the drug court judge, the drug court operates in the context of the criminal process and, therefore, differs notably from substance abuse treatment that might be provided to addicted citizens in civilian contexts outside of the justice system. Within those judge-enforced boundaries marking the criminal process, however, the drug court model has innovated a new working relationship between the criminal court and health, treatment and related services that adapts the criminal process to the needs of treatment and an understanding of addiction. The drug court seeks to resolve the apparently contradictory aims of the typically punitive justice process and the more supportive treatment process.

Until recently, the likelihood that an offender identified as having a serious drug problem would be placed in treatment was poor and depended on being convicted and, usually, sentenced to probation. (Only in rare settings would a sentence to incarceration include the realistic possibility that drug treatment would be available.) For many decades, drug treatment has also been available as a condition of diversion for less serious offenses but has not demonstrated significant impact. From a judicial perspective, however, the court's typical involvement in substance abuse treatment was to order or ''refer'' offenders ''out'' to treatment, when such treatment was recommended by probation staff at the pre-sentence stage. The judge in such cases would have little other involvement in the treatment process, except to set treatment as a condition of probation, and, later, hear allegations of noncompliance at revocation hearings. By keeping a judicial distance from the treatment process, judges deferred to the expertise and practices of treatment providers and probation agencies whose responsibilities were to manage and monitor the treatment process.

Drug courts were ''invented'' to reinvent a helping justice role, similar to the one formerly played by probation services, but this time entrusted to the power, authority, symbolism and centrality of the criminal court judge and occurring at an earlier stage. Many early drug courts consciously excluded probation departments from their drug court design, although there were some notable exceptions (e.g., Maricopa County, Baltimore, and Oakland). While early drug courts ultimately found top-notch treatment providers willing to craft customized approaches to fit the needs of the drug courts, under former practices, treatment providers operated under their own rules and discretion in determining eligibility, level of care and termination, which reflected a different professional orientation and view of how substance abusers should be treated.

Previously there was little judicial input into the content of treatment programs and little two-way communication between the court and the treatment provider, except when the program needed to notify the court of an individual's completion of treatment or failure to comply with the requirements of the treatment process. Judges delegated the responsibility for treatment and supervision to probation and treatment providers. Under the ''refer-out'' model, treatment providers controlled admission screening (some resisted accepting criminal justice clients), the level of care to be provided (the mix and location of services, from outpatient to inpatient and including ancillary services), and the termination process. Typically, treatment providers could discharge an ''uncooperative'' client

that was having a difficult time fulfilling the conditions of the program.

The drug court innovation sought to build the new approach based on a "hands-on" and engaged judicial role, a strong supervisory and case management approach (initially not necessarily involving probation), agreed upon, acceptable and relevant treatment services, and a more connected relationship with treatment providers in a court-treatment process in which the judge controlled the admission and termination criteria. Thus, by design under the drug court model, the drug court treatment program could not, on its own, reject difficult criminal justice clients accepted into the drug court and could not, without judicial approval, terminate participants when they had failed to comply with treatment program requirements.

Within these general elements of the treatment-oriented philosophy, the central judicial role and new criminal court-treatment relationship, drug courts are characterized by other distinguishing elements. The Drug Court Program Office of the U.S. Department of Justice sponsored an initiative by the National Association of Drug Court Professionals (NADCP) to identify key components of drug courts. The ten components identified by NADCP, adopted as a standard by the Justice Department in reviewing grant applications, include integration of treatment and case processing; a non-adversarial approach which also respects due process and public safety; early identification and enrollment of participants; provision of a continuum of treatment services; drug testing; court responses to performance in treatment; hands-on judicial supervision of treatment; monitoring and evaluation; continuing interdisciplinary education; and, forging partnerships between the court and other criminal justice, health, social service agencies and the community.

Prior to the NADCP practitioner-oriented process to identify key components of drug courts for the purposes of constructing standards, a working typology of drug courts identified eight critical dimensions of the drug court innovation mainly for the purposes of evaluation. These include the target problems drug courts were designed to address, specific criminal justice target populations they sought to enroll in treatment, mechanisms employed to identify and evaluate court treatment candidates, the ways in which they involved modifications to the traditional court process, the structure and content of the treatment delivered to substance abusing offenders, the methods employed in the drug courts to encourage positive and discourage negative behavior by participants (including the use of sanctions and rewards), the productivity of the courts (in terms of measurable outcomes such as reduced substance abuse and criminal behavior), and the extent of system-wide support in and outside criminal justice and health systems.

Although there are common elements shared by most drug courts, proliferation of the drug court model is not explained by the wholesale adoption of a fixed, "cookie-cutter" approach in the many jurisdictions across the nation. The original Miami model evolved in its successive adaptations in other settings, and was itself transformed in substance and procedure as the basic model traveled across the United States and to locations abroad. The drug court methodology has been adapted to grapple with other problems associated with court populations, including community issues, domestic violence and mental health and has directly and indirectly spawned a variety of related innovations, so that one can now speak of "problem-solving" or "problem-oriented" courts to refer to a more active, "hands-on" judicial and justice-system philosophy.

The rapidly growing volume of drug courts (as well as of other "problem-solving" courts) suggests that nationally the drug court experiment struck a fundamental chord of dissatisfaction with traditional justice machinery that seemed only to punish and process. A number of states (California, New York, Louisiana, Ohio, Florida), large counties (Los Angeles County, Clark County, Nevada), and large urban centers (Miami; Brooklyn; Buffalo; Portland, Oregon; Seattle) have incorporated drug courts into their administrative and budgetary planning processes because their growing numbers raise questions for court systems as a whole about priorities, resources, effective management, and performance standards.

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DRUG ENFORCEMENT ADMINISTRATION *See* U.S. Government

DRUG-FREE SCHOOLS *See* Parents Movement; Prevention Movement

DRUG INTERACTION AND THE BRAIN

When two or more drugs are taken at the same time complex interactions may occur. Drugs can interact to change biological functions within the body through PHARMACOKINETIC or PHARMACODYNAMIC mechanisms or through their combined toxic effects. Changes in the pharmacokinetic properties of a drug can include changes in absorption, distribution, metabolism, and excretion of the drug, and each of these can affect blood and plasma concentrations and, ultimately, brain levels of the drug. Although a change in the speed at which a drug reaches the bloodstream is rarely clinically relevant, a change in the amount of drug absorbed can be important, because this can lead to changes in the plasma levels of the drug, which can influence the amount of drug that reaches the brain.

The distribution of a drug throughout the body can be affected by changes in the binding of the drug to proteins in the bloodstream or by displacing the drug from tissue binding sites, both of which can affect the plasma concentration of the drug and potentially affect the amount of drug that reaches the brain. Drug metabolism can be either stimulated or inhibited, resulting in decreased or increased plasma concentrations of the drug, respectively. The stimulation (induction) of drug-metabolizing enzymes in the liver can be produced by drugs such as the BARBITURATES, but a week or more is often required before maximal effects on drug metabolism are observed. As drug metabolism increases, the amount of drug available to enter the brain decreases.

The inhibition of drug metabolism often occurs much more rapidly than the stimulation, usually as soon as a sufficient concentration of the metabolic inhibitor is achieved, which results in increased plasma and brain concentrations of the drug. The renal (kidney) excretion of drugs that are weak acids or weak bases can be influenced by drugs that alter urinary pH to change the reabsorption of the drug from urine into the kidney. The active secretion of the drug into the urine can also be affected. Both processes can ultimately affect the plasma and subsequent brain concentrations of the drug. Pharmacodynamic mechanisms can either enhance or reduce the response of a given drug. For example, if two drugs are agonists for the same receptor site—DIAZEPAM (Valium) and CHLORDIAZEPOXIDE (Librium) for BENZODIAZEPINE receptor-binding sites—then an additive biological response is likely to occur unless a maximum response is already present. If, however, an AGONIST competes with an ANTAGONIST for the same binding site (e.g., see MORPHINE and NALOXONE in OPIOIDS, discussed below), then a decreased biological response is likely.

Enhanced or diminished biological responses can be observed even if the drugs do not interact with the same receptor-binding sites. In this case, the net effect is the sum of the pharmacological properties of the drugs. For example, if two drugs share a similar biological response (e.g., central nervous system depression) even though they produce their effects at different sites, then the concur-

rent ingestion of both drugs can result in an enhanced depression of the central nervous system (see the ALCOHOL [ethanol] and Valium discussion below). Finally, the concurrent ingestion of two or more drugs, each with toxic effects on the same organ system, can increase the chance for extensive organ damage.

DEPRESSANTS

Alcohol (Ethanol) and Valium. Reactions that are additive (combined) or synergistic (cooperative effects greater than the sum of the independent effects of the drugs taken alone) are common side effects that result from the consumption of two or more drugs with similar pharmacological properties. For example, although alcohol (ethanol) is considered by many to be a stimulant drug because, typically, it releases an individual's latent behavioral inhibitions (i.e., it produces disinhibition), alcohol actually produces a powerful depression of the central nervous system similar to that seen with general anesthetics. The subsequent impairment of muscular coordination and judgment associated with alcohol intoxication can be enhanced by the concurrent administration of other central nervous system depressants. Often, Valium or Librium (benzodiazepines that are considered relatively safe drugs) may be purposely ingested along with ethanol in an attempt to "feel drunk" faster or more easily. Since ethanol actually increases the absorption of benzodiazepines, and also enhances the depression of the central nervous system, the potential toxic side effects of the two drugs are augmented. Ethanol is often a common contributor to benzodiazepine-induced coma as well as to benzodiazepine-related deaths, demonstrating that interactions of these drugs with alcohol can be especially serious. Furthermore, the combination of alcohol with the SEDATIVE-HYPNOTIC BARBITURATES (e.g., pentobarbital, secobarbital) can also produce a severe depression of the central nervous system, with decreased respiration. The intentional ingestion of ethanol and secobarbital (or Valium) is a relatively common means of SUICIDE.

Alcohol (Ethanol) and Opioids. Alcohol can also enhance the respiratory depression, sedation, and hypotensive effects of MORPHINE and related opioid drugs. Therefore, the concurrent ingestion of the legal and socially acceptable drug ethanol with other sedatives, hypnotics, anticonvulsants, ANTIDEPRESSANTS, antianxiety drugs, or with an ANALGESIC agent (such as morphine) can result in serious and potentially fatal drug interactions through a potentiation of the depressant effects of these drugs on the central nervous system. Since the 1960s, a significant number of musicians, actors, and other high-profile personalities have either accidentally or intentionally overdosed from a combination of alcohol and other central nervous system depressants. A few notable examples include actress Marilyn Monroe, musicians Jimi Hendrix, Janis Joplin, Jim Morrison, Keith Moon, and John Bonham.

STIMULANTS

Stimulants and Toxic Effects. Synergistic toxic effects are also often obscured with other classes of drugs. For example, the concurrent ingestion of central nervous system stimulants (e.g., AMPHETAMINE, COCAINE, CAFFEINE) can also produce additive side effects, especially with respect to toxic reactions involving the heart and cardiovascular system. These toxic reactions are often manifested as an irregular heartbeat, stroke, heart attack, or even death. Drugs with apparently different mechanisms of action can result in dangerous and unexpected synergistic side effects with fatal consequences. For example, some amphetamine and cocaine users often attempt to self-medicate their feelings of "overamp," or the excessive STIMULANT high resulting from prolonged central nervous system stimulation, through the concurrent administration of central nervous system depressants such as alcohol, barbiturates, or heroin (i.e., a "speedball"). The rationale behind this potentially dangerous practice is that a few beers, a Quaalude, or perhaps a shot of heroin will help the individual "mellow out" for a while before inducing a stimulant high again. High doses of cocaine or amphetamine can, however, result in respiratory depression from actions on the medullary respiratory center. Therefore, the concurrent ingestion of a central nervous system stimulant (e.g., cocaine) with a depressant (e.g., heroin) can result in increased toxicity or death from the enhanced respiratory depression produced by the combination of the two drugs. The most well-known casualty from this type of pharmacological practice was comedian John Belushi.

CLINICAL USES

The principles of drug interactions can be used clinically for the treatment of acute INTOXICATION and for WITHDRAWAL—by transforming, reducing, or blocking the pharmacological properties and/or the toxic effects of drugs used and abused for nonmedical purposes. Although these interactions often involve a competition with the abused drug for similar central nervous system RECEPTOR sites, other mechanisms are also clinically relevant.

Disulfiram and Alcohol (Ethanol). One such nonreceptor-mediated interaction involves DISULFIRAM (Antabuse) and ethanol (alcohol). Since an ethanol-receptor site has not yet been conclusively identified, specific receptor agonists and antagonists are not yet available for the treatment of ethanol intoxication, withdrawal, and abstinence (as they are for opioids). Disulfiram is sometimes used in the treatment of chronic ALCOHOLISM, although the drug does not cure alcoholism; rather, it interacts with ethanol in such a way that it helps to strengthen an individual's desire to stop drinking. Although disulfiram by itself is relatively nontoxic, it significantly alters the intermediate metabolism of ethanol, resulting in a five- to tenfold increase in plasma acetaldehyde concentrations. This acetaldehyde syndrome results in vasodilatation (dilation of blood vessels), headache, difficulty breathing, nausea, vomiting, sweating, faintness, weakness, and vertigo. All of these reactions are obviously unpleasant, especially at the same time, thus well worth avoiding. The acetaldehyde syndrome therefore helps to persuade alcoholics to remain abstinent, since they realize that they cannot drink ethanol for at least three or four days after taking disulfiram. The consumption of even small or moderate amounts of ethanol following disulfiram pretreatment can result in extremely unpleasant drug interactions through the acetaldehyde syndrome.

Opioids. Drug interactions involving opioids (morphine-like drugs) and opioid receptors are classic examples of how knowledge of the molecular mechanisms of the actions of a class of drugs can assist in the treatment of acute intoxication, withdrawal, and/or abstinence. Naloxone, the opioid-receptor antagonist, can be used as a diagnostic aid in emergency rooms. In the case of a comatose patient with unknown medical history, the intravenous administration of naloxone can provide information on whether or not the coma is the result of an opioid overdose. The antagonist competes with the agonist (usually heroin or morphine) for the opioid-receptor sites, displacing the agonist from the binding sites to reverse the symptoms of an overdose effectively and rapidly. Continued naloxone therapy and supportive treatment are often still necessary.

If, however, naloxone is administered to an individual dependent on opioids but not in a coma, a severe withdrawal syndrome develops within a few minutes and peaks after about thirty minutes. Depending on the individual, such precipitated withdrawal can be more severe than that following the abrupt withdrawal of the opioid-receptor agonist (e.g., heroin). In the former instance, the binding of the agonist to opioid receptors is suddenly inhibited by the presence of the antagonist (e.g., naloxone); even relatively large doses of the agonist (e.g., heroin) cannot effectively overcome the binding of the antagonist. Quite the contrary, respiratory depression can develop if higher doses of the agonist are administered. Therefore, opioid-receptor antagonists are not recommended for the pharmacological treatment of opioid withdrawal. Rather, longer acting, less potent, opioid receptor-agonists, such as METHADONE, are more commonly prescribed.

Methadone. The symptoms associated with methadone withdrawal are milder, although more protracted, than those observed with morphine or heroin. Therefore, methadone therapy can be gradually discontinued in some heroin-dependent people. If the patient refuses to withdraw from methadone, the person can be maintained on methadone relatively indefinitely. TOLERANCE develops to some of the pharmacological effects of methadone, including any reinforcing or rewarding effects (e.g., the euphoria or "high"). Therefore, the patient cannot attain the same magnitude of euphoria with continued methadone therapy, although the symptoms associated with opioid withdrawal will be prevented or attenuated. Cross-tolerance also develops to other opioid drugs, so the patient will not feel the same high if heroin is again used on the street.

This type of maintenance program makes those who are heroin dependent more likely to accept other psychiatric or rehabilitative therapy. It also reduces the possibility that methadone patients will continue to seek heroin or morphine outside the clinic. In this way, the principles of drug interac-

tions involving opioid receptors in the central nervous system have helped to stabilize TREATMENT strategies for opioid withdrawal and abstinence.

(SEE ALSO: *Accidents and Injuries; Complications; Neurological; Drug Abuse Warning Network*)

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DRUG INTERACTIONS AND ALCOHOL

The term *alcohol-drug interaction* refers to the possibility that alcohol may alter the intensity of the pharmacological effect of a drug, so that the overall actions of the combination of alcohol plus drug are additive, potentiated, or antagonistic. Such interactions can be divided into two broad categories—PHARMACOKINETIC and PHARMACODYNAMIC. Pharmacokinetics are concerned with the extent and rate of absorption of the drugs, their distribution within the body, binding to tissues, biotransformation (metabolism), and excretion. Pharmacokinetic interactions refer to the ability of alcohol to alter the plasma and tissue concentration of the drug and/or the drug metabolites, such that the effective concentration of the drug at its target site of action is significantly decreased or increased. Pharmacodynamics are concerned with the biochemical and physiological effects of drugs and their mechanisms of action. Pharmacodynamic interactions refer to the combined actions of alcohol and the drug at the target site of action, for example, binding to enzyme, receptor, carrier, or macromolecules. Pharmacodynamic interactions may occur with or without a pharmacokinetic component. For many drugs acting on the central nervous system that exhibit cross-tolerance (a similar tolerance level) with alcohol, pharmacodynamic interactions with alcohol are especially important.

Most drugs are metabolized in the liver by an enzyme system usually designated as the cytochrome P450 mixed-function oxidase system, and the liver is the principal site of many alcohol-drug pharmacokinetic interactions. Two major factors—blood flow to the liver and the activity of drug-metabolizing enzymes—strongly influence the overall metabolism of drugs. Biotransformation of drugs that are actively metabolized by liver enzymes mainly depends on the rate of delivery of the drug to the liver. These may be flow-limited drugs, where the liver can transform as much drug as it receives, or capacity-limited drugs, which have a low liver-extraction ratio—their clearance (removal from the blood) primarily depends on the rate of their metabolism by the liver.

There are a number of factors other than the drugs themselves that influence the speed and intensity of alcohol/drug interactions in the human body. These include the patient's sex, weight, age, and race; the presence or absence of food in the stomach; and history of alcohol intake. For example, the levels of alcohol dehydrogenase (ADH), a stomach enzyme that oxidizes alcohol to acetaldehyde, are lower in women than in men; lower in Asians than in Western Caucasians; and lower in alcoholics than in nonalcoholics. Elderly persons are at greater risk of alcohol/drug interactions than younger adults, because they usually take more prescription medications, are more likely to have a serious illness, and show age-related changes in the absorption and clearance of certain medications.

With regard to stomach contents, food generally slows the rate of alcohol absorption. Consequently, medications that increase the rate of gastric emptying, such as erythromycin (Eryc, Ilotycin) or cisapride (Propulsid), enhance the rate of alcohol metabolism.

ALCOHOL-DRUG INTERACTIONS

Alcohol-drug interactions are complex. The consequences of using alcohol and drugs together vary with the dosage of the drug; the amount of alcohol consumed; the mode of administering the drug (oral, intravenous, intramuscular, etc.); and the nature of the drug (anticonvulsant, vasodilator, analgesic, etc.). The alcohol may alter the effects of the drug; the drug may change the effects of alcohol; or both may occur.

Alcohol-drug interactions are most important with drugs that have a steep DOSE-RESPONSE CURVE and a small therapeutic ratio—so that small quantitative changes at the target site of action lead to significant changes in drug action. In alcoholics, changes in susceptibility to drugs are due to changes in their rates of metabolism (pharmacokinetics) and the adaptive and synergistic effects on their organs, such as the central nervous system (pharmacodynamics). The clinical interactions of alcohol and drugs often appear paradoxical: Sensitivity to many drugs, especially sedatives and tranquilizers, is strikingly increased when alcohol is present at the same time; however, alcoholics, when abstinent, are tolerant to many drugs. These acute and chronic actions of alcohol have been attributed, respectively, to additive and adaptive responses in the central nervous system (pharmacodynamic interactions).

It is now recognized that alcohol can also interact with the cytochrome P450 drug-metabolizing system, binding to P450, being oxidized to acetaldehyde by P450, increasing the content of P450, and inducing (causing an increase in the activity of) a unique isozyme of P450. Inhibition of drug oxidation when alcohol is present at the active site of P450 is due to displacement of the drug by alcohol and competition for metabolism; this increases the half-life and circulating concentration of drugs. Induction of P450 by chronic-alcohol treatment can result in the increased metabolism of drugs, as long as alcohol is not present to compete for oxidation. These pharmacokinetic interactions may contribute to either increased sensitivity or the tolerance observed with alcohol-drug interactions.

Alcohol can affect drug pharmacokinetics by altering drug absorption from the alimentary tract. For example, diazepam (Valium) absorption is enhanced by the effects of alcohol on gastric emptying. Alcohol placed in the stomach at concentrations of 1 percent to 10 percent increases the absorption of pentobarbital, PHENOBARBITAL, and theophylline, whereas drugs such as DISULFIRAM and CAFFEINE decrease alcohol absorption by decreasing gastric emptying. Cimetidine (Tagamet)—a drug used to treat stomach ulcers—increases blood alcohol concentrations by inhibiting ADH in the stomach and first-pass metabolism of alcohol. Binding of a drug to plasma proteins will change the effective therapeutic level of the drug, because when the drug is linked to the proteins, it is not available to act on the tissue. Alcohol itself and alcohol-induced liver disease cause a decreased synthesis and release of such plasma proteins as albumin. The resulting hypoproteinemia can result in decreased plasma-protein binding of such drugs as quinidine (Quinidex), dapsone (DDS), triamterene (Dyrenium), and fluorescein (Fluorescite). Alcohol may also directly displace drugs from plasma proteins.

The effects of alcohol on blood flow in the liver are controversial, although most recent reports suggest an increase; this could be significant with respect to metabolism of flow-limited drugs. At higher concentrations, alcohol can act as an organic solvent and "fluidize" cellular membranes, which may increase the uptake or diffusion of drugs into the cell.

METABOLISM

Many alcohol-drug interactions occur at the level of actual metabolism. Ethanol (ethyl alcohol—common in wines and liquors) will compete with such other alcohols as METHANOL (methyl alcohol—called wood alcohol) or ethylene glycol (antifreeze), for oxidation via alcohol dehydrogenase. In fact, treatment against poisoning by methanol or ethylene glycol involves the administration of ethanol—as the competitive inhibitor—or the addition of inhibitors of alcohol dehydrogenase such as pyrazole or 4-methylpyrazole.

As discussed above, the presence of alcohol will inhibit the oxidation of drugs by cytochrome P450.

Alcohol has been shown to inhibit oxidation of such representative drugs as aniline, pentobarbital (Nembutal), benzphetamine (Didrex), benzpyrene, aminopyrine, ethylmorphine, METHADONE, meprobamate (Equanil, Miltown), phenytoin (Dilantin), propranolol (Inderal), caffeine, tolbutamide (Orinase), warfarin (Coumadin), phenothiazine, BENZODIAZEPINE, CHLORDIAZEPOXIDE, amitriptyline (Elavil), chlormethiazole, chlorpromazine (Thorazine), isoniazid (INH), imipramine (Tofranil), dextropropoxyphene, triazolam (Halcion), industrial solvents, and acetaminophen (Tylenol). As this partial list indicates, oxidation of many classes of drugs can be inhibited in the presence of alcohol; these include HYPNOTICS, OPIOIDS, psychotropic drugs, anticonvulsants, vasodilators, antidiabetics, anticoagulants, ANALGESICS, and antibacterials. Chronic consumption of alcohol induces the P450 drug-metabolizing system, which could increase oxidation of drugs in sober or abstinent alcoholics. Among the drugs that may be more rapidly metabolized in abstinent alcoholics are ethoxycoumarin, ethylmorphine, aminopyrine, antipyrine, pentobarbital, meprobamate, methadone, theophylline (Bronkodyl, Theo-Dur), tolbutamide, propranolol, rifamycin, warfarin, acetaminophen, phenytoin, deoxycline, and ethanol itself. An important consequence of this ability of chronic ethanol intake to increase drug-clearance rates is that the effective therapeutic level of a drug will be different in an abstaining alcoholic than it is in a nondrinker. This metabolic drug tolerance can persist for several days to weeks after alcohol WITHDRAWAL.

PHARMACODYNAMIC IMPLICATIONS

These alcohol-drug pharmacokinetic interactions can have major pharmacodynamic implications. Some examples include the following: The concurrent administration of alcohol plus amitriptyline (Elavil) to healthy volunteers resulted in an increase in the plasma-free concentration of amitriptyline, since the alcohol inhibited drug clearance. Other pharmacodynamic interactions between alcohol and amitriptyline include decreased driving skills (and other psychomotor skills), greater than additive loss of righting reflex, unexpected blackouts, and even death. Laisi et al. (1979) showed that plasma levels of the tranquilizer DIAZEPAM (Valium—an antianxiety drug) were increased in the presence of beer and wine, so the combination of alcohol plus diazepam produced impaired tracking skills, increased nystagmus (nodding off), and impaired oculomotor (eye) coordination, as compared to diazepam alone. Therapeutic doses of the tranquilizers diazepam or chlordiazepoxide (Librium) plus alcohol have been consistently shown to produce impairment of many mental and psychomotor skills; EEG (electroencephalogram) abnormalities could still be detected sixteen hours after administration of fluorazepam in the presence of alcohol to volunteers. Alcohol also decreases the rates of elimination of several benzodiazepines in humans. Phenothiazines and alcohol compete for metabolism by P450, resulting in the decreased clearance of chlorpromazine (Thorazine), for example, and enhanced sedative effects, impaired coordination, and a severe potentially fatal respiratory depression. Alcohol inhibits the metabolism of BARBITURATES, prolonging the time and increasing the concentration of these drugs in the bloodstream, so that central nervous system interactions are intensified. In humans, alcohol doubles the half-life of pentobarbital; this is associated with a 10 to 50 percent lower concentration of barbiturate sufficient to cause death by respiratory depression, as compared to the lethal dose in the absence of alcohol. Striking pharmacokinetic and pharmacodynamic interactions occur between alcohol and the hypnotic drug CHLORAL HYDRATE—the so-called Mickey Finn or knockout drops. Alcohol inhibition of MORPHINE metabolism increases morphine accumulation, potentiates central nervous system actions, and increases the probability of death.

OTHER CONSEQUENCES

Pharmacokinetic interactions between alcohol and drugs also have important toxicological and carcinogenic consequences. The metabolism of certain drugs produces reactive metabolites; these are much more toxic than the parent compound. The induction of P450, especially the P4502E1 isozyme by alcohol, results in the increased activation of drugs and SOLVENTS to toxic reactive intermediates—such as carbon tetrachloride, acetaminophen, benzene, halothane, enflurane, COCAINE, and isoniazid. In a similar manner, procarcinogens—such as aflatoxins, nitrosamines, and aniline dyes—are activated to carcinogenic metabolites af-

ter alcohol induction of P4502E1. Since P4202E1 is localized largely in the perivenous zone of the liver cell, the increased activation of these toxins (and alcohol itself) after induction by alcohol may explain the preferential perivenous toxicity of several hepatotoxins, carcinogens, and alcohol itself.

(SEE ALSO: *Complications; Drug Interaction and the Brain; Drug Metabolism; Psychomotor Effects of Alcohol and Drugs*)

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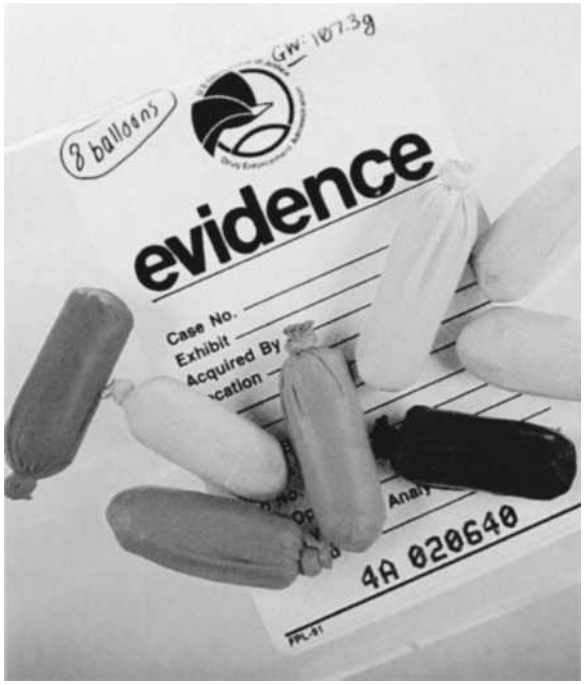
DRUG INTERDICTION The interdiction of illicit drugs into the United States is the effort to seize them, together with the transport and/or persons that carry them on their way from the producing country to the importing country; many of the SEIZURES occur just as the drugs are brought across the border. The principal drugs subject to U.S. interdiction are COCAINE and MARIJUANA, both of which are imported primarily from Latin America. The United States, uniquely among modern nations, has made interdiction a significant part of its effort to control the supply of drugs, at least for cocaine and marijuana, since about 1975. In addition to other federal agencies, it has involved the military in this effort. Though interdictors have seized large quantities of drugs, there are still numerous questions about the effectiveness of the program as a method of reducing the use of drugs, particularly cocaine.

GOALS

Interdiction has two general goals. The primary one is to reduce the consumption of specific drugs within the nation by making it more expensive and risky for smugglers to conduct their business. Drug seizures raise costs by increasing the amount that has to be shipped in order to ensure that a given quantity will reach the market. Additionally, an effective interdiction program will, among other things, raise the probability that a courier is arrested, thereby increasing the price smugglers have to pay to those who undertake the task. These higher fees raise smugglers' costs of doing business and thus the price they must charge their customers, the importers. Finally, the increased costs lead to a higher retail price and serve to lower consumption of the drug.

At one time it was thought that interdiction could impose a physical limit on the quantity of drugs available in this country. With a fixed supply available in the producing nations, each kilogram seized on its way to the United States would be one less kilogram available for consumption here. However, this has not proven to be the case. It is now generally accepted that production is expandable and that increased seizures can be compensated for with increases in production, although farmers may have to receive higher prices to provide greater production.

A second, more modest, general goal is to increase the difficulty of smuggling itself and to provide suitable punishment. Smugglers, or at least the



In one method of smuggling heroin, couriers swallow heroin-filled latex balloons before boarding commercial airlines. (Drug Enforcement Administration)

principals in smuggling organizations, are among the most highly rewarded participants in the drug trades. There is support for programs that conspicuously make their lives less easy and that subject them to the risks of punishment.

Three illegal drugs have traditionally dominated imports: cocaine, HEROIN, and marijuana. Heroin is subject to only modest interdiction efforts because it is usually smuggled in conventional commercial cargo, or it is carried on (or within) the person of the smugglers who travel by commercial traffic; seizures are made only in the course of routine inspection of cargo and traffic. It is estimated that ten tons of heroin are smuggled into the United States each year, and seizures of more than ten kilograms are rare. The Drug Enforcement Administration (DEA) reported that a total of 667 kilograms of heroin had been seized in 1999, about the same amount seized in 1990. Cocaine and marijuana have been the primary targets of interdiction, although an effective program of interdiction against Colombian maritime smuggling has led to a sharp rise in the share of the U.S. marijuana market served by domestic producers.

TECHNIQUES

The techniques of interdiction inevitably mirror those of smugglers. Drugs enter the United States by air, land, and sea, by private vessel and commercial carrier. Interdiction must, if it is to have any substantial effect on the drug trade, act against all the modes of smuggling; otherwise smugglers will rely on the mode that is not subject to interdiction.

Interdiction has three separate elements: monitoring, detection and sorting, and pursuit and apprehension. For example, U.S. COAST GUARD ships supported by an extensive radar system patrol the Caribbean, which constitutes the major thoroughfare for smuggling from Latin America. The Coast Guard patrol vessels attempt to see, either directly or through radar, all ships moving along certain routes. This constitutes the monitoring activity. The interdictors must then sort, from all that traffic, the relatively small number that are carrying illegal drugs. Finally, they must pursue the smugglers that have been detected, arrest the personnel, and seize the drugs and the ship itself. The interdiction system is as weak as its weakest component; for example, a system that has good pursuit capacities but is unable to sort smugglers from innocents effectively will waste much of that pursuit capacity in chasing nonsmugglers. Similarly, good detection will lead to few captures without effective monitoring capabilities.

The Coast Guard and Customs Service share primary responsibility for marine and air interdiction. The Coast Guard patrols more distant routes, with Customs having a greater role in the U.S. coastal zone. Both agencies also conduct interdiction against private planes, with Customs having primary responsibility over the Mexican land border, a major trafficking area. The DEA expanded its air surveillance, having a fleet of ninety-five aircraft in 2000. The Border Patrol, a unit of the Immigration and Naturalization Service, has primary responsibility for the interdiction of drugs carried in cars or on persons crossing the land border. In the late 1990s, new technology, such as x-ray machines that examine commercial vehicles, was installed at border stations in the Southwest.

Both Customs and the Border Patrol make many seizures and arrests in the course of routine inspection. For example, Customs may find a shipment of cocaine concealed inside a cargo container being unloaded in the Miami port; the Border Patrol, in the course of pursing illegal immigrants, might find a "mule" (a person) carrying a backpack full of cocaine or heroin. Drugs are shipped in an amazing array of forms; for example, suspended in frozen fruit pulp being imported from Ecuador or in hollowed lumber from Brazil.

MILITARY INVOLVEMENT

For a variety of reasons, there was pressure throughout the 1980s to increase the extent of military involvement in drug interdiction. The drug problem was viewed as a national crisis with an important international element. The military was seen as having unique capabilities, both of equipment and of training, to protect the borders.

The military had been ambivalent about entering the drug interdiction arena to any significant degree, seeing it as potentially corrupting and an inappropriate diversion from its primary mission. With the collapse of its principal strategic enemy, the Soviet Union, the U.S. military has become more willing to play a major interdiction role. This has been reflected in large increases to the military budgets to handle these new responsibilities. Current law prohibits arrests by military personnel. Accordingly, military participation has been confined to detection and monitoring rather than pursuit and apprehension.

The U.S. Navy provides a number of ships for interdiction patrols in both the Caribbean and the Pacific, combining training with a useful mission. The military runs the integrated radar and communication system that links the Customs Service, the Coast Guard, the Border Patrol, and other agencies. There have been no reports of significant problems of corruption associated with the military role in drug interdiction, but relations between the military and the civilian law-enforcement agencies with primary jurisdiction have sometimes been strained, the result largely of differences in organizational cultures.

With the proliferation of U.S. government units involved in interdiction, the need arose for coordinated command. The government has two Joint Inter-Agency Task Forces, one based in Key West, Florida, the other in Alameda, California. These task forces coordinate transit zone activities, including the U.S.-Mexico land border and air and maritime traffic along the borders and sea coasts. The U.S. Interdiction Coordinator is the Commandant of the U.S. Coast Guard.

EVALUATION TECHNIQUES

Evaluation of the effectiveness of interdiction has been a vexed issue ever since the activity became prominent in the late 1970s. Very large quantities of drugs, particularly of cocaine, have been seized, but the size if such seizures has been cited both as evidence of success and of failure. It has been questioned whether more cocaine is being seized because interdictors are getting better at their job or because more cocaine is being shipped.

At a minimum, it would be desirable to express seizures as a fraction of total shipments (consumption plus seizures), but, unfortunately, estimates of consumption lack any systematic basis. Even expressed as a fraction of shipments, seizures are clearly an inadequate measure of the effectiveness of interdiction, since the program imposes two other costs on smugglers—namely, seizure of assets (e.g., boats, planes, real estate, and financial holdings) and the arrest and imprisonment of smuggling agents (e.g., crew members on ships, pilots, and couriers for financial transactions).

Reuter et al. (1988) suggest that the most appropriate measure is a price increase in the smuggling sector of drug distribution. Effective interdiction should raise smugglers' costs; the increase will be reflected in the difference between the price at which smugglers purchase drugs in the producer country (export price) and that at which they sell it in the importing country (import price). However, the process cannot serve as an operational criterion for any individual component of interdiction, since prices are set in a national market serviced by all modes and routes of smuggling. Anderberg (1992) concluded that the available data supported only inappropriate and/or inadequate measures of effectiveness, while any more cogent measure requires data that are not available and are not likely to be readily obtained.

One negative consequence of interdiction identified by Reuter et al. (1988) has received little attention. By seizing drugs on their way from the source country, interdiction may actually increase export

demand for those drugs. As noted earlier, more stringent interdiction has two effects; it raises prices and thus reduces final demand in the United States, but it also increases the amount that must be shipped to meet a given consumption (because of a higher replacement rate). It appears that, based on reasonable assumptions about the cost structure of the cocaine trade, the second effect has proven greater than the first.

THE EFFECTIVENESS OF INTERDICTION

Interdiction clearly has had some important consequences for the drug trade in the United States. In contrast to the 1970s, little marijuana is now imported from Colombia, though that nation remains a low-cost producer. Successful interdiction, particularly against marine traffic from Colombia, has imposed such high costs on Columbian imports that now both Mexican and U.S. producers have come to dominate the U.S. market. Interdiction against Mexican-produced drugs is more difficult and thus the import price of Mexican marijuana is less than that of Colombian.

For cocaine there is much less evidence of success, though interdictors have certainly forced changes in modes of smuggling. In the early 1980s much of the cocaine was brought up by private plane directly from Colombia, but now most of it enters either by transshipment through MEXICO or by commercial cargo. However, though interdictors now seize a large share of all shipments, they have not managed to prevent a massive decline in the landed price of the drug. Street prices of cocaine and heroin have dropped dramatically since the early 1980s.

The reasons for this limited success are not hard to find. Smugglers defray the risks of getting caught by carrying across very large quantities, so that the risks per unit smuggled are low. A pilot who charges 250,000 dollars for the risks (imprisonment, suffering injury or death in the course of landing) involved in bringing across a shipment of 250 kilograms is asking for only one dollar per gram, less than 1 percent of the retail price. Even if interdictors make smuggling much more risky, so that the pilot doubles the demand to 500,000 dollars, the higher fee still adds only another 1 percent to the retail price.

Moreover, it is difficult to make smuggling very risky when the nation is determined also to maintain the free flow of commerce and traffic. Hundreds of millions of people enter the country each year; cargo imports also amount to hundreds of millions of tons. Only a few hundred tons of cocaine need to be concealed in that mountain of goods and only a few thousand of those who enter need be in the smuggling business to ensure an adequate and modestly priced supply of cocaine.

Interdiction has accounted for a significant portion of federal government expenditures on drug control. By the end of the 1990s, many critics of the interdiction effort argued that these resources should be put into drug treatment programs and other programs that could reduce the demand for illegal drugs. Nevertheless, the U.S. government has remained committed to interdiction operations.

(SEE ALSO: *Border Management; Dogs in Drug Detection; Foreign Policy and Drugs; International Drug Supply Systems; Operation Intercept; U.S. Government: The Organization of U.S. Drug Policy*)

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DRUG LAWS: FINANCIAL ANALYSIS IN ENFORCEMENT The application of financial investigative techniques to sophisticated forms of CRIME began decades ago in campaigns to bring underworld bosses to justice. They were charged

not with the underlying offenses of bootlegging or extortion, but for reaping financial windfalls from activities that either were not federal offenses at the time or that prosecutors just could not prove. Beginning with the federal tax case against Al Capone in 1931, Treasury investigators had to find ways around both the lack of federal laws proscribing racketeering activity and the difficulties in catching underworld bosses for their offenses. The approach was creative but simple: Internal Revenue agents gathered evidence to prove that the racketeers spent more income than they reported on their tax returns. The differential between what was reported and what the government alleged they earned would establish that their target received substantial amounts of unreported income. In an underworld without pay stubs and annual wage statements, how did the government know what the racketeers earned? To tax investigators, it was simple: Show how much the person spent—or at least the portion of income spent that could be substantiated.

As Prohibition gave way to different forms of industrial racketeering, syndicated gambling, and drug trafficking, federal agents grew more frustrated over their poor showing against criminals who were developing increased sophistication. Investigators turned more and more to financial analysis as an alternative. They reasoned that what worked against Al Capone and his cohorts would probably work against other high-profile racketeers too insulated by their underlings to be implicated in syndicate transactions.

Proving that individuals—whether they were Mafia bosses or Colombian drug importers—received more income than they could substantiate was easier said than done. Typically, there were no records that acted as a smoking gun by pointing directly to one large unreported sum of yearly income. Rather, evidence of unreported income was gathered from a variety of sources and was traced to documented purchases that left a paper trail of deposit slips, bank statements, advices, credit card receipts, and mortgages. As investigators soon came to find out, moreover, financial analyses frequently turned up large amounts of money in the possession of people who recently had approved plans for a lucrative drug deal or some other illegal transaction.

For investigators struggling to tie drug traffickers to crimes they only planned, finding the proceeds of those transactions was welcome evidence. For one thing, it could tie their target to the drug or other transactions that other evidence showed they had planned or approved. Drug traffickers and other racketeers who never touch drugs do touch, or otherwise control, the money that was exchanged for the drugs. Hundreds of criminals have been sent to prison on the basis of financial analyses tying large sums in question to the defendants and alleged criminal transactions.

As organized crime began to wane in national prominence in the 1970s, its place was quickly taken by an amalgam of homegrown and foreign-based drug traffickers. Often just as smart and insulated as Mafia bosses, drug traffickers were surprised to find themselves equally vulnerable to cases built on financial evidence. Passage of a number of federal drug reform laws (in 1970, 1978, 1984, 1986, and 1988) added the remedy of asset forfeiture to the government's arsenal of weapons. In order to show that their targets acquired assets with tainted funds that rendered them forfeitable, investigators resorted to the same financial investigative techniques that had helped build criminal tax cases against the same kinds of underworld leaders.

In the mid-1990s, virtually all federal enforcement agencies provide some type of basic training in financial investigation, and several—such as the Drug Enforcement Administration, Federal Bureau of Investigation, and Internal Revenue Service—have highly specialized programs at their academies. DEA and FBI, expert investigators and financial analysts support major drug-trafficking cases by providing evidence of unexplained income to prove the drug charges, and to tie the money to drug activity for the purpose of forfeiture.

(SEE ALSO: *International Drug Supply Systems; Money Laundering*)

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DRUG LAWS, PROSECUTION OF Drug arrests in the United States involve a wide variety of controlled substances, including MARIJUANA, COCAINE, HEROIN, PHENCYCLIDINE (PCP), and others, and a number of different charges, including possession, dealing (selling), and conspiracy to sell.

After arrest, the prosecutor, or district attorney, exercises the discretion to choose among this broad range of legal options in deciding whether to bring a charge and for what activity.

Drug offenses can violate either federal or state laws. Since the majority of arrests are made by local law-enforcement officials, most defendants are charged in state courts. The cases received by federal prosecutors, called U.S. attorneys, from such federal enforcement agencies as the Federal Bureau of Investigation (FBI) or the DRUG ENFORCEMENT ADMINISTRATION (DEA), frequently involve more complex matters. However, the volume of federal drug prosecutions rose in the 1990s, as tougher federal drug laws and sentencing provisions led state prosecutors to refer these cases to federal jurisdiction. In addition, federal prosecutors have used the Racketeer Influenced and Corrupt Organizations Act (RICO) to prosecute drug traffickers and to confiscate property used in drug enterprises. The federalization of drug crimes has had a profound impact on the work of the federal courts and the budget of the federal prison system.

In determining what charges should be filed against the offender, the prosecutor looks to many factors: the criminal history of the defendant, the seriousness of the drug involved, and the quality of the evidence. Most states give the district attorney the discretion to charge an enhanced-penalty crime for a repeat offender.

The vast majority of the cases lead to guilty pleas, through some form of plea bargaining between the prosecutor and the defense attorney. In these agreements, which must be approved by the court, the defendant pleads guilty, often in return for a fine, court-ordered counseling, or a lessened prison term. Repeat offenders face tougher agreements.

In deciding what plea to accept, prosecutors consider many of the same factors they did when they brought the original charges. A critical factor is the quality of the evidence. Many drug cases are very easy to prove, because the defendant purchased or sold the drugs directly to a police officer or because a search warrant leads to the discovery of drugs in an area controlled by the defendant. District attorneys face much more difficult challenges in convicting suspects involved in complicated conspiracy charges such as those associated with the shipment or distribution of drugs. In many drug prosecutions, motions to suppress evidence are filed by defense attorneys to determine whether the search that turned up the drugs was conducted in a legal manner. Rulings by the U.S. Supreme Court provide wider latitude to officers who have secured a search warrant.

Another important factor involves the level of cooperation provided by the defendant. The district attorney often accepts a more lenient agreement for defendants who assist law-enforcement officers and/or testify in court concerning who sold them the drugs they possessed or resold. These plea agreements allow the police to target other offenders and also relieve the pressure on the courts. Plea bargaining does, however, raise serious questions in the public's mind about the dangers of leniency; it raises other questions, among defendants and their attorneys, about equity and fairness. Additionally, narcotic officers and prosecutors often disagree about the outcome or the handling of a case. These differences are often mediated by task forces in which prosecutors with specialized drug experience are assigned to work with a select group of narcotics officers.

Generally less than 10 percent of drug cases go to trial. In a trial the police officer is a witness in the case brought by the prosecutor. By questioning the officer, the prosecutor, as lawyer for the state, elicits evidence designed to show that the defendant possessed or sold drugs.

Ultimately the judge determines the actual sentence. However, federal and state sentencing guidelines limit the judge's discretion. Many drug crimes carry with them mandatory minimum sentences. But commonly in a plea agreement or after trial, the prosecutor can modify the severity of the sentence by reducing the charge or by recommending that the court reduce the sentence. Across the country, and even within large counties, great differences occur in sentencing and in sanction recommendations.

Participants in the criminal justice system recognized that drug-related crimes should be addressed in different ways. The emergence of drug courts in the 1990s signaled a new way of prosecuting drug offenders. Drug courts seek to reduce drug use and associated criminal behavior by retaining drug-involved offenders in treatment. Drug courts divert drug offenders from jail or prison and refer them to community treatment. Defendants who complete the program either have their charges dismissed or probation sentences reduced. A 1994 federal law

authorizes the U.S. Attorney General to make grants to state and local governments to establish drug courts. By 1999, 416 drug courts were operating in the United States, with over 270 more in the planning stages. These courts shift discretion from the prosecutor and place it with the judge, who has broad discretion in a drug court.

Federal and state prosecutors have also used asset forfeiture laws to attack drug traffickers. Forfeiture laws authorize prosecutors to file civil lawsuits asking a court for permission to take property from a criminal defendant that was either used in the crime or was the fruit of a criminal act. According to Department of Justice statistics, over 28,000 properties were seized in 1996, with a combined value of $1.264 billion.

Aside from civil forfeiture, prosecutors have also used non-criminal statutes and ordinances to attack drug crimes. For example, prosecutors may use public nuisance laws, zoning laws and public health laws to remove drug offenders from property where drugs are being used and sold. Though the legal action is addressed at the landlord or property owner, it has the effect of removing drug users and traffickers from apartments and houses. Increasingly, cities are condemning and destroying buildings that have been used as crack houses and such.

(SEE ALSO: *Exclusionary Rule; Mandatory Sentencing; Rockefeller Drug Law*)

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STEPHEN GOLDSMITH

DRUG METABOLISM Most drugs are taken by mouth and, in order to be absorbed through the stomach and intestine, they need to be lipid-soluble. This solubility permits them to easily cross the membrane barrier. After absorption, organs with plentiful blood-flow such as the brain, liver, lungs, and kidneys are first exposed to the drug. Only highly lipid-soluble drugs can enter the brain by crossing the blood-brain barrier.

Drug concentration at the target organ is an important index for therapy and generally has an optimal range. The drug level can be raised by increasing dose, or by more frequent administration, but too high a level could cause toxicity. The drug level at the target organ can also be lowered by elimination through the urine or by metabolic steps that convert the drug to more water-soluble forms. Water-soluble metabolites are eliminated quickly in the urine. Most drugs given orally are lipid-soluble enough to be reabsorbed in the kidneys and are eliminated only slowly in small amounts in the unchanged form in urine (see Figure 1). Therefore drug metabolism is an important factor that controls drug levels in the body, because without the metabolic step the drug usually remains in the body or accumulates if it continues to be taken. Drug metabolism is a biochemical process and involves enzymes; drugs are metabolized sequentially or by parallel pathways to various products called metabolites. Many enzymes have been identified and some are very specific for drugs or substrates, whereas others have broad or less stringent structure requirements (see Table 1).

Many factors can modify drug metabolism. Genetic factors or inherited deficiency of an enzyme could cause accumulation of certain drugs. Increased levels and increased toxicity may be caused by inhibition of drug metabolism by other concurrently administered drugs. Decreased plasma levels of drugs after repeated administration have been observed and this is attributed to increased enzyme activity by a process called induction; autoinduction causes the increased metabolism of the inducing drug and cross-induction refers to the accelerated metabolism of other drugs.

DRUG-METABOLIZING ENZYMES

Drug-metabolizing enzymes change the chemical nature of drugs by inserting oxygen, hydrogen, water, or small molecules such as amino acids and sugar molecules. The resulting metabolites may thus contain hydroxyl (the univalent group or ion OH), or hydrogenated or hydrolysis products, or be

conjugated with sugar or other functional groups. By far the most commonly occurring metabolic step is hydroxylation (the addition of oxygen) by the enzyme oxygenase—and this will be discussed in detail.

OXIDATION BY CYTOCHROME P450 MONOOXYGENASE

Oxygen is vital for living organisms, and enzymatic reactions involving this molecule for drug metabolism are numerous and well characterized. Lipid-solubility is an important factor for absorption across the stomach and intestinal wall, and the insertion of an oxygen atom to lipid-soluble compounds results in hydroxylated groups (-OH) that are more water-soluble than the parent compound. The pioneering work on the oxygenation reaction involved the metabolism of BARBITURATES, a class of centrally acting drugs very popular in the 1950s. A long-acting barbiturate, PHENOBARBITAL, very slowly hydroxylates compared to other barbiturates, such as hexobarbital, pentobarbital, and secobarbital. The oxygenation enzymes involved were named cytochrome P450 after the wavelength of light they absorbed in a spectrophotometer (*P*eak at 450 nanometers [nm]). Subcellular fractionation by centrifugation yielded "microsome" pellets which contained the cytochrome P450 activity. Cytochrome P450 is most abundant in the liver and, before the full nature of cytochrome P450 was known, the microsomal oxygenase was often called mixed function oxidase. Cytochrome P450 consists of a superfamily of enzymes, with wide and sometimes overlapping substrate specificities.

Although phenobarbital is no longer widely used for therapeutic purposes, because of better alternatives with fewer side effects, it is an excellent inducer of certain forms of cytochrome P450 (e.g., the CYP2B family).

Other important drugs of abuse that are metabolized by cytochrome P450 include BENZODIAZEPINES (tranquilizers such as DIAZEPAM [Valium], CHLORDIAZEPOXIDE, alprazolam, triazolam) and OPIOIDS (CODEINE, oxycodone, dextromethorphan). The first group of drugs is hydroxylated and the second group is metabolized by loss of a carbon moiety (dealkylation). The dealkylation reactions are also mediated by cytochrome P450.

Many cytochrome P450 enzymes have been isolated and characterized. With molecular biology techniques, the genetic code DNA has been identified for many cytochrome P450 enzymes. Among these, two forms of cytochrome P450 are known to be deficient in certain individuals. In the mid-1970s, a deficiency of the specific cytochrome P450 called CYP2D6 was independently reported for sparteine (a labor-inducing or antiarrhythmic drug) and for debrisoquine (an antihypertensive agent). Since then, more than thirty clinically useful drugs have been shown to be metabolized by this enzyme. The presence of this cytochrome P450 in a population is polymorphic, that is, some people lack this enzyme. A simple urine test using dextromethorphan, a cough suppressant, is commonly used to identify the enzyme deficiency in a patient. Another cytochrome P450 deficiency involves metabolism of mephenytoin (CYP2C type) but not many drugs are metabolized by this enzyme. The frequency of both deficiencies were first established in Caucasians, and CYP2D6 deficiency was reported to be 7 percent while CYP2C deficiency was 3 percent. Because of the presence of deficient subjects, the population data do not show a bell-shaped normal distribution curve but rather a bimodal distribution indicating polymorphism.

ALCOHOL METABOLISM

ALCOHOL (ethanol) metabolism predominantly involves a type of oxidation called dehydrogenation (loss of hydrogen) and the subcellular fraction called the mitochondria is the major site. Alcohol is metabolized by successive dehydrogenation steps, first producing acetaldehyde and secondly acetic acid. The major organ for alcohol metabolism is the liver. In heavy drinkers, however, alcohol induces another enzyme, cytochrome P450, and the proportion of the metabolism by this route compared to dehydrogenation becomes significant. Because the amount of alcohol ingested must be relatively large to have pharmacological effects, the amount of alcohol exceeds the amount of enzyme, resulting in saturation. Acetaldehyde, in general, is toxic because it is reactive and forms a covalent bond with proteins. When the enzyme that metabolizes acetaldehyde to acetic acid is inhibited by an external agent, acetalaldehyde levels increase and produce a toxic syndrome. Inhibitions of this enzyme,

such as DISULFIRAM (Antabuse), have been used in the treatment of excessive drinking.

TRANSFERASES FOR CONJUGATION/ SYNTHETIC REACTIONS

Products formed by oxidation (e.g., by cytochrome P450) are often metabolized further with small molecules such as glucuronic acid (glucose metabolite) or sulphate. The enzymes involved are called transferases. Other conjugation reactions are carried out by transferases linking glutathione with reactive metabolic products, acetyl-CoA with an amino group on aromatic rings, and glycine (amino acid) with salicylate.

Glucuronic-acid conjugations are catalyzed by various forms of glucuronyl transferases, which appear to have broad substrate specificity. Glucuronide conjugates are very water-soluble and likely to be quickly eliminated via the kidneys. The plasma levels of glucuronide conjugates of oxazepam (a benzodiazepine antianxiety agent) are, however, several-fold higher than the parent drug. This can be explained by the relatively rapid process of conjugation reaction in the liver compared to the renal (kidney) clearance of its conjugate. Because glucuronidation involves a glucose metabolite, which is abundant, the transferase would not reach saturation easily, although sulfo-transferase utilizes the sulphate which is of limited supply via foods and can be saturated. For example, ACETAMINOPHEN (Tylenol) forms both glucuronide and sulfate conjugates and the sulfation process can be easily saturated after a few tablets.

Glutathione conjugation is very important as a detoxification pathway. Unstable or reactive metabolites formed from other metabolic reactions may cause toxicity by reacting with so-called house-keeping enzymes in the body. Glutathione, because of its abundance, can react with these metabolites instead and acts as a scavenger; an epoxide whose formation is catalyzed by cytochrome P450 is detoxified, except in an overdose case, by glutathione transferase. Some epoxide intermediary metabolites have been shown to be ultimate carcinogens, and detoxification by gluthione would be beneficial.

Glycine is the smallest amino acid and the conjugation with salicylic acid (formed rapidly from aspirin) is the major metabolic pathway for salicylates. Salicylate poisoning, especially in children, was very common before the introduction of the child-proof cap for drug containers in the 1960s. The difficulty of treating the salicylate poisoning was due to saturable glycine conjugation; the higher the dose, the slower was the rate of elimination.

Acetylation is also important for the detoxification of carcinogens containing aromatic amines. One form of N-acetyltransferase is polymorphic (people have different forms of the enzyme). The frequency of slow acetylator types shows a large variation ranging from 5 to 10 percent in Oriental and Inuit (Eskimo) subjects to as high as 50 percent in Caucasians and Africans. Drugs affected by this genetic polymorphism are isoniazid (antituberculosis), procainamide (antiarrhythmic), sulfamethazole (antibiotic), and other amine-containing compounds.

CLINICAL CONSEQUENCES

Drug metabolites are often pharmacologically less active than the parent drug. Yet some biotransformation products are active—for example CODEINE is relatively inactive but is metabolized to the active drug MORPHINE. Because the liver is the major site of drug metabolism, acute or chronic liver diseases would alter drug metabolism, resulting in prolonged drug half-lives and effects.

(SEE ALSO: *Complications: Liver (Alcohol); Drug Interaction and the Brain; Drug Interactions and Alcohol*)

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DRUG POLICY FOUNDATION (DPF)

The Drug Policy Foundation (4455 Connecticut Avenue, NW, Washington, DC) is a not-for-profit organization established to stimulate debate about

drug policy in the United States and to oppose the current "war on drugs" approach. It favors shifting from policies emphasizing law enforcement and drug prohibition to ones that either legalize drug use entirely or at least medicalize the distribution of certain drugs. Founded in 1986 by Arnold Trebach, a lawyer and professor at American University in Washington, DC, the foundation reports that its membership had grown to more than 24,000 by 2000. The Drug Policy Foundation has merged with the Lindesmith Center to become the Lindesmith Center-Drug Policy Foundation and is now known by the acronym TLC-DPF. The Lindesmith Center is located in New York City (400 West 59th Street). The Lindesmith Center—Drug Policy Foundation's director is Dr. Ethan Nadelmann, who has taught at Harvard and Princeton Universities and lectured throughout the world on drug policy and international law enforcement.

In 2000 the board members included Ira Glasser, executive director of the American Civil Liberties Union; Dr. Jocelyn Elders, former Surgeon General of the United States; Nicholas Pastore, fellow at the Criminal Justice Policy Foundation; Hon. Robert W. Sweet, Senior Judge for the Southern District of New York and Danny Sugarman, manager of the musical group the Doors and author of several books.

The guiding principle of the organization is harm reduction, an alternative approach to drug policy and treatment that focuses on minimizing the adverse effects of both drug use and drug prohibition. TLC-DPF is deeply involved in educating Americans and others about alternatives to current drug policies on issues ranging from marijuana and adolescent drug use to illicit drug addiction, the spread of infectious diseases, policing drug markets and alternatives to incarceration. It promotes drug policies based on common sense, science, public health and human rights. Particular attention is focused on analyzing the experiences of foreign countries in reducing drug-related harms.

TLC–DPF's policy priorities are predicated on the premise that the current drug war is excessive and ineffective in creating a safer or healthier society. The policy areas include: improving drug education and prevention, especially for young people; ending civil asset forfeiture; shifting current practices away from drug testing toward impairment testing when appropriate; and decriminalizing marijuana.

The organization also administers a grant program that distributes approximately $1.5 million every year to drug policy reform efforts both within the United States and abroad. Since its inception, the grant program has given over $4 million to 306 drug policy reform organizations worldwide. TLC–DPF awards its grants to a wide variety of drug reform organizations, including those that specialize in criminal justice, drug policy, harm reduction, medical marijuana, methadone maintenance, and syringe exchange. TLC–DPF provides three types of funding: project, general support, and technical assistance. For example, in 1998, Positive Health Project (PHP), a Manhattan-based harm reduction agency, received a grant to design and implement New York City's first syringe-exchange media campaign. Using a social marketing firm, PHP designed an ad promoting the value and availability of syringe-exchange services in New York City which was placed inside 1,140 N.Y.C. subway cars for 1 month. The primary goals of the campaign were to educate drug users about the importance of HIV prevention through syringe exchange and thereby increase the use of syringe exchange throughout the city.

The Lindesmith Center Library, located in New York City, is a rapidly growing library housing one of the largest collections on drugs and drug policy in the world. It contains over 10,000 books, reports, government documents, periodicals, videos, and articles from the U.S. and abroad as well as in-depth collections on drug-related policies in Canada, Latin America, Great Britain, Germany, the Netherlands, Switzerland, and Australia. The library also maintains an online library at www.lindesmith.org/library/lib.html.

The Drug Policy Foundation sponsors annual conferences, at which speakers discuss their perspectives on current drug policy, and has enlisted in its cause many individuals prominent in public life. For example, the Thirteenth Annual Conference on International Drug Reform was held in Washington, D.C. in May 2000.

Aside from advocating public policy, the foundation has a legal affairs office that uses the court system to promote change. For example, the office serves as counsel in a federal class action lawsuit on behalf of California physicians and patients to enjoin the federal government from penalizing doc-

tors who recommend medical marijuana to seriously ill patients. The foundation also serves as a consultant to county and state agencies in California to design procedures by which seriously ill people can safely obtain and use medical marijuana.

The organization publishes a bi-monthly newsletter. *The Drug Policy Letter*, and the Drug Policy Foundation Press publishes books and papers that support viewpoints of the foundation. The DPF also receives support from several other foundations not ordinarily associated with the advocacy of drug legalization, such as the John D. and Catherine T. MacArthur Foundation. A major contributor since 1992 has been the Open Society Foundation, a charitable organization that receives its funding from the financier George Soros.

(SEE ALSO: *Prevention Movement; Policy Alternatives*)

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DRUG RESPONSE *See* Causes of Substance Abuse: Drug Effects and Biological Responses

DRUG TESTING METHODS AND CLINICAL INTERPRETATIONS OF TEST RESULTS As interest increases in employment-related drug testing, the technologies and the interpretive skills of analysts continue to evolve. Although recent literature indicates that significant refinements and modifications to drug testing technology have been made, the complexity of drug effects is so great that many problems exist in interpretation of the test results. The most frequent problems that confront the toxicology laboratory relate to developing technology that can determine how much and when the drug was taken, how long after use the tests are capable of showing positive results, the causes and rates of false positive and false negatives, and how tests can be "beaten" by employees. These problems will be discussed and the various laboratory procedures that are used to combat these problems will be examined.

DRUG PROPERTIES: ABSORPTION, DISTRIBUTION, AND ELIMINATION PHASES

Detection of a drug depends largely on its absorption, distribution, and elimination properties. There are various routes of drug administration; oral (e.g., drinking ALCOHOL or swallowing pills), intravenous (e.g., HEROIN injected into a vein) and inhalation (e.g., smoking MARIJUANA; snorting COCAINE; sniffing GLUE). Drugs taken orally are usually the slowest to be absorbed (i.e. the speed at which the drug reaches the brain and other body organs) whereas intravenous and inhalation routes result in the fastest absorption. Once the absorbed drug enters the blood stream it is rapidly distributed to the various tissues in the body. The amount of drug stored depends on the nature of the drug, the quantity, duration of ingestion, the tissue holding the drug and the frequency of use.

Some drugs are fat-soluble and are deposited in fat tissues. For example, δ^9-TETRAHYDROCANNABINOL (THC), the active ingredient in marijuana, is highly fat-soluble, resulting in rapid reductions in blood levels as the drug is being distributed to the various tissues. Blood levels of δ^9-THC peak and start to decline in half the time it takes to smoke a marijuana "joint." Concentrations are known to fall by almost 90 per cent in the first hour. Depending on the amount of drug stored in the fat tissues, detection may be possible in the urine for many days after last use. There are cases where marijuana metabolites have been detected for as long as sixty days after last use, since small amounts from fat go back into blood and appear in the urine. Ethanol or ethyl alcohol (the beverage alcohol) is not fat-soluble but is distributed in the total body water. Since blood is mostly made up of water, the presence of alcohol is easier to detect than fat-soluble drugs like δ^9-THC.

The "absorption" and "distribution" phases are followed by an "elimination" phase. The liver is the major detoxification centre in the body where the drugs are metabolized as blood circulates through this organ. The metabolites are then excreted into the urine through the kidneys. At the same time, drugs deposited in fat tissues are also slowly released into the blood stream and metabolized.

A laboratory technician tests a urine sample for the presence of drugs, 1986. (© Ed Kashi/CORBIS)

Drugs vary by their elimination half-life. An elimination half-life is the amount of time needed for the drug level to fall by 50 percent. Every half-life the drug level falls by 50 percent. Table 1 shows the impact of the half-life on the amount of drug left in the body. At the end of 7 half-lives over 99 percent of the drug will be eliminated from the body. (See Table 2 for drug half-lives). The half-life of a drug is heavily influenced by a variety of factors including the individual's age, sex, physical condition as well as clinical status. A compromised liver and concurrent presence of another disease or drug have the potential of enhancing the toxic effects of the drug by slowing down the elimination process. Under different clinical conditions, however, this process may be speeded up. Therefore, great variation can be found in the half-lives of the same drug.

Approximately six half-lives are required to eliminate 99 per cent of any drug. Because cocaine's half-life is relatively short, averaging one hour, only six hours are needed for elimination of 99 per cent of the drug. On the other hand, cocaine's metabolites have a longer half-life and can be detected for a considerably longer period of time through urine drug assays. Compared to cocaine, PHENOBARBITAL has a much longer half-life of 80-120 hours, so that at least 480 hours (or 20 days) are required to eliminate 99 per cent of the drug. Since there is much variation in the half-life of different drugs and the absolute amount of drug present can be very small, it is crucial that the appropriate body fluid for analysis is selected for testing.

Ethanol is absorbed from the stomach by simple diffusion. Gastric absorption is fastest when strong drinks, distilled spirits containing 40 to 50 percent ethanol by volume are consumed. Dilute beverages, such as beer (4-5% ethanol) or wine (11-12% ethanol) are absorbed slowly. Alcohol is absorbed very rapidly from the small intestines. The essential action of food is to delay gastric emptying and thus slow the absorption process. Typically, studies have shown that peak BAC is reached between 30 minutes and 90 minutes of consumption; earlier on an empty stomach and later on a full stomach. Once absorbed, ethanol rapidly diffuses throughout the aqueous compartments of the body, going wherever water goes.

TABLE 1
Impact of Half-Life

	Amount of drug left in the body	*Amount of drug eliminated*
Start	*100%*	*100%*
End of 1st half-life	50.0%	50.0%
End of 2nd half-life	25.0%	75.0%
End of 3rd half-life	12.5%	87.5%
End of 4th half-life	6.25%	93.75%
End of 5th half-life	3.125%	96.87%
End of 6th half-life	1.56%	98.44%
End of 7th half-life	0.78%	99.22%

TABLE 2
Drug Half-Lives and Approximate Urine Detection Periods*

Drug	*Half-life (t2)*	*Detection period*
Methamphetamine Amphetamine (metabolite of methamphetamine)	12–34 hours 7–34 hours	2–3 days
Heroin Morphine (metabolite of heroin)	60–90 minutes 1.3–6.7 hours	In minutes Opiates positive for 2–4 days (EIA)
6-Mono-acetyl-morphine (MAM)	30 minutes	Few hours
Phencyclidine (PCP)	7–16 hours	2–3 days
Cocaine	0.5–1.5 hours	Few hours
Benzoylecgonine (metabolite of cocaine)	5–7 hours	3–5 days
δ^9-Tetrahydrocannabinol	14–38 hours	90% fall in 1 hour (blood)
δ^9-Tetrahydrocannabinoic acid (marijuana metabolite in urine)		Depending on use, few days to many weeks
Benzodiazepines	Few hours to days	days to weeks, depending on half-life
Diazepam	15–40 hours	2 weeks
Flunitrazepam (rohypnol)	9–25 hours	0.2% excreted unchanged!
Methadone	15–40 hours In chronic patients ~22–24 hours	
Barbiturate (phenobarbital)	35–120 hours	1–2 weeks after last use
Alcohol (ethanol)	Blood levels fall by an average of 4–5 mmol/L/hour (15–18 mg/100 mL)/hour	1.5 > 12 hours depending on the peak blood level. Urine typically positive for an additional 1–2 hour.
Gamma-hydroxybutyrate (GHB)	0.3–1.0 hour	Less than 12 hours

*The detection period is very much dose-dependent. The larger the dose, the longer the period the drug/metabolite can be detected in the urine

Absorption, distribution into different tissues and elimination are dynamic processes and take place simultaneously. The rate of removal of ethanol from the body is the sum of the rates of excretion in urine, breath and sweat, and the rate of the metabolism in the liver and other tissues. In humans, alcohol metabolism follows a "zero" order kinetics, i.e., it is largely independent of alcohol concentration in the blood and its levels decline almost linearly over time. The implication of this is that BAC falls at a constant rate over time. In social drinkers it is from 0.015 to 0.018 percent (15 mg/100mL to 18 mg/100mL) per hour and in heavy drinkers it is typically between 0.018 and 0.025

percent (18mg/100mL to 25mg/100mL) per hour. In the alcoholic patient, the elimination rate is generally higher. In forensic calculations, a rate of 0.015 percent (15mg/100mL) per hour is usually used. In our studies we have found 0.018 percent (18mg/100mL) per hour to be the average rate of metabolism. The larger the dose of alcohol given, the longer the duration of the measurable blood alcohol concentration.

SELECTION OF DRUGS TO BE TESTED

A number of different criteria can be applied to the drug(s) or category of drugs that should be tested or monitored. Drug availability, clinical effects and robustness of the analytical method(s) used for analysis are probably the most important.

Availability. Prescription patterns of psychoactive and other drugs vary from place to place and country to country. Abuse of BENZODIAZEPINE nitrazepam is common in Europe but almost unknown in North America, since it is not sold here. The psychoactive chemical CATHINON (cathine), the active ingredient in the leaves of the KHAT plant, is chewed in northeast Africa, is not a problem in North America. CODEINE, an OPIOID available in Canada as OVER-THE-COUNTER preparations, is sold only by prescription in the United States.

A wide availability of "legal" STIMULANTS poses an interesting problem since they are a common finding in accident victims. A study carried out by the U.S. National Transportation Safety Board from October 1987 to September 1988 showed that over-the-counter stimulants—such as ephedrine, pseudoephedrine and phenylpropanolamine—were commonly found among drivers killed in heavy truck accidents. Amongst the eight States that participated in this safety study almost all AMPHETAMINE use was in the California region. Similar findings are also reported from emergency rooms over the past five years as well as from admissions in a trauma unit due to motor-vehicle accidents. All this suggest that drug use varies not only from place to place but also region to region within a given country.

Thus, the selection of a drug to be tested and monitored, appropriate for one country and place, may not necessarily be appropriate for another country.

Clinical Effects. Drugs that manifest abuse potential and impair behavior such that job performance can be affected are prime candidates for testing or monitoring in the workplace. Alcohol and cocaine are examples of this.

Analytical Methods. A false positive finding can have a serious impact on the livelihood of the person being tested. Therefore, special attention needs to be paid to the testing methods. Ideally the analytical method should be specific for the drug being tested (i.e., no false positive), easy and inexpensive to perform. Confirmation methods should also be readily available. Availability of technical and scientific expertise to perform the tests is also essential.

Interpretation of the analytical results also needs to be carefully considered as even a normal diet can result in a positive drug identification. For example, poppy seed ingestion can result in a true positive *analytical* result (OPIATES, like heroin, are derived from the poppy plant PAPAVER SONIFERUM) but it is a false positive for drug use. Some ethnic diets may also lead to these confounding problems, as when food containing poppy seeds is eaten during Ramadan.

What should be analyzed? Ideally the analysis should look for the parent drug rather than its metabolite, although this may not always be possible as some drugs are very rapidly metabolized (e.g., heroin metabolism to MORPHINE). Sensitivity of the analytical procedure should be dictated by the drugs' psychoactive pharmacological properties. If the drug is shown to be devoid of abuse potential then its detection beyond the time of pharmacological activity, although important in the clinical management of the patient, does not necessarily serve a useful purpose for a workplace drug screening programme.

The guidelines developed by the NATIONAL INSTITUTE ON DRUG ABUSE in April 1988, address five "illegal" drugs: marijuana, PHENCYCLIDENE, AMPHETAMINE, cocaine and heroin. Rapid screening methods that allowed for "mass screening" were available at that time, as were the confirmation methods for these five drugs. Mood altering substances such as benzodiazepines, BARBITURATES and some stimulants such as antihistamines are at present excluded from these regulations in the United States. This is probably due to the wide availability of these drugs as medications within the general population and the technological re-

quirements for screening and monitoring of these drugs.

TYPES OF TESTING: BLOOD, URINE, AND HAIR SPECIMENS

Blood and urine are the most commonly used biological fluids in the analysis for drugs other than alcohol. Blood, obtained by an invasive procedure, is available only in small quantities and drug concentration levels in blood are typically low. Urine is the preferred sample of choice as it is available in larger volumes, contains the metabolite and requires less invasive procedures in its collection. Both sampling procedures, however, are limited in their ability as they only determine the absolute amount of drug present in the fluid being examined. This quantity is dependent upon the amount of the drug used, when it was last used, as well as the half-life of the drug.

Recently, hair samples have been used to detect drug use. A number of technical problems must be overcome before hair can be used as a definitive proof of drug use. Hair treatment and environmental absorption are but two of the many concerns and problems that have been cited. An advisory committee of the Society of Forensic Toxicology has recently reported that "The committee concluded that, because of these deficiencies, results of HAIR ANALYSIS alone do not constitute sufficient evidence of drug use for application in the workplace."

Various body fluids such as sweat, saliva, blood, urine and breath, have been used for alcohol analysis. Breath, though not a body fluid, is commonly used by law enforcement authorities. Although a number of variables can effect breath/blood ratio, a 2100:1 alveolar breath/blood conversion ratio has been used and accepted for use with BREATHALYZERS. Breath-testing equipment calibrated with a blood:breath conversion factor of 2100 consistently underestimate actual BLOOD ALCOHOL CONCENTRATIONS (BAC). Accuracy of breath analysis results is subject to various instruments and biological factors. Potential errors in breath analysis can also be caused by the presence of residual alcohol in the mouth. Immediately after drinking there is enough alcohol vapour in the mouth to give artificially high concentrations on breath analysis. Generally this effect disappears twenty minutes after drinking but high values for as long as forty-five minutes have been reported.

As of the early 1990s, all existing technologies are limited in terms of determining how much or when the drug was consumed.

Blood and saliva concentrations reflect the current blood alcohol concentration, but generally a blood sample is used in hospitals to access the patient in the casualty wards. In programmes requiring monitoring of alcohol use, urine is probably the sample of choice. Urine alcohol concentration, which represents the average blood alcohol concentration between voiding, has the potential of being "positive" while the blood may be "negative."

MEASURING IMPAIRMENT

Except for alcohol, the degree to which a person is influenced or impaired by a drug at the time of the test cannot be determined from test results alone. Correlations between positive blood levels and degree of impairment are usually stronger than correlations between urine levels and degree of impairment; however, neither blood nor urine tests are sufficiently accurate to indicate impairment even at high levels of concentration. Human studies using marijuana and cocaine have shown that a "perceived high" is reached *after* the drug concentration has peaked in the blood. Generally, blood can only show positive results for a short time after drug consumption, whereas urine can be positive for a few days to weeks after last use. For example, metabolites of δ^9-THC (active ingredient in marijuana) that are lipid-soluble can be detected in the urine from a few days to many weeks, depending on the drug-habit of the user. Excretion of the drug in urine and its concentrations are also affected by several factors, such as dilution and pH (acidity) of the urine. I have seen many cases where a strong, positive urine sample for CANNABINOIDS was found in the morning, a borderline positive in the afternoon, followed by a strong positive the next morning; I have also seen similar cases with respect to phenobarbital.

A positive urine test cannot reveal the form in which the drug was originally taken—or when and how much was taken. For example, CRACK-cocaine, impure cocaine powder or cocaine paste (which can be smoked, inhaled, injected or chewed) all give the same result in the urine test. The consumption of poppy seeds has been reported

to give positive results for opiate use, because some seeds contain traces of opiates and some have been known to be contaminated with OPIUM derivatives. Similarly, consumption of herbal COCA tea has resulted in positive results for cocaine use. These diverse incidences illustrate the difficulties involved in measuring impairment using urine results.

The problem of interpreting urine-test results is one of the major bases of concern for restricting their use in the employment setting. Even the effectiveness of preemployment drug-screening tests, due to the difficulties in interpretation is being questioned. Based on a study of 2,229 pre- employment drug screening tests and follow-up, one group of researchers have come to the following conclusion "our findings raise the possibility that a preemployment drug screening may be decreasingly effective in predicting adverse outcomes associated with marijuana use after the first year of employment". They make a similar comment about cocaine.

There is no threshold for alcohol effects on performance or motor- vehicle-accident risk. Although the effects of alcohol on impairment and crash risk appear more dramatically above 80mg/100mL, a review of literature would suggest that impairment may be observed at levels as low as 15mg/100mL. It is not possible to specify a blood alcohol concentration level above which all drivers are dangerous and below which they are safe or at "normal" risk. An author of a major literature review on the behavioral effects of alcohol concluded that "that alcohol sensitivity can vary from time to time, person to person, and situation to situation, the setting of a "safe" BAC will always be arbitrary, being based on a low, but a non-zero, incidence of effects below that level" and "the most striking feature to emerge from any review of the effects of alcohol on behaviour is the marked lack of agreement between authors, amounting, in many instances, to direct contradiction. This is especially true for the effects of smaller dose."

"Legal" BAC levels differ in different countries. Some even have more than one legal limit over which the driver of a vehicle is considered as "impaired". Some European countries have 50mg/100ml others have 80mg/100ml as their legal limits. In the United States, the legal limits vary from 80mg/100mL to 100mg/100mL in different states, but employees who are regulated by the U. S. Department of Transportation have a BAC legal limit of 40mg/100mL. In Canada there are also two limits: 50mg/100mL and 80mg/100mL. BAC levels between 50mg/100mL and 80mg/100mL call for suspension of driving privileges but above 80mg/100mL are subject to criminal charges.

URINE TESTING METHODS

Urine is the most commonly used fluid for drug screening. The methods most commonly used in toxicology laboratories are: *immunoassay*, *chromatographic* and *chromatography coupled with mass spectrometry*. These methods vary considerably with respect to their sensitivity and reliability. Thin-layer chromatography is least expensive, gas chromatography coupled with mass spectrometry (GC/MS), which is considered as nearly perfect or "gold standard", is the most expensive. Table 2 summarizes the various methods.

Immunoassays (EIA, EMIT, FPIA, CEDIA and KIMS). Immunoassay methods are used for preliminary screening (i.e., initial screening). Since these methods are based on an antibody-antigen reaction, small amounts of the drug or metabolite(s) can be detected. Antibodies specific to a particular drug are produced by injecting laboratory animals with the drug. These antibodies are then tagged with markers such as an enzyme (enzyme immunoassay, EIA), a radio isotope (radioimmunoassay, RIA) or a fluorescence (fluorescence polarization immunoassay, FPIA) label. Reagents containing these labelled antibodies can then be introduced into urine samples, and if the specific drug against which the antibody was made is present, a reaction will occur. RIA is the oldest immunoassay method used to detect drugs. The major drawback of this method is that it requires a separation step and generates radioactive waste. RIA also requires special equipment to measure radioactivity.

Typically, immunoassays are designed for a *class* of drugs. Thus, their specificity (the ability to detect the presence of a *specific* drug) is not very good, since substances that have similar chemical structures will "cross react" and give a false positive reaction. For example, the immunoassay method for cannabinoids was developed to detect the carboxylic acid metabolite of δ^9-THC. Yet, there is a suggestion in the literature that some nonsteroidal anti-inflammatory drugs, such as ibuprofen (a nonprescription drug in the U. S. and

Canada) and naproxyn give random or sporadic false positive results for cannabinoids. Cough-syrup codeine will also give a positive reaction for the morphine (a metabolic product of heroin use) immunoassay and many antihistamines that are available over-the-counter may yield positive reactions for amphetamines. While some reagent manufacturers claim to have overcome many of these cross-reactivity problems, confirmation by a non-immunoassay method is very important.

Urine test kits, designed to detect drugs, have been available in North America for the past few years. More recently, single and multiple test immunoassay kits designed for home and on-site testing have also been introduced. These kits generally carry a cautionary disclaimer that positive test results must be confirmed by the reference GC/MS method. When used in the non-laboratory environment, they are prone to procedural inaccuracies, poor quality control, abuse and misinterpretations. Therefore, these kits should be used with great caution. The risk of labelling a person with a false positive is high without the accompanying confirmatory analysis. Table 3 summarizes the advantages and disadvantages of immunoassay testing.

Chromatographic methods. Separation of a mixture is the main outcome of the chromatographic method. For illustrative purposes, if one were to put a drop of ink on a blotting paper and hold the tip of the paper in water, one would observe the water rise in the paper. After a period of time and under the right conditions, the single ink spot would separate into many different compounds (spots) of different colours (blue ink is a mixture of many dyes). This process, where a mixture of substances is separated in a stationary medium (filter paper), is called chromatography. The types of chromatographic processes used in the analysis of drugs include thin-layer, gas, and liquid chromatography as well as a combination of gas or liquid chromatography with mass spectrometry.

Of the several chromatographic methods, thin layer Chromatography (TLC) is the one most similar to the ink separation example mentioned above. This method requires extensive sample preparation and technical expertise on the part of the analyst, but it is inexpensive and very powerful if used properly. With the exception of *Cannabis*, which requires separate sample preparation, a large number of drugs (e.g., cocaine, amphetamine, codeine and morphine) can be screened at the same time. By combining different TLC systems, a high degree of specificity can be obtained, although the training of the analyst is crucial because of the subjectivity involved in interpreting the results. To identify positive TLC "spots," the technologist looks for the drugs and or its metabolite pattern, often by spraying with reagents that react to form different colors with different drugs. The trained technologist can comfortably identify more than forty different drugs.

TABLE 3
Common Drug-Testing Methods

1.	*Immunoassays*
	Enzyme immunoassay (EIA)
	Enzyme-multiplied immunoassay technique (EMIT)
	Fluorescence polarization immunoassay (FPIA)
	Radio immunoassay (RIA)
	Kinetic interaction of microparticles in solution (KIMS)
	Cloned enzyme donor immunoassay (CEDIA)
	Rapid slide tests (point-of-care testing)
2.	*Chromatographic Methods*
	Thin-layer chromatography (TLC)
	Liquid chromatography (HPLC)
	Gas chromatography (GC)
3.	*Chromatography/Mass Spectrometry*
	Gas chromatography/mass spectrometry (GC/MS)
	Liquid chromatography/mass spectrometry (HPLC/MS)

Similar to TLC, gas chromatography (GC) requires extensive sample preparation. In GC, the sample to be analyzed is introduced via a syringe into a narrow bore (capillary) column which sits in an oven. The column, which typically contains a liquid adsorbed onto an inert surface, is flushed with a carrier gas such as helium or nitrogen. (GC is also sometimes referred to as gas-liquid chromatography (GLC). In a properly set up GC system, a mixture of substances introduced into the carrier gas is volatilized, and the individual components of the mixture migrate through the column at different speeds. Detection takes place at the end of the heated column and is generally a destructive process. Very often the substance to be analyzed is "derivatized" to make it volatile or change its chromatographic characteristics.

In contrast to GC, high pressure liquid chromatography (HPLC) a liquid under high pressure is used to flush the column rather than a gas. Typically, the column operates at room or slightly above room temperature. This method is generally used for substances that are difficult to volatilize (e.g., STEROIDS) or are heat labile (e.g., benzodiazepines).

Gas chromatography/mass spectrometry(GC/MS) is a combination of two sophisticated technologies. GC physically separates (chromatographs or purifies) the compound, and MS fragments it so that a fingerprint of the chemical (drug) can be obtained. Although sample preparation is extensive, when the methods are used together the combination is regarded as the "gold standard" by most authorities. This combination is sensitive i.e., can detect low levels, is specific, and can identify all types of drugs in any body fluid. Furthermore, assay sensitivity can be enhanced by treating the test substance with reagents. When coupled with MS, HPLC/MS is the method of choice for substances that are difficult to volatilize (e.g. steroids).

Given the higher costs associated with CG/MS, urine samples are usually tested in batches for broad classes of drugs by immunoassays and positive screens are later subjected to confirmation by this more expensive technique.

Table 4 gives a summary of the advantages and disadvantages of each method of chromatographic drug testing and Table 5 compares all the methods of testing. The initial minimal immunoassay and GC/MS (cut-off) levels for five drugs or classes of drugs as suggested by the U.S. National Institute of Drug Abuse, are listed in Table 6.

Procedures for alcohol testing. Since the introduction of the micro method for alcohol analysis

TABLE 4
Advantages and Disadvantages of Immunoassays

Advantages

1. Screening tests can be done quickly because automation and batch processing are possible.
2. Technologists doing routine clinical chemistry testing can be easily trained.
3. Detection limits are low and can be tailored to meet the program screening requirements. For example, lower detection thresholds can be raised to eliminate positives due to passive inhalation of marijuana smoke.
4. Immunoassays are relatively inexpensive, although the single-test kits can be very expensive when quality assurance and quality control samples are included.
5. Immunoassays do not require a specialized laboratory. Most clinical laboratories have automated instruments to do the procedures.

Disadvantages

1. Although the tests are useful for detecting classes of drugs, specificity for individual drugs is weak.
2. Since the antibody is generated from laboratory animals, there can be a lot-to-lot or batch-to-batch variation in the antibody reagents.
3. Results must be confirmed by another nonimmunoassay method.
4. A radioactive isotope is used in RIA that requires compliance with special licensing procedures, use of gamma counters to measure radioactivity, and disposal of the radioactive waste.
5. Only a single drug can be tested for at one time.

TABLE 5
Summary of Chromatographic Methods

Advantages

All the chromatographic methods are specific and sensitive and can screen a large number of drugs at the same time.

TLC	Negligible capital outlay is needed.
GC	The procedure can be automated.
HPLC	Of the chromatographic procedures, this has the easiest sample preparation requirements. The procedure can be automated.
GC/MS	This is the "gold standard" test. Computerized identification of fingerprint patterns makes identification easy. The procedure can be automated. This is currently the preferred method for defense in the legal system.

Disadvantages

All chromatographic methods are labor-intensive and require highly trained staff. Although the chromatographic methods are specific, confirmation is still desirable.

TLC	Interpretation is subjective, hence, training and experience in interpretation capabilities of the technologist are crucial.
HPLC or GC	Equipment costs are high, ranging between $25,000 to $60,000, depending on the type of detector and automation selected (1994 $)
GC/MS	Equipment costs are the highest, ranging from $120,000 to $2000,000, depending on the the degree of sophistication required (1994 $). Due to the complexity of the instrument, highly trained operators and technologists are required.

TABLE 6
Cut-off Levels for Initial and Confirmatory Tests[a]

Test	*Initial Test*	*Confirmatory Test*
THC metabolite[b]	100 ng/mL	15 ng/mL
Cocaine metabolites[c]	300 ng/mL	150 ng/mL
Opiate metabolites[d]	2000 ng/mL	
Morphine		300 ng/mL
Codeine		300 ng/mL
Phencyclidine (PCP)	25 ng/mL	25 ng/mL
Amphetamines	1000 ng/mL	
Amphetamine		500 ng/mL
Methamphetamine		500 ng/mL
Alcohol	10 mg/100 mL	10 mg/100 mL

[a]April 1988, National Institute of Drug Abuse (NIDA) Guidelines, SAMHSA 1998.
[b]THC metabolite is 11-nor-delta-9 THC carboxylic acid.
[c]Cocaine metabolite is benzoylecgonine.
[d]25 ng/mL if immunoassay is specific for free morphine.

in blood by Widmark in 1922, many new methods and modifications have been introduced. The distillation/oxidation methods are generally *nonspecific* for alcohol (ethanol), whereas biochemical methods (spectrophotometric) using alcohol dehydrogenase (ADH) obtained from yeast and the gas chromatographic method that are currently used are specific for ethanol. The radiative attenuation energy technique and those using alcohol oxidase method are non-specific and will detect not only ethanol but also other alcohols. The recently introduced alcohol dipstick based on the ADH enzyme system is not only specific for ethanol, but also sensitive and does not require instrumentation. It can be used for the detection of ethanol in all body fluids and can provide semi-quantitative results in ranges of pharmacological-toxicological interest. Alcohol dipsticks are being used in a number of laboratories as a screening device.

Breath can be analyzed by using a variety of instruments. Most of the instruments used today detect ethanol by using thermal conductivity, colorimetry, fuel cell, infrared or gas chromatography. Typically in most countries, local statutes define the instrument and method that can be used for evidenciary purposes. A variety of breathalyser instruments ranging in costs from $100 to $1000 are available to do the test. These instruments are compact and portable. Canadian law enforcement authorities use the breathalyser ''Alert'' which can give a ''pass'' or ''fail'' result as a roadside alcohol-screening device. The ''failed'' person is generally subjected to a ''Borkenstein'' breathalyser to measure the BAC before any charges are brought. Many devices are available to preserve the breath sample for later analysis if a breathalyser is not available immediately. In forensic laboratories, gas chromatography (North America) or biochemical procedures (many European countries) are used to analyze biological samples.

Blood samples that cannot be analyzed soon after collection should have sodium fluoride (NaF) added as a preservative. Alcohol dehydrogenase (ADH), the enzyme responsible for the oxidation of alcohol, is also present in the red blood cell and will slowly metabolise the alcohol, causing its concentration to drop if the preservative is not added. Large amounts of alcohol can be produced *in-vitro* in the urine samples of diabetic patients if samples are not processed immediately or properly preserved.

INTERPRETATIONS OF TEST RESULTS

False negatives. A positive or negative result is highly dependent on the sensitivity of the drug detection method. A false negative occurs when the drug is present but is not found because the detection limit of the method used is too high or the absolute quantity of the drug in the specimen is too low.

Large amounts of fluids consumed prior to obtaining a sample for analysis can affect detection of drugs in urine samples. Under conditions of dilution, although the absolute amount of drug or metabolite excreted maybe the same over a period of time, the final concentration per millilitre will be reduced and may give a false negative result. Acidity levels in the urine may also affect the excretion of the drug into the urine. In some cases elimination is enhanced, whereas in other cases, the drug is reabsorbed.

Several measures can be used to decrease the likelihood of obtaining a false negative result. First, sensitivity of the method can be enhanced by analyzing for the drugs' metabolites. Heroin use, for example, is determined by the presence of its metabolite, morphine. Increasing the specimen volume used for analysis or treating it with chemicals can also make laboratory methods more sensitive. Studies have shown that a 5mg dose of Valium® is usually detected for three to four days; however, when these improved methods are utilized, sensitivity can be increased, such that, the same dose can be detected for up to 20 days. One important drawback of such high sensitivities is, that estimates of when the drug was taken are far less accurate.

False positives. A false positive occurs when results show that the drug is present, when in fact it is not. False-positive tests are obtained if an interfering drug or substance is present in the biological fluid and it cross-reacts with the reagents. An example of this is Daypro (oxaprozin) will give a false positive for benzodiazepines. Other substances may have a metabolite that will give a positive reaction. An example of this is Selegiline, an antiparkinson drug, which has amphetamine as one of its metabolites. Although this would be analytically a true positive, it is a false positive from a drug abuse perspective. As discussed in the previous section on immunoassay, an initially positive test based on an

TABLE 7
Comparison of All Testing Methods

	EMIT FPIA	*RIA*	*TLC*	*GC HPLC*	*GC/MS*
Ease of sample preparation	X	X		X	
Less highly trained technologists required	X	X			
Limited equipment required	X	X	X		
Low detection limits	X	X	X	X	X
Adjustable lower threshold	X	X			
Highly specific and sensitive			X	X	X
Computerized identification possible					X
Screen for several drugs at a time			X	X	X
Procedure can be automated	X	X		X	X
Special atomic energy license required		X			
Confirmation of results required	X	X	X	X	
Interpretation is subjective			X		

immunoassay technique should always be confirmed with an nonimmunoassay method. A confirmed positive finding only implies that the urine sample contains the detected drug and nothing more.

At times false positives are attributable to ingested substances such as allergy medications. Some authors have suggested that employees subject to drug screening refrain from using popular over-the-counter medications, such as Alka-Seltzer Plus and Sudafed, because they have caused false-positives. Some natural substances such as herbal teas and poppy seeds can also give positive responses to screens. These may be analytically true positives but need to be distinguished from those due to illegal drug use. In some instances, false-positives have been due to mistakes or sabotage of the chain of custody for urine samples.

COMMON ADULTERATION METHODS

The method of switching "clean" urine for "dirty" urine; resubmitting one's own or urine that is provided by someone else are the most common ways to fool the drug screening system. A number of entrepreneurs have attempted to bypass urine-specimen inspection by substituting clean urine. For example, a company in Florida sells lyophilized (freeze dried) clean urine samples through newspaper and magazine advertisements. Hiding condoms containing "clean" urine on the body or inside the vagina is another common trick.

Some have substituted apple juice and tea in samples for analysis. Patients are known to add everything from bleach, liquid soap, eye-drops, and many other household products, hoping that their drug use will be masked. Others may hide a masking substance under their fingernails and release it into the urine specimen. Another method is to poke a small hole into the container with a pin so that the sample leaks out by the time it reaches the laboratory.

Since addition of table salt (NaCl) or bleach to the urine is a common practice, many laboratories routinely test for Na and Cl in the urine. Liquid soap and crystalline drain cleaners that are strong alkaline products containing sodium hydroxide (NaOH) are also used to adulterate the urine sample. These contaminators can be detected by checking for high levels of pH in the urine sample. In-vivo alkalizing or acidifying the urine pH can also change the excretion pattern of some drugs

including amphetamines, barbiturates and phencyclidine (PCP).

Water-loading (drinking large amounts of water prior to voiding) poses an interesting challenge to testing laboratories. Specific gravity has been used to detect dilution; however, the measurement range is limited so it is not yet useful. Creatinine levels on random urine samples appear to be a promising method for detection of water-loading. A number of adulteration methods are being advertised on the Internet. Invariably, one of the instructions for adulteration is to drink copious amounts of fluids to bring about *in-vivo* dilution or water loading. Some Internet sites even sell adulterants that can be added to the urine. Typically these products either try to oxidise the drug present or try to change the pH of the urine to interfere with the analytical method. Most of the laboratories involved in drug testing routinely test for the various adulterants. To detect resubmitted samples a "urine fingerprinting" method using dietary components has been described.

Drug users are very resourceful and their ingenuity should not be underestimated. To reduce the opportunities for specimen contamination, some workplaces require that employees provide a urine sample under direct supervision. Another technique used to detect any sample adulteration is to take the temperature of the sample. In a study, we took the temperature of urine samples when taken within one minute of voiding; it falls between 36.5°C and 34 degrees Celsius, reflecting the inner body core temperature. It is very difficult to achieve this narrow temperature range by hiding a condom filled with urine under the armpit or adding water from a tap or toilet bowl to the urine sample. It is important that the temperature of the specimen be measured immediately after the sample is taken, since it can drop rapidly.

LABORATORY PROCEDURAL AND SECURITY STANDARDS

It is important that the laboratory drug testing facility has qualified individuals who follow a specific set of laboratory procedures and meet recommended security standards.

SUMMARY AND CONCLUSION

In this paper, major issues related to drug testing are discussed. For example, drug-testing techniques measure drug presence but are not sophisticated enough to measure impairment from drug use. It is also very difficult to determine the route of drug administration, quantity, frequency, or when the drug was last taken.

Selection of the drug to be tested should depend on the local availability of the drug, its abuse potential, and clinical effects, as well as the available analytical technology and expertise in testing and interpretation of the laboratory results. The most sophisticated drug-testing approach, gas chromatography in combination with mass spectrometry, is considered as a gold standard and thus utilized in confirmatory testing. Typically GC/MS is preceded by a rapid immunoassay method to eliminate the majority of negative samples.

Despite the existence of sophisticated drug-testing methods, incorrect test results can still occur. These can be due to the presence of interfering substances or adulteration of the urine sample. Patients have been known to adulterate urine samples to avoid drug detection. A number of techniques can be employed to reduce the likelihood of obtaining erroneous results, as well as detect adulterated urine samples. [Parana "positive" drug finding can have a serious impact on the livelihood of an individual, therefore the performance of these tests should adhere to the strictest laboratory standards of performance. Only qualified and experienced individuals with proper laboratory equipment should perform these analyses. Standards of laboratory performance must meet local legal and forensic requirements. Access to the patient samples as well as laboratory records must be restricted in order to prevent tampering of samples and results. To maintain confidentiality and assure proper interpretation of results, the results must be communicated only to the physician reviewing the case/patient. Chain of custody and all documents pertaining to the urine sample must be maintained so that they can be examined in case of a legal challenge. Laboratory must have a complete record on quality control. Finally, specific initial and confirmatory testing requirements should be met.

BHUSHAN M. KAPUR

DRUG TRAFFIC CONTROL *See* Drug Interdiction

DRUG TRAFFICKING *See* International Drug Supply Systems

DRUG TYPES There are many ways to classify drugs, depending on the purposes for the classification. For example, a classification can be based on the chemical properties of drugs and may actually disregard the effects the drugs have on the body, or it may be based on legal principles, such as legal versus illegal, or prescription versus over-the-counter (prescription not needed). For purposes of discussion and teaching, the various drugs that are used and abused by humans for nonmedical purposes are usually grouped into several major categories, each based on their pharmacological actions and their subjective effects. Although the mechanisms of action may vary among drugs within a single category, the general subjective effects of the drugs are similar.

The major categories include: (1) ethanol (ALCOHOL); (2) NICOTINE and tobacco: (3) central nervous system depressants (BARBITURATES, BENZODIAZEPINES); (4) central nervous system stimulants (AMPHETAMINES, COCAINE); (5) cannabinoids; (6) OPIODS (MORPHINE, HEROIN, METHADONE); (7) psychedelics (LSD, MESCALINE); (8) INHALANTS (glue, nitrous oxide); (9) arylcyclohexylamines (PCP). Some categorizers might put cocaine and the amphetamines into separate categories and group alcohol and the central nervous system depressants together. Some might have a separate category for CAFFEINE; others, one for "DESIGNER DRUGS" (such as MDMA), and refer to them as *entactogens*. There might also be a miscellaneous category, where drugs such as BETEL NUT, KAVA-KAVA, or NUTMEG would be included.

Drugs from the various categories are described below in terms of their pharmacology, abuse, DEPENDENCE, and WITHDRAWAL as well as their toxicity. The legal and readily available drugs (alcohol and tobacco) are described first because the worldwide use and abuse of these drugs is far more widespread than *all* the other categories of abused drugs combined. The ill health associated with the ongoing use of alcohol and tobacco has become a far-reaching problem—not only because of the vast numbers of people who suffer and die each year from the toxic effects of these drugs but also because of the financial drain in terms of employee absenteeism as well as the staggering increases in annual health-care costs. Prescription drugs are covered next, since more prescriptions are written for diazepam (Valium) and the related benzodazepines each year than for any other drug.

The illegal drugs are then discussed. Although the illicit use of heroin, cocaine, and other drugs remains a major social, legal, financial and health problem in the United States today, the proportion of the population physically dependent on these drugs is actually relatively low when compared to the legal drugs listed above. Finally, it is important to take into consideration the fact that individuals often do not restrict their drug use only to drugs within a single category. Alcoholics typically smoke cigarettes and often use benzodiazepines as well. Heroin users also smoke and may consume alcohol and other sedatives, as well as CANNABIS and stimulants in some instances. Multiple-drug use is, therefore, a relatively common occurrence for those using legal and/or illegal drugs.

ETHANOL

Although alcohol (ethyl alcohol, called ethanol) has been in use since prehistory and worldwide throughout recorded history, it is generally accepted that its therapeutic value is extremely limited and that chronic ALCOHOLISM is a major social and medical problem. Perhaps 65 percent of all adults in the United States use alcohol occasionally. Hundreds of thousands of individuals suffer and die each year, however, from complications associated with chronic alcoholism—and tens of thousands of innocent individuals are injured or killed each year in alcohol-related traffic ACCIDENTS. Therefore, alcoholism is a far-reaching problem, affecting the lives of individuals who consume ethanol as well as those who do not. Although alcohol is considered by many people to be a stimulant drug because it typically releases an individual's latent behavioral inhibitions (i.e., through disinhibition), alcohol actually produces a powerful primary and continuous depression of the central nervous system similar to that seen with general anesthetics. In general, the effects of alcohol on the central nervous system are proportional to the

blood concentrations of the drug. Initially, MEMORY and the ability to concentrate decrease, and mood swings become more evident. As the intoxication increases, so does the impairment of nervous function until a condition of general anesthesia is reached ("passing out" or "sleeping it off"). There is little margin of safety, however; between an anesthetic close of ethanol and severe respiratory depression (unconsciousness or coma).

In chronic (long-term) alcoholism, brain damage, memory loss, sleep disturbances, psychoses, and increased seizure susceptibility often occur. Chronic alcoholism is also one of the major causes of cardiomyopathy (heart disease) in the United States due to ethanol-induced, irreversible damage to the heart muscle. Ethanol also stimulates the secretion of gastric acid in the stomach and can contribute to the production of ulcers of the stomach and gastrointestinal system. One of the primary metabolic products of ethanol is acetaldehyde. In chronic alcoholism, acetaldehyde can accumulate in the liver, resulting in hepatitis and cirrhosis of the liver. Finally, the long-term use of alcohol can result in a state of PHYSICAL DEPENDENCE. With relatively low levels of dependence, withdrawal from alcohol may be associated with rather minor problems such as SLEEP disturbances, ANXIETY, weakness, and mild tremors. In more severe dependence, the alcohol withdrawal syndrome includes more pronounced tremors, seizures, and DELIRIUM, as well as a number of other physiological and psychological effects. In some cases, this withdrawal can be life-threatening.

Since alcohol has CROSS-TOLERANCE with other central nervous system (CNS) depressants (i.e., ethanol shares many of the same biological effects as other CNS depressants), benzodiazepines or barbiturates can be substituted for ethanol to successfully decrease the severity of the alcohol withdrawal syndrome. Longer-acting benzodiazepines and related drugs can be used as an ethanol substitute, and the dose of the benzodiazepine can then be gradually reduced over time to attenuate or prevent the occurrence of convulsions and other potentially life-threatening toxic reactions generally associated with alcohol withdrawal.

As outlined above, the chronic use of ethanol can result in a wide range of toxic effects on a variety of organ systems; however, the mechanisms through which ethanol produces its varied effects are not clearly understood. The anesthetic or central nervous system depressant effects may result, in part, from general changes in the function of ion channels that occur when ethanol dissolves in lipid (fat) membranes. Other research suggests that alcohol may interact with specific receptors—binding sites associated with the inhibitory NEUROTRANSMITTER GAMMA-AMINOBUTYRIC ACID (GABA), in a manner somewhat analogous to other central nervous system depressants (e.g., benzodiazepines or barbiturates). Since an ethanol RECEPTOR site has not yet been conclusively identified, specific receptor AGONISTS and ANTAGONISTS are not yet available for the treatment of ethanol intoxication, WITHDRAWAL, and abstinence. The drug DISULFIRAM (Antabuse) is sometimes used in the treatment of chronic ALCOHOLISM, although it does not cure alcoholism. Rather, disulfiram interacts with ethanol to alter the intermediate metabolism of ethanol, resulting in a five- to tenfold increase in plasma acetaldehyde concentrations. Those who drink while on disulfiram experience the acetaldehyde syndrome—vasodilatation, headache, difficulty breathing, nausea, vomiting, sweating, faintness, weakness, anti vertigo. Taking the drug helps persuade alcoholics to remain abstinent, since they realize that they cannot drink ethanol for at least three or four days without provoking ill-effects.

NICOTINE AND TOBACCO

TOBACCO was first introduced to Europe by the crews that accompanied Columbus to the New World, and by the middle of the nineteenth century, tobacco use had become widespread. By the 1990s, almost 30 percent of the adults in the United States were still regular tobacco smokers. This relatively high use of tobacco continues despite the growing warnings that are based on a wealth of scientific evidence linking cigarette smoking to numerous life-threatening health disorders, including lung CANCER and heart disease. The constituents of tobacco smoke that most likely contribute to these health problems include carbon monoxide, NICOTINE, and "tar." However, nicotine also appears to be the primary component of tobacco smoke that promotes smoking. In regular cigarette smokers, nicotine facilitates memory, reduces aggression, and decreases weight gain. Each of these effects could, by itself, provide a rationale for continued tobacco use since most individuals find increased alertness and memory, decreased irritabil-

ity, and decreased weight gain to be somewhat pleasant or desirable; however, these effects may actually be secondary to the primary reinforcing effects of nicotine itself. Nicotine is self-administered by laboratory animals, and in laboratory settings, smokers report that the intravenous injection of nicotine produces a pleasant feeling on its own. It is of interest to note, however, that nicotine is aversive to nonsmokers, often resulting in dizziness, nausea, and vomiting. TOLERANCE rapidly develops to these unpleasant effects in tobacco smokers, however.

Although nicotine obviously binds to nicotinic receptors associated with the neurotransmitter ACETYLCHOLINE, there is evidence that the reinforcing or rewarding properties of nicotine may result from an activation of ascending limbic neurons, which release the neurotransmitter DOPAMINE (i.e., the mesocorticolimbic dopaminergic system). Interestingly, this same system has been implicated in the reinforcing properties of a variety of drugs, including stimulants and opiates. As stated above, tobacco smoking has been associated with a wide variety of serious health effects, including cancer and heart disease; however, the chances of developing them decrease once smoking is terminated. Although some of the smoking-induced damage is irreversible, the incidence rates for cancer and heart disease gradually become more similar between smokers and nonsmokers the longer the smoker refrains from smoking. However, withdrawal from tobacco smoking results in a withdrawal syndrome that varies in intensity from individual to individual and often leads to a relapse of smoking. This syndrome consists of cravings for tobacco, irritability, weight gain, difficulty concentrating, drowsiness, and sleep disturbances. The recent introduction of nicotine-containing chewing gum and transdermal patches have significantly helped to facilitate abstinence from smoking in a number of individuals by delivering nicotine through a relatively less toxic route of administration.

CENTRAL NERVOUS SYSTEM DEPRESSANTS

In general, the incidence and prevalence of the nonmedical use of central nervous system depressants (approximately 6 to 8% of young adults) exceeds that of the opioids. These drugs include the barbiturates, benzodiazepines, and related drugs. The shorter-acting barbiturates, such as pentobarbital (“yellow jackets”) or SECOBARBITAL (“red devils”), are usually preferred to the longer-acting drugs such as phenobarbital. Nonbarbiturates such as MEPROBAMATE, GLUTETHAMIDE, methyprylon, METHAQUALONE (Quaalude) and some of the shorter-acting benzodiazepines are also abused. Presumably, the quicker the onset of action for a particular central nervous system depressant, the better the “high.”

There is no general rule that can be used to predict the pattern of use of a central nervous system depressant for a given individual. Often there is a fine line between appropriate therapy for insomnia or ANXIETY and drug dependence. Some individuals exhibit cyclic patterns of use with gross intoxication for a few days interspersed with periods of abstinence. Other barbiturate or benzodiazepine users maintain a chronic low level of intoxication without observable signs of impairment because of the development of tolerance to many of the actions of these drugs. When higher doses are used, however, the intoxication may resemble alcohol intoxication, with slurred speech, difficulty thinking, memory impairment, sluggish behavior, and emotional instability. Withdrawal from chronic barbiturate or benzodiazepine use can also be manifested to varying degrees. In the mildest form, the individual may only experience mild anxiety or insomnia. With greater degrees of physical dependence, tremors and weakness may also be included. In severe withdrawal, delirium and tonic-clonic (epileptic) seizures may also be present. This severe withdrawal syndrome can be life-threatening. The degree of severity of the withdrawal syndrome appears to be related to the pharmacokinetics of the drug used. Shorter-acting benzodiazepines and barbiturates produce much more severe cases of withdrawal than do the longer-acting drugs. Therefore, in the case of severe withdrawal symptoms associated with the chronic use of a short-acting drug, a longer-acting drug should be substituted. The dose of this drug can be gradually decreased so that the individual experiences a much milder and less threatening withdrawal.

Receptor-binding sites for benzodiazepines and barbiturates are part of a macromolecular complex associated with chloride ion channels and the inhibitory neurotransmitter GABA. The interaction of these drugs with their distinct binding sites re-

sults in a facilitation of GABAergic neurotransmission, producing an inhibitory effect on neuronal impulse flow in the central nervous system.

CENTRAL NERVOUS SYSTEM STIMULANTS

Central nervous system stimulants include caffeine, cocaine, and amphetamine, although the use and abuse of amphetamines and cocaine represent a much greater health risk, with deviation from social norms.

Caffeine. Perhaps 80 percent of the world's population ingests caffeine in the form of TEA, COFFEE, COLA-flavored drinks, and CHOCOLATE. In the central nervous system, caffeine decreases drowsiness and fatigue and produces a more rapid and clearer flow of thought. With higher doses, however, nervousness, restlessness, insomnia, and tremors may result. Cardiac and gastrointestinal disturbances may also be observed. Tolerance typically develops to the anxiety and dysphoria experienced by some individuals. Some degree of PHYSICAL DEPENDENCE has, however, been associated with the chronic consumption of caffeine. The most characteristic symptom of caffeine withdrawal is a long throbbing headache, although fatigue, lethargy, and some degree of anxiety are also common. In general, the long-term consequences of chronic caffeine consumption are relatively minor.

COCAINE AND AMPHETAMINE

The problems associated with chronic cocaine and amphetamine use and withdrawal are much more serious than those associated with caffeine. By the mid-1980s, more than 20 million people had used cocaine in the United States. With the recent introduction of cocaine in the free alkaloid base ("FREEBASE" or "CRACK") form, there has been a significant increase in cocaine-related medical, economic, social, and legal problems. In the free-base form, cocaine can be smoked, resulting in blood levels and brain concentrations of the drug that compare to those observed when the drug is injected intravenously. In non-user subjects in a laboratory setting, the administration of cocaine or amphetamine produces an elevation of mood, an increase in energy and alertness, and a decrease in fatigue and boredom. In some individuals, however, anxiety, irritability, and insomnia may be observed.

In nonlaboratory settings, heavy users of cocaine often take the drug in bouts or binges, only stopping when their supply runs out or they collapse from exhaustion. Immediately following the intravenous administration or inhalation of cocaine, the individual experiences an intense pleasurable sensation known as a "rush" or "flash," followed by euphoria. Cocaine rapidly penetrates into the brain to produce these effects, but then is rapidly redistributed to other tissues. In many cases, the intense pleasure followed by the rapid decline in the cocaine-induced elevation of mood is sufficient for the individual to begin to immediately seek out and use more of the drug to prolong these pleasurable effects. Following the intranasal administration of cocaine, the pleasure is less intense and the decline in brain concentrations of the drug progresses much more slowly, so that the craving for more of the drug is less pronounced. Cocaine and amphetamine appear to produce their reinforcing or pleasurable effects through interactions with the neurotransmitter dopamine, especially in limbic and cortical regions of the brain (i.e., within the mesocorticolimbic dopaminergic system). Both cocaine and amphetamine block the reabsorption of dopamine into the NEURONS, where it was released, thereby prolonging the action of dopamine in the synapse—the space between nerve cells. Amphetamine can also cause the direct release of dopamine from nerve cells and can inhibit the metabolism of the neurotransmitter. It is important to note, however, that every drug that augments the action of dopamine does not produce pleasurable or rewarding subjective effects.

The toxicity associated with cocaine or amphetamine use can be quite severe; it is often unrelated to the duration of use or to preexisting medical conditions in the individual. This potential for serious toxic side effects is amplified by the fact that tolerance usually develops to the subjective feelings of the cocaine-induced rush and euphoria, but not to some of the other central nervous system effects of the drug (especially seizure susceptibility). Some of the more minor toxic reactions include dizziness, confusion, nausea, headache, sweating, and mild tremors. These symptoms are experienced by virtually all cocaine and amphetamine users to some degree, as a result of stimulation of the sympathetic nervous system. More serious reactions are also fre-

quently observed. These serious toxic effects can include irregular heartbeats, convulsions and seizures, heart attacks, liver failure, kidney failure, heart failure, respiratory depression, stroke, coma, and death. The effects on the heart and cardiovascular system can sometimes be treated with alpha and beta noradrenergic-receptor antagonists or calcium channel blockers, although even prompt medical attention is not always successful. The convulsions can sometimes be controlled with diazepam (Valium); ventilation (oxygen) may be required for the respiratory depression. In addition to the effects described above for cocaine, amphetamine has been reported to produce direct and irreversible neuronal damage to dopaminergic neurons. A similar effect for cocaine has not yet been identified.

Psychiatric abnormalities resulting from chronic central nervous system stimulant abuse can include anxiety, DEPRESSION, HALLUCINATIONS, and, in some cases, a paranoid psychosis that is virtually indistinguishable from a paranoid SCHIZOPHRENIC psychosis. A withdrawal syndrome is also observed following the abrupt cessation of chronic cocaine or amphetamine use. This syndrome begins with exhaustion during the "crash" phase and is followed by prolonged periods of anxiety, depression, anhedonia (loss of pleasure), hyperphagia (gluttony), and high craving for the drug. This craving may persist for several weeks, depending on the individual. The administration of dopaminergic agonists or tricyclic ANTIDEPRESSANTS may have some utility in decreasing the severity of the withdrawal symptoms.

CANNABINOIDS

Marijuana is probably still the most commonly used illicit drug in the United States, with about 55 percent of young adults reporting some experience with the drug during their lifetimes. The active ingredient in MARIJUANA is delta^9-TETRAHYDROCANNABINOL (Δ^9-THC), which exerts its most prominent effects on the central nervous system and the cardiovascular system. A marijuana cigarette that contains approximately 2 percent of the active ingredient can produce an increase in feelings of well-being, euphoria, and relaxation when smoked; however, short-term memory can be impaired as is the ability to carry out goal-directed behavior. The ability to drive or operate machinery is similarly impaired, often much longer than the persistence of subjective effects. With higher doses, paranoia, hallucinations, and anxiety or panic may be manifested.

Chronic marijuana users sometimes exhibit what is called the AMOTIVATIONAL SYNDROME—which consists of apathy, impairment of judgment, and a loss of interest in personal appearance and the pursuit of conventional goals. However, it is not clear whether this syndrome results from the use of marijuana alone or from other factors. Although this is seldom severe, Δ^9-THC also produces a dose-related increase in heart rate. Tolerance does develop to the effects of marijuana, and in some countries, regular users of HASHISH (a concentrated resin containing increased amounts of Δ^9-THC) consume quantities of the drug that would be toxic to most marijuana users in the United States. The withdrawal associated with the cessation of marijuana smoking is relatively mild—consisting of irritability, restlessness, nervousness, insomnia, weight loss, chills, and increased body temperature.

OPIOIDS

The use of opioids in the United States is much less prevalent than reported for the other drugs discussed above. For example, as of the 1990s, less than 0.5 percent of young adults have reported trying heroin at some time during their lives. There are three basic patterns of opioid use and dependence in the United States. The first group constitutes the smallest percentage of opioid users—those who initially began using morphinelike drugs medically, for the relief of PAIN. The second group began using illegal drugs through experimentation and then progressed to chronic use and dependence. The third group represents physically addicted individuals who eventually switched to oral METHADONE, obtained through organized treatment centers. Interestingly, the incidence of opioid addiction is greater among physicians, nurses, and related health-care professionals (who have access to these drugs) than in any group with a comparable educational background. In many instances (but not all), those addicted either to heroin (usually purchased illegally on the street) or to methadone (usually from treatment centers) are able to hold jobs and raise a family. Opioids reduce pain, aggression, and sexual drives, so the use of these drugs is unlikely to induce crime. Those who can-

not afford opioids, those who like the "drug life," and those who are unable or unwilling to hold a job, resort to crime to support their drug habits.

Opioid drugs produce their pharmacological effects by binding to opiate RECEPTORS. The euphoria associated with the use of opioids results from interactions of these drugs with the mu-opiate receptor, possibly resulting in the stimulation of mesocorticolimbic dopaminergic neuronal activity. The rapid intravenous injection of morphine (or heroin, which is converted to morphine once it enters the brain) results in a warm flushing of the skin and sensations in the lower abdomen that are often described as being similar in intensity and quality to sexual orgasm. This initial rush ("kick" or "thrill") lasts for about 45 seconds and is followed by a high—described as a state of dreamy indifference. Depending on the individual and the social circumstances, good health and productive work are not incompatible with the regular use of opioids. Tolerance can develop to the ANALGESIC, respiratory depressant, sedative, and reinforcing properties of opioids, but the degree and extent of tolerance depends largely on the pattern of use. Desired analgesia can often be maintained through the intermittent use of morphine. Tolerance develops more rapidly with more continuous opioid administration.

The abrupt discontinuation of opioid use can lead to a withdrawal syndrome that varies in degree and severity depending on the individual as well as the particular opioid used. Watery eyes (lacrimation), a runny nose (rhinorrhea), yawning and sweating occur within twelve hours from the last dose of the opioid. As the syndrome progresses, dilated pupils, anorexia, gooseflesh ("cold turkey"), restlessness, irritability, and tremor can develop. As the syndrome intensifies, weakness and depression are pronounced, and nausea, vomiting, diarrhea, and intestinal spasms are common. Muscle cramps and spasms, including involuntary kicking movements ("kicking the habit"), are also characteristic of opioid withdrawal; however, seizures do not occur and the withdrawal syndrome is rarely life-threatening. Without treatment, the morphine-induced withdrawal syndrome usually runs its course within seven to ten days. Opiate-receptor antagonists (e.g., NALOXONE) are contraindicated in opioid withdrawal, since these drugs can precipitate a more severe withdrawal on their own. Rather, longer-acting, less potent, opiate-receptor agonists such as methadone are more commonly prescribed. The symptoms associated with methadone withdrawal are milder, although more protracted, than those observed with morphine or heroin. Therefore, methadone therapy can be gradually discontinued in some heroin-dependent individuals. If the patient is unwilling or unable to withdraw from methadone, the individual can be maintained on methadone indefinitely.

PSYCHEDELICS

Psychedelics include drugs related to the indolealkylamines, such as lysergic acid diethylamide (LSD), PSILOCYBIN, psilocin, DIMETHYLTRYPTAMINE (DMT) and diethyltryptamine (DET), to the phenylethylamines, such as mescaline, or to the phenylisopropylamines, such as 2,5-dimethoxy-4-methylamphetamine (DOM or "STP") as well as 3,4-methylene-dioxyamphetamine (MDA) and 3,4-methylene-dioxymethamphetamine (MDMA or "ecstasy"). The feature that distinguishes these psychedelic agents from other classes of drugs is their capacity to reliably induce states of altered perception, thought, and feeling. There is a heightened awareness of sensory input accompanied by an enhanced sense of clarity, but a diminished control over what is experienced. The effects of LSD and related psychedelic drugs appear to be mediated through a subclass of receptors associated with the inhibitory neurotransmitter serotonin (i.e., serotonin $5HT_2$ receptors). Immediately after the administration of LSD, somatic symptoms such as dizziness, weakness and nausea are present, although euphoric effects usually predominate. Within two to three hours, visual perceptions become distorted; colors are heard and sounds may be seen. Vivid visual hallucinations are also often present. Many times this loss of control is disconcerting to the individual, resulting in the need for structure—in the form of experienced companions during the "trip." The entire syndrome begins to clear after about twelve hours.

Little evidence exists for long-term changes in personality, beliefs, values, or behavior produced by the drug. Tolerance rapidly develops to the behavioral effects of LSD after three or four daily doses of the drug. In general, however, the psychedelic drugs do not give rise to patterns of continued use over extended periods. The use of these drugs is generally restricted to the occasional trip. With-

drawal phenomena are not observed after the abrupt discontinuation of LSD-like drugs, and deaths directly related to the pharmacological effects of LSD are unreported in humans—however, fatal ACCIDENTS and SUICIDES have occurred during periods of LSD intoxication.

INHALANTS

The intoxicating and euphorigenic properties of nitrous oxide and ethyl ether were well known even before their potential as anesthetics was recognized. Physicians, nurses and other health-care professionals have been known to inhale anesthetic gases even though they have access to a wide variety of other drugs. Adolescents with restricted access to alcohol often resort to "glue sniffing" or the inhalation of vapors from substances with marked toxicity, such as gasoline, paint thinners, or other industrial solvents. The alkyl nitrites (butyl, isobutyl, and amyl) have been used as aphrodisiacs, since the inhalation of these agents is suggested to intensify and prolong orgasm. At least 12 percent of young adults have reported some experience with inhalants—however, fatal toxic reactions (usually due to cardiac arrhythmias) are often associated with the inhalation of many of these drugs. Inhalation from a plastic bag can result in hypoxia (too little oxygen) as well as an extremely high concentration of vapor. Fluorinated hydrocarbons can produce cardiac arrhythmias and ischemia (localized anemia). Chlorinated solvents depress heart muscle (myocardial) contractility. Ketones can produce pulmonary (lung) hypertension. Neurological impairment can also occur with a variety of solvents.

ARYLCYCLOHEXYLAMINES

Arylcyclohexylamines include phencyclidine (PCP or "angel dust") and related drugs that possess central nervous system stimulant, central nervous system depressant, hallucinogenic, and analgesic properties. These drugs (also known as dissociative anesthetics) are well absorbed following all routes of administration. With even small doses, intoxication is produced, with associated staggering gait, slurred speech, and numbness in the extremities. PCP users may also exhibit sweating, catatonia, and a blank stare as well as hostile and bizarre behavior. Amnesia during the intoxication may also occur. In higher doses, anesthesia, stupor, convulsions, and coma may appear. The typical high from a single dose can last four to six hours and is followed by a prolonged period of "coming down."

PCP and related compounds bind with high affinity to a number of distinct sites in the central nervous system, although it is not certain which site(s) is responsible for the primary pharmacological effects of these drugs. PCP binds to the sigma site, which also has a high affinity for some selected opioids, although the function of the sigma site is unknown. PCP blocks the cation channel (e.g., Ca^{2+}) that is regulated by N-methyl-D-aspartate (NMDA), one type of receptor for excitatory amino acid neurotransmitters such as glutamate or aspartate. PCP also blocks the reabsorption of the neurotransmitter dopamine into the neurons, where it was released, resulting in a prolonged action of the neurotransmitter, especially within the mesocorticolimbic dopaminergic neuronal system.

There appears to be some degree of tolerance to the effects of PCP, and some chronic users of PCP complain of cravings and difficulties with recent memory, thinking, and speech after discontinuing the use of the drug. Personality changes can range from social withdrawal and isolation to severe anxiety, nervousness, and depression. Although the frequency is uncertain, deaths due to direct toxicity, violent behavior, and accidents have been reported following the use of PCP. PCP can also produce acute behavioral toxicity—consisting of intoxication, aggression, and confusion, as well as coma, convulsions, and psychoses. A PCP-induced psychosis can persist for several weeks following a single dose of the drug.

(SEE ALSO: *Addiction: Concepts and Definitions; Complications; Epidemiology of Drug Abuse; National Household Survey on Drug Abuse; Treatment*)

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NICK E. GOEDERS

DRUNK DRIVING Drunk driving results in one of the most costly social consequences of ALCOHOL abuse. The toll on human life and health exacted by drunk drivers can, on its own, make alcohol abuse one of the most serious U.S. social problems. The extent and consequences of drunk driving demonstrate the challenges of harmonizing a drinking culture with a modern industrial society.

The combination of drinking and driving has been recognized as a serious problem since the invention of the automobile in the 1880s. In 1904, the *Quarterly Journal on Inebriety* editorialized that "the precaution of railroad companies to have only total abstainers guide their engines will soon extend to the owners of these new motor wagons . . . with the increased popularity of these wagons, accidents of this kind will multiply rapidly." By 1910, drunk driving had already been codified as a misdemeanor offense. Moreover, the dangerous mixture of alcohol and driving was a key point in the Prohibitionists' argument in favor of the Eighteenth Amendment.

During the 1950s and 1960s, with postwar prosperity and a developing highway network, both alcohol abuse and traffic safety became serious national widespread issues. The Highway Safety Act of 1966 was crucial to mobilizing attention and resources in an attack against drunk driving. In effect, it established a federal (not just a state and local) jurisdiction by creating the National Highway Safety Bureau, the precursor of the National Highway Safety Administration (NHTSA), and it authorized the U.S. Department of Education's 1968 Report, *Alcohol and Highway Safety*. This report found that "the use of alcohol by drivers and pedestrians leads to some 25,000 deaths and a total of at least 800,000 crashes in the United States each year." The report warned that "this major source of human morbidity will continue to plague our mechanically powered society until its ramifications and many present questions have been exhaustively explored and the precise possibilities for truly effective countermeasures determined."

NHTSA became the main sponsor of research and action projects aimed at reducing drunk driving. In 1970, NHTSA launched the Alcohol Safety Action Project (ASAP), the first major U.S. initiative against drunk driving. The ASAP, established in thirty-five communities, sought to achieve a significant reduction in drunk driving through a mixture of intensive countermeasures—including law enforcement, rehabilitation, and education. These programs were rigorously monitored. Unfortunately, despite huge increases in arrests and tens of thousands of referrals to drunk-driver schools and rehabilitation programs, a significant decrease in drunk driving could not be confirmed, and the ASAP was terminated in 1977.

The attack on drunk driving did not subside. In the late 1970s, there emerged a remarkable grassroots anti-drunk driving movement comprised of

victims, their families, and many other concerned citizens. MOTHERS AGAINST DRUNK DRIVING (MADD), Students Against Driving Drunk (SADD), and REMOVE INTOXICATED DRIVERS (RID) opened local chapters throughout the United States and vigorously campaigned for new and tougher drunk-driving countermeasures. The crusade launched by these groups attracted a great deal of media attention, vaulting drunk driving to the top of the nation's social problems agenda. In 1982, President Ronald W. Reagan appointed a Presidential Commission on Drunk Driving. Congress linked state highway funds to the states' passage of specified anti-drunk driving measures, including a minimum drinking age of twenty-one. Ultimately, every state raised its drinking age accordingly. The states passed a deluge of legislation, providing for more and better law enforcement and more severe criminal penalties of a greater range—including mandatory jail terms, automatic license forfeiture, public education, drunk-driver schools, and rehabilitation.

MAGNITUDE OF THE PROBLEM

More than 2 million people are arrested each year for drunk driving. The actual number of offenses, while unknown and unknowable, must be far greater, since only a fraction of all violators are apprehended. A few researchers have mounted roadside surveys in which drivers are stopped and asked to voluntarily provide a breath sample from which the amount of alcohol in the blood can be calculated. While this is the best strategy for determining the actual amount of drunk driving, there are many problems with this methodology (which roads? what times? how many refusals?). A 1985 Minnesota roadside survey found that of 838 drivers on the road between 8:00 P.M. and 3:00 A.M. (prime time for drunk driving), 82.3 percent tested negative for any alcohol, 6 percent tested at BLOOD ALCOHOL CONCENTRATION (BAC) 0.05–0.09 percent (included as a lesser Driving While Intoxicated [DWI] offense in some states), and 2.4 percent tested above the drunk-driving threshold of BAC 0.1 percent.

Drunk drivers do not pose a uniform risk to themselves, their passengers, other motorists, and pedestrians. The most dangerous of the drunk drivers are the vehicular equivalent of the "fighting drunk"; they drive far in excess of the speed limit, weave in and out of traffic, and cross into lanes of traffic going in the opposite direction. At the low end of the continuum are drunk drivers who make an impaired effort to drive safely; although operating with diminished skill and judgment, they pose less of a risk than the agressive drunk drivers.

The most impressive experimental study of the causal role of alcohol in traffic crashes was carried out during the 1960s by Professor Robert Borkenstein (inventor of the BREATHALYZER) and his University of Indiana colleagues. The researchers obtained breath samples from 6,000 accident-involved drivers and, as controls, from 7,500 non-accident-involved drivers. They found that 6.3 percent of the accident-involved drivers, but fewer than 1 percent of the control drivers, had BACs equal to or greater than 0.1 percent (the prevailing definition of drunk driving in the 1980s). Moreover, each higher BAC level included a disproportionate number from the accident-involved group. Thus Borkenstein and his colleagues concluded that "BACs above .04% are definitely associated with an increased accident rate. The probability of accident-involvement increases rapidly at BACs above .15%. When drivers with BACs over 0.08% have accidents, they disproportionately involve only the driver's vehicle, and are more costly in terms of personal injury and property damage."

While most drunk-driving episodes do not result in a crash or injuries, the aggregate personal and property damage perpetrated by drunk drivers is staggering. A good deal of methodological controversy exists about the percentage of the approximately 45,000 annual traffic fatalities in the United States that can be attributed to drunk drivers. NHTSA's Fatal Accident Reporting System, which has been operating since the mid-1970s, presents important information about alcohol and traffic fatalities but does not attempt to estimate what proportion of all traffic deaths were *caused by* drunken driving. James Fell and Terry Klein, using statistical modeling techniques, have estimated that approximately 30 percent of all traffic fatalities can be attributed to drunk driving; other analyses have put this estimate at 50 percent.

Drunk drivers themselves, often in single-car collisions, comprise a large proportion of those who are killed, giving fatal drunk-driving episodes as much resemblance to suicide as to homicide. Nevertheless, each year thousands of innocent pedestrians and motorists are killed by drunk drivers,

and tens of thousands are badly injured. There is also a huge amount of property damage.

THE OFFENDER

It is difficult to find reliable data on which to base a profile of the drunk driver. Using arrest data, we find that the vast majority, 90 percent, of drunk drivers are male and white. Despite the common belief that teenage drivers are most likely to be drunk, it is the mid-twenties age group that deserves this notoriety. Since it takes heavy drinking (from four to six drinks in two hours, depending on the drinker's weight) to reach the prohibited level, it is unlikely that light drinkers very often commit drunk-driving offenses. Thus, people who drive drunk are likely to be heavy drinkers and alcohol abusers. Nevertheless, light and moderate drinkers may on occasion drive drunk, perhaps due to a binge. Since light and moderate drinkers greatly outnumber heavy and abusive drinkers, they may in fact comprise a substantial proportion of arrested drunk drivers.

The consensus of studies based on screening tests of drunk drivers is that about 50 percent arrested for this offense are alcohol abusers, about 35 percent are social drinkers, and the remainder fall in between. While the categories *alcohol abuser* and *social drinker* are amorphous, a disproportionately high percentage of those arrested for drunk driving are actually heavy drinkers.

The large majority of drunk drivers arrested in any given year have not been arrested before. A well-executed Minnesota study found that only 7 percent of drivers involved in fatal accidents had been convicted of drunk driving in the preceding three years; of drunk drivers involved in fatal accidents, 13 percent had a DWI conviction in the previous 3 years, and 25 percent had a license revocation during the preceding 8 years. The low official rate of recidivism probably means that the chance of a drunk driver's being caught is extremely small.

THE CRIME OF DRUNK DRIVING

The first drunk-driving laws made it an offense to drive while intoxicated or to drive under the influence (DUI) of alcohol. Starting in the 1950s, states began to pass *per se* laws, which made it an offense to operate a motor vehicle with a BAC that exceeds certain levels. When a suspect is arrested for drunk driving, he or she is asked to take a deep breath and blow into a machine (the Breathalyzer is one model) that measures the amount of alcohol in the breath and converts it into a measure of the amount of alcohol in the blood. Pursuant to *implied consent* laws, suspects who refuse to provide a deep-lung breath sample are penalized by loss of their driver's license and sometimes by other sanctions as well. The evolution of breath-testing equipment, including hand-held devices (like the Intoxilyzer), has greatly eased the identification and conviction of drunk drivers. Despite folklore to the contrary, it is extremely rare that suspects who "fail" the breath test obtain acquittals. Indeed, the conviction rate for drunk driving is well over 90 percent.

In most states, a first drunk-driving offense is a misdemeanor, and a second offense within a specified time period (up to ten years in some states) is a felony. In a few states, a first offense is treated as a traffic violation, a second offense a misdemeanor, and a third offense a felony. Punishments vary from state to state; however, the usual range of punishments includes forfeiture of a driver's license for up to 1 year, fines of 500 to 1,000 dollars, and incarceration up to 30 days. In the late 1980s, spurred by the anti-drunk driving citizens' groups and federal financial incentives, several states passed laws mandating at least forty-eight hours of incarceration for a first DWI offense and a longer time for a second or subsequent offense. Another penalty that has been gaining popularity is the automatic and immediate forfeiture of the driver's license at the police station when the suspect fails or refuses to take the breath test (*administrative per se* law). In the early 1990s, and once again in response to federal pressure, states began lowering the prohibited BAC from 0.1 percent to 0.08 percent.

At least since the early 1970s, the criminal justice system's processing of drunk drivers has been linked to alcohol-treatment programs. In many jurisdictions, all drunk-driving offenders are routinely screened for ALCOHOLISM and alcohol abuse. Alcoholics and abusers may be diverted from prosecution to TREATMENT. More likely, however, the judge will require the offender to participate in treatment as a condition of probation or in order to obtain a provisional or regular driver's license. In some jurisdictions, the criminal-justice system is the largest source of clients flowing into alcohol-

treatment programs. Thus, enforcement of the drunk-driving laws is one of the major ways that alcohol abusers are brought into the alcohol-treatment matrix.

In addition to the standard alcohol treatments, the attack on drunk driving has produced one unique kind of treatment—the drinking-driver school, which several million people have passed through since the mid-1970s. States and localities that have such schools often require all drunk drivers to attend. New York's school consists of five two-hour sessions and two three-hour sessions. The classes provide information about such matters as the deterioration of driving skills at different BAC levels, the inability to counteract intoxication with coffee or cold showers, and criminal penalties for drunk driving. People taking these classes are also required to fill out the Michigan Alcohol Screening Test (MAST) to determine whether they are alcohol abusers.

ENFORCEMENT

Enforcement of drunk-driving laws is the responsibility of local police, county sheriffs, and the state police or highway patrol. The Fourth Amendment to the U.S. Constitution prevents police from stopping cars at random and requiring drivers to take breath tests. Police must have probable cause to believe that drunk driving or some other offense (including traffic offenses) has been, is, or is about to be committed. Once a driver has been legitimately stopped, the police officer can order the driver to submit to a *field sobriety test*, which may consist of walking heel-to-toe, counting backwards, or performing other tasks that reveal intoxication. If the driver's performance on the test gives the officer probable cause to believe that the driver is intoxicated, the officer will arrest the driver. At the station, drivers will be told that they are required by the implied-consent law to submit to a breath test; refusal to cooperate will lead to license revocation.

In the 1980s, as states and localities searched for more effective anti-drunk driving strategies, some police departments mounted roadblocks at which every car (or every *n*th car) was briefly stopped and the driver briefly observed and sometimes questioned. If the officer detected alcohol, the driver was pulled over and required to submit to a breath test. Since these drunk-driving roadblocks were not based upon probable cause, they were challenged. In 1990, however, the U.S. Supreme Court upheld drunk-driving roadblocks under its *administrative search* doctrine (*Michigan Dept. of State Police v. Sitz*, 110 S. Ct. 2481). The Court ruled that as long as the roadblocks are situated in a fixed location, overseen by high-level officials, and operated nondiscriminatorily, they do not violate the Fourth Amendment.

DETERRING THE DRINKING DRIVER

Deterrence based on the threat of arrest, conviction, and punishment remains the chief strategy in the attack on drunk driving. During the 1980s, state and local governments have established dozens of strike forces and passed hundreds of laws aiming to raise the costs to the offender of driving while intoxicated. In a series of empirical evaluations of police crackdowns and elevated maximum punishments in the United States and abroad, the sociologist H. Laurence Ross found that this type of law-enforcement escalation usually produces a reduction in drunk driving (as measured by single-vehicle fatalities), but not a long-term reduction. "No such policies have been scientifically demonstrated to work over time under conditions achieved in any jurisdiction . . . the option of merely increasing penalties for drinking and driving has been strongly discredited by experience to date."

While Ross has done far more empirical research than anybody else on deterring the drunk driver, his conclusion is not uncontroversial. One criticism is that he uses single-vehicle automobile fatalities to measure the amount of drunk driving; however, this kind of accident might not be strongly associated with the full range of drunk driving, but only with a narrow group, the most drunken and reckless of drunk driving. Possibly, while law-enforcement escalations cannot affect the kind of drunk drivers who kill themselves in single-vehicle crashes, they might be effective in the far more numerous non-fatal drunk-driving episodes that are engaged in by less pathological alcohol abusers and sociopathological persons.

The number of traffic fatalities has fallen from the late 1980s into the 1990s; drunk-driving fatalities seem to have fallen more than non-alcohol-related accidents. There may be reasons for this other than deterrence, including general re-

ductions in alcohol consumption and abuse and more responsible public attitudes toward sober driving—however, a marginal deterrence effect cannot be ruled out.

OTHER ANTIDRUNK-DRIVING STRATEGIES

In addition to deterrence, states and localities have implemented many other anti-drunk driving strategies. Since all these strategies are being used simultaneously, it is impossible to attribute any reductions to one strategy over another.

Some courts have made punitive damages available in drunk-driving cases. This allows the victim of a drunk driver to recover any amount of money a jury deems appropriate for punishment. Some states permit insurance coverage of punitive damages, thereby negating whatever deterrent effect such damages might produce, but not negating a windfall for the victim.

In some states, legislatures and courts have expanded civil (tort) liability for causing drunk-driving injuries to include commercial hosts and package sellers of alcohol. While these DRAMSHOP LAWS vary from state to state, they essentially make purveyors of alcohol to underage or intoxicated persons liable for the injuries caused by such persons to themselves or others. A few state courts have even made social hosts liable for the alcohol-related traffic injuries caused by their guests.

An essential strategy for incapacitating drunk drivers is taking away their licenses to drive. Several studies have shown that drunk drivers who lose their drivers' licenses are less likely to have a recurrence than drunk drivers who are fined, sent to jail, or assigned to mandatory treatment programs (actually, all these sanctions can be imposed together). Nevertheless, when licenses are suspended or revoked, a good deal of licenseless driving takes place—which is not surprising in a society where people depend on automobiles for their economic and social lives. Several states also have laws that authorize vehicle impoundment or forfeiture, but these sanctions are rarely used, perhaps because of the sacred status of the automobile as expensive private property.

Opportunity blocking refers to anti-crime strategies that change the environment to reduce the opportunities of committing particular offenses. The best opportunity-blocking strategy for drunk driving involves fixing the defendant's vehicle so that it cannot be started until he blows alcohol-free breath into a tube affixed to the vehicle. Such equipment is now available, and several jurisdictions have implemented experimental programs. Other opportunity-blocking strategies include the twenty-one-year-old drinking age and a spate of new laws and regulations on bars, taverns, and package-goods stores.

The anti-drunk driving movement has spawned a large number of educational strategies. These include public-service announcements on radio and television and educational materials for primary and secondary schools. The effects of such programs are very difficult to evaluate. Rehabilitation strategies for drunk drivers are closely linked to the matrix of community alcoholism and alcohol-abuse services. Drunk drivers are regularly screened for alcohol problems, and those who are identified as abusers are typically channeled into treatment through a probationary sentence.

(SEE ALSO: *Accidents and Injuries; Addiction: Concepts and Definitions; Distilled Spirits Council; Driving, Alcohol, and Drugs; Minimum Drinking Age Laws; Prevention Movement; Psychomotor Effects of Alcohol and Drugs; Social Costs of Alcohol and Drug Abuse*)

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JAMES B. JACOBS

DSM *See* Diagnostic and Statistical Manual

DTS *See* Delerium Tremens

DUAL DIAGNOSIS *See* Comorbidity and Vulnerability; Complications: Mental Disorders

DUI/DWI *See* Driving, Alcohol, and Drugs; Driving Under the Influence; Drunk Driving

DYNORPHIN Dynorphin is a neuropeptide transmitter; it is an OPIOID peptide, a member of the endorphin family of peptides. All neurotransmitters like Dynorphin have receptors. Its greatest affinity is for the Kappa opioid receptor. Dynorphin's role in drug abuse was originally anticipated based on its location in anatomical areas strongly associated with the mechanism of action of drugs of abuse. It is localized in the NUCLEUS ACCUMBENS, AMYGDALA, and VENTRAL TEGMENTAL AREA.

Dynorphin induces feelings of dysphoria, or despair. This was first documented in animals, and later confirmed in humans. It is surprising because the best known opiate-like drugs are morphine and heroin, and they present great abuse liability since they illicit feelings of euphoria and absence of pain. However, there seem to be two opioid systems controlling behavior one influencing feelings of REWARD (through-endorphins) and one influencing feelings of AVERSION, (through dynorphin). The physiological substrate underlying the effects of dynorphins is believed to be at the level of the MESOLIMBIC DOPAMINE neurons in the ventral tegmental area. Dynorphin tonically inhibits the firing of dopamine neurons, thus preventing its release in the striatum. Elevations of dopamine levels in the nucleus accumbens are believed to underlie the reinforcing properties of many PSYCHOSTIMULANT-like drugs, as well as OPIATES.

Due to the ADVERSE feeling associated with WITHDRAWAL from many drugs of abuse, the dynorphin system has been implicated in contributing to this state. Studies have found that there are long-term changes in dynorphin levels in brain areas associated with drug abuse, and that these changes also exist during withdrawal. Prenatal exposure to cocaine also effects the levels of dynorphin in the brain. These changes are present in both animal and human models of drug abuse. Since drugs modulate dynorphin systems, we can gain an understanding of how drugs work in the brain by studying the dynorphin system.

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VOLUME 2

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ECONOMIC COSTS OF ALCOHOL ABUSE AND ALCOHOL DEPENDENCE

Alcohol abuse and alcohol dependence continue to be major health problems in the United States. The terms *alcohol abuse* and *alcohol dependence* are based on the diagnostic criteria as stated in the American Psychiatric Association's DIAGNOSTIC AND STATISTICAL MANUAL *of Mental Disorders, Third Edition, Revised* (1987). As such, they cost the nation billions of dollars in health-care costs and reduced or lost productivity each year. Since the mid-1980s, researchers have issued studies that estimate the economic costs associated with alcohol and alcohol abuse in the United States. In 1985, alcohol abuse and dependence cost an estimated 70.3 billion dollars and in 1988 an estimated 85.8 billion dollars (Rice et al., 1990, 1991). In 1998, the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse (NIAAA), which are parts of the National Institutes of Health (NIH), released a study on these costs based on 1992 survey data. This report, which also analyzed drug abuse, forms the basis of this article.

EXTENT OF THE PROBLEM

The economic cost to society from alcohol and drug abuse was $246 billion in 1992. Alcohol abuse and alcoholism cost an estimated $148 billion, while drug abuse and dependence cost an estimated $98 billion. When adjusted for inflation and population growth, the alcohol estimates for 1992 were very similar to cost estimates produced over the past 20 years. The 1992 estimates were significantly greater than the 1985 estimate for alcohol: 42 percent higher for alcohol over and above increases due to population growth and inflation. Between 1985 and 1992, inflation accounted for about 37.5 percent and population growth for 7.1 percent increases. Over 80 percent of the increase in estimated costs of alcohol abuse was attributed to changes in data and methodology employed in the new study. This suggests that the previous study significantly underestimated the costs of alcohol abuse.

In 1992, there were an estimated 107,400 alcohol-related deaths in the United States. Many of the alcohol-related deaths were among persons between ages twenty and forty, because the major causes of death, such as motor vehicle crashes and other causes of traumatic death are concentrated among younger-aged people. However, alcohol is also involved in numerous premature deaths among the older population because of long-term, excessive alcohol consumption. Total costs attributed to alcohol-related motor vehicle crashes were estimated to be $24.7 billion. This included $11.1 billion from premature mortality and $13.6 billion from automobile and other property destruction.

In 1992, total estimated spending for health care services was $18.8 billion for alcohol problems and the medical consequences of alcohol consumption. Specialized services for the treatment of alcohol problems cost $5.6 billion. This included special-

ized detoxification and rehabilitation services as well as prevention, training, and research expenditures. Costs of treatment for health problems attributed to alcohol were estimated at $13.2 billion.

An estimated $67.7 billion in lost potential productivity was attributed to alcohol abuse in 1992. This accrued in the form of work not performed, including household tasks, and was measured in terms of lost earnings and household productivity. These costs were primarily borne by the alcohol abusers and by those with whom they lived. About $1 billion was for victims of fetal alcohol syndrome who had survived to adulthood and experienced mental impairment. This study did not estimate the burden of drug and alcohol problems on work sites or employers.

The costs of crime attributed to alcohol abuse were estimated at $19.7 billion. These costs include reduced earnings due to incarceration, crime careers, and criminal victimization; and the costs of criminal justice and drug interdiction. Alcohol abuse is estimated to have contributed to 25 to 30 percent of violent crime.

The study estimated that 3.3 percent of social welfare beneficiaries in 1992 received benefits because of an administrative determination of drug- or alcohol-related impairment. While 1996 federal welfare reform legislation has largely terminated alcohol or drug dependence as a primary cause for benefit eligibility, these impairments resulted in transfers of $10.4 billion in 1992, with administrative and other direct service expenses of $683 million for those with alcohol problems.

A large amount of the economic burden of problems falls on the population that does not abuse alcohol. Governments bore costs of $57.2 billion (38.6 percent) in 1992, compared with $15.1 billion for private insurance, $9 billion for victims, and $66.8 billion for alcohol abusers and members of their households. Costs are imposed on society in a variety of ways, including alcohol-related crimes and trauma (e.g., motor vehicle crashes), government services, such as criminal justice and highway safety, and various social insurance mechanisms, such as private and public health insurance, life insurance, tax payments, pensions, and social welfare insurance.

CONCLUSION

Alcohol abuse and alcohol dependence are costly to the United States in resources used for care and treatment of persons suffering from these disorders, lives lost prematurely, and reduced productivity. Data show clearly that the measurable economic costs of alcohol abuse continue to be high.

(SEE ALSO: *Accidents and Injuries from Alcohol; Alcohol and AIDS; Cancer, Drugs, and Alcohol; Complications; Crime and Drugs; Drug Interactions and Alcohol; Social Costs of Alcohol and Drug Abuse*)

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DOROTHY P. RICE
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ECONOMICS OF ALCOHOL AND DRUG ABUSE *See* Productivity; Social Costs of Alcohol and Drug Abuse

ECSTASY *See* MDMA

ED50 The ED50 is the median effective dose—the dose of a drug that is required to produce a specific effect (e.g., relief from headache) in 50 percent of a given population. The ED50 can be estimated from a dose-effect curve, where the dose of the drug is plotted against the percentage of a population in which the drug produces the specified effect. Therefore, if the ED50s for two drugs in producing a specified amount of relief from headache are 5 and 500 milligrams, respectively, then the first drug can be said to be 100 times more potent than the second for the treatment of headaches.

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NICK E. GOEDERS

EDUCATION AND PREVENTION American adolescents increased their use of most illicit substances throughout the 1990s after a significant drop in the previous decade, and in 1999 Drug Czar Barry McCaffrey responded to the recent Monitoring the Future study by saying drug use "remains unacceptably high" (University of Michigan Institute for Social Research, 1999). Data on special populations such as infants, the homeless, the ELDERLY, and those with HIV/AIDS indicate increasing needs for prevention and education throughout the life span. COCAINE and HEROIN patients in emergency rooms have also increased since 1990 and the American Lung Association estimates that 430,700 Americans die each year from diseases directly related to smoking. Clearly, the use of ALCOHOL, TOBACCO, and other drugs—whether licit or illicit—by various age groups and special populations continues to be a problem in the United States.

The concept of *prevention* has evolved since the 1960s to become much broader, one that has shifted from a focus primarily on adolescents to a life-span perspective that includes all ages from the fetus through the elderly. Prevention services recognize all potentially addictive substances—including alcohol, tobacco, MARIJUANA, cocaine, OPIOIDS, INHALANTS, HALLUCINOGENS, and prescription and nonprescription (OVER-THE-COUNTER, OTC) medications. Linkages have been developed with several services to include PREVENTION, intervention, and TREATMENT. Prevention programs now emphasize comprehensive long-term systematic programming for individuals, peer groups, FAMILIES, and/or communities. Such programs utilize prevention concepts based on the positive results of controlled experiments and quasi-experimental studies. They contain a core of pro-social skills central to the prevention of substance abuse as well as other social problems—SUICIDE, unwanted pregnancies, and VIOLENCE.

CONTEMPORARY PRINCIPLES OF PREVENTION

Several authorities have analyzed prevention programs for substance abuse and have listed principles of effective prevention programs (Dryfoos, 1990; Falco, 1992; Hawkins et al., 1992; The U.S. General Accounting Office, 1992; and The Higher Education Center for Alcohol and Other Drug Prevention, 1999). The principles in this section emerged from this literature as well as other sources. This type of "lumping," of necessity, ignores many subtle points applicable to specific programs or to particular issues. Nonetheless, widespread agreement exists that these principles provide a foundation for planning effective, cost-effective, prevention programming.

1. Effective prevention programs provide for comprehensive, coordinated services to individuals and their families along a continuum of care.

Comprehensive prevention programming in a community includes services for all age groups, with multiple forms of programming for any age group. Comprehensive services are arrayed along a continuum to include education, prevention, intervention, and referral to treatment when necessary. Further, most people in high-risk substance-abuse environments need a variety of other services—health, nutrition, prenatal care—along with sub-

stance-abuse prevention services. All of these services need to be coordinated for maximum effect and efficiency. In any community, pregnant women, children, adolescents, workers and/or elderly, some are in need of intervention rather than prevention; a comprehensive strategy provides for intensive services as required.

Effective prevention programs also involve the families of the target populations, either as the focus of the service or as a tangent to a service array. Such programs include training in relationships and parenting skills, while reinforcing family awareness of the purposes and procedures of substance-abuse prevention programs. Bry, Conboy, and Bisgay (1986) reported reduced substance use and fewer problems in programs for youth that taught their parents needed parenting skills.

Student-assistance programs and EMPLOYEE-ASSISTANCE PROGRAMS (EAPs) have emerged to fill an important gap in the care continuum. Such programs identify those whose performance (academic or work) deteriorates, to assist them in obtaining the most appropriate help. They are considered by businesses to be beneficial (U.S. Department of Labor, 1991), and schools perceive them as essential to their total programming (Swisher et al., 1993).

2. Effective prevention programs are developmentally appropriate, culturally relevant, and sensitive to ethnic minority members, females, and persons in special circumstances (e.g. homeless persons).

They must also be developmentally appropriate and adjusted to the emotional and mental development of the individual or group. Too often prevention programs have attempted to provide a diluted version of a program to a younger age group without considering the developmental stage. Programs must be adapted to an individual's needs in the various transitions of our lives. Some programs, for the oldest members of a community, must be designed for their particular needs and frequent involvement with chronic illness (Garrity & Lawson, 1989).

Prevention programs are most effective when they are culturally relevant to the norms and assumptions of the various ethnic and minority groups. Role models and media materials must be culturally sensitive or they will be rejected by the audience either consciously or subconsciously. Several authorities have compiled examples of successful experiences that a variety of programs have had with participants from diverse racial and ethnic orientations (e.g., Resnick & Wojcicki, 1991; Marcus & Swisher, 1992). A recent novella aimed at Hispanic youths and their families received accolades for cultural sensitivity and scope, and reader responses suggested the work had some positive impact on Hispanic youth attitudes toward alcohol (Lalonde, Rabinowitz, Shefsky, & Washienko, 1997).

Those in special circumstances (e.g., the homeless) require different approaches in the effective delivery of prevention services. For example, reaching and engaging the homeless requires different strategies (Federal Task Force on Homelessness and Severe Mental Illness, 1992) and some researchers have been successful (reduced drug use) with prevention programming for the homeless (Botvin and Dusenbury, 1992).

3. Effective prevention programs use behavior change technology to equip people with life skills, knowledge of substance abuse, and awareness of the services available to them.

Equipping people with life skills includes decision making; coping; knowledge about the effects of alcohol, tobacco, and other drugs; awareness of services; and assertiveness/refusing. This cluster of skills also equips people with the ability to manage their immediate situations with the healthiest outcomes. Such strategies teach people to understand that they are engaging in risky behaviors and give them the skills to resist peer pressure and other influences, such as ADVERTISING. Recent studies have shown that alcohol advertising may increase consumption, while counter-advertising and bans decrease alcohol use to some degree (Saffer, 1997).

There is somewhat dated but nonetheless relevant literature of prevention technologies, such as *Life Skills Training* (e.g., Botvin & Tortu, 1988) or *Normative Education* (Hansen, 1990), which provide intensive instruction in a variety of competencies. Similarly, there are several comprehensive curricula offered sequentially from kindergarten through twelfth grade (Center for Health Promotion, 1990). Only two of these comprehensive school curricula have had positive outcomes based on experimental evaluations; these are the *Here's Looking At You* editions (Comprehensive Health Education Foundation, 1990) and *Growing Healthy* (e.g., Connell, Turner, & Mason, 1985). *Growing Healthy* is a comprehensive health curric-

ulum that includes a limited focus on alcohol, tobacco, and other drugs, whereas *Here's Looking at You: 2000* is an alcohol, tobacco, and other drug-use-prevention curriculum.

The results of a groundbreaking study were released in 1996, when the U.S. Department of Education published the results of a word association test called the Environmental Assessment Initiative (EAI). The EAI looks at the language people use and from that determines attitudes and beliefs about alcohol—indeed, the EAI study reported 80 percent accuracy in noting differences between users and nonusers regarding perceptions about drugs and alcohol (Katz, 1996). The study suggests increasing the influence of students who do not overindulge in alcohol as a way of improving campus life. Possible steps include offering numerous activities that do not involve alcohol, as well as developing strategies and rules that shed romanticized views of alcohol abuse.

Effective PREVENTION PROGRAMS must provide accurate information that there are risks associated with the use of various substances. This scientifically based information—highlighting the relationships between an abused substance and its consequences—has been an important component in changing behavior in all age groups (Johnston, Bachman & O'Malley, 1993).

4. Effective prevention programs emphasize the early identification of risks and resiliency factors and program accordingly.

Effective substance abuse prevention programs emphasize early identification and intervention to reach a substance abuser and his or her family as early as possible, even in preschool programs. Risk status assessment coupled with interventions have become standard in effective prevention programs (Lorion, Bussell, & Goldberg, 1991).

Some communities are expanding programs such as Drug Abuse Prevention Education (DARE) from elementary classrooms into the junior high schools as well, hoping to send youths a positive message early and often—and at an age when many children are first exposed to drugs and alcohol.

Research by Hawkins and Lishner (1985) lists risk factors for school-age youth. These risk factors are important to a total process in planning for prevention services.

1. family history of alcoholism
2. family history of antisocial behavior or criminality
3. family management problems
4. early antisocial behavior and hyperactivity
5. parental drug use and positive attitudes toward use
6. academic failure
7. little commitment to school and education
8. alienation, rebelliousness, and lack of social bonding to society
9. antisocial behavior in early adolescence
10. friends (peers) who use drugs
11. favorable attitudes toward drug use
12. early first use of drugs

Risk factors for other age groups need to be researched if prevention practitioners are to be maximally effective in addressing all populations in a given community. Efforts have also focused on developing resilience in people at high risk (Northeast Regional Center for Drug-Free Schools and Communities, 1992).

5. Effective prevention programs operate in communities that establish positive norms through enforcement of clear policies.

Communities that establish positive norms regarding alcohol, tobacco, and other drug use have also been successful in delaying the onset of use. Such communities have changed their policies toward access to substances by children and adolescents, including the location of advertisements and beverage-serving establishments; they have also promoted positive lifestyles. Gerbner (1990) has underscored the importance of communities reducing their ambivalence about communicating about all substances, licit or illicit.

Prevention services and policy changes have reduced the regular use of alcohol, tobacco, and other drugs, and there has been a concurrent reduction in consequences—including reduced highway ACCIDENTS because of alcohol; improved general health because of tobacco prevention; and reduced criminal activity because of illicit substance abuse. A 1992 report from the Office of the Inspector General confirmed an almost total lack of enforcement efforts by state agencies to control cigarette access, despite numerous provisions in existing state laws. In a study of media programming targeted to specific audiences and combined with community follow up, significant differences in the use of alcohol, tobacco, and other drugs were found between ex-

perimental and control groups (Flay & Sobol, 1983).

Pentz and colleagues (1989) demonstrated the effectiveness of community immersion in prevention through a program that included policy changes, refusal-skill training for junior high students, parent training, and mass media coordination. In this program, community groups monitored the availability of alcohol, tobacco, and other drugs and, in turn facilitated enforcement of existing policies or implemented new policies where needed.

An example of an ambitious prevention initiative is The Higher Education Center for Alcohol and Other Drug Prevention, created by the U.S. Department of Education in 1993. Alarmed by a Harvard study that confirmed almost half of U.S. college students engaged in heavy episodes of drinking, The Center formed the Presidents Leadership Group in 1997. This collaboration marked the first time in a decade that a group of college and university leaders joined forces to review alcohol abuse and develop a plan of prevention.

The Group published a report in 1997 that asked university presidents to acknowledge three major facts: student alcohol abuse is a problem all institutions of higher education share; student substance abuse is a problem of the community as a whole, not simply the campus; and student drinking is a problem that will never completely go away. The Group then listed their thirteen Proposals for Effective Prevention, among them: college presidents should use every opportunity to speak out and write about alcohol and other drug prevention to reinforce it as a priority concern and to push for change; college presidents should work to ensure that all elements of the college community avoid providing mixed messages that might encourage alcohol and other drug abuse; college presidents should appoint a campus-wide task force; college presidents should offer new initiatives to help students become better integrated into the intellectual life of the school, change student norms away from alcohol and other drug abuse, and make it easier to identify students in trouble with substance abuse; and college presidents should take the lead in identifying ways to effect alcohol and other drug prevention through economic development in the community (The Higher Education Center for Alcohol and Other Drug Prevention, 1997).

In November, 1996, forty-nine college presidents in Ohio decided to address the problem of student binge drinking by signing a letter of commitment. Institutions soon formed action teams to develop prevention plans. Educators found that communities reacted positively to university commitment against alcohol abuse.

6. Effective prevention programs provide staff development and training.

Effective prevention programs provide training for staff at all levels. The behavior-change and the other intervention techniques require constant upgrading of staff skills, supervision, and feedback. New prevention and intervention techniques require intensive training for proper implementation. This specialized training should be available at colleges, universities, and vocational training centers. Moreover, there is a world of information on alcohol and drug abuse education available on the Internet, including home pages for DARE and The Higher Education Center for Alcohol and Other Drug Prevention, as well as dozens of support sites.

The results of the several controlled-outcome studies of teacher training have been summarized by Swisher and Ashby (1993). They concluded that for each negative result (e.g., increase in beer use) there were five positive findings (e.g., reduced use of various substances). The training involved a ten-day retreat in which teams of teachers were given planning skills and prevention techniques. The planning skills led to an action plan to be implemented upon return to one's school; the prevention techniques were designed to be immediately implemented and reinforced with additional training sessions and technical assistance. Students in these schools have reported an improved school climate and improved academic functioning.

ENDURING MYTHS

Myths about prevention of substance abuse continue to impede progress toward more effective services. Some of the myths that need to be addressed as part of an advocacy for effective prevention principles include the following: (1) substance abuse cannot be prevented because it is caused by genetic and other biological phenomena; (2) there is no evidence that prevention works; and (3) scarce resources should be given to increasing availability of treatment for those in need.

Instead, most problems in this area are seen as being caused by the interaction of the biology, psychology, and social environment of the individual and the term that is emerging is *biopsychosocial problems*, indicating an interaction of these domains in social problems. There is clear evidence that genetic and other biological factors play a role in substance abuse, but more important is the social environment at all ages, which plays a significant role in increasing risk for the onset of a disorder. In some cases, it is possible to provide at-risk individuals with coping skills before a crisis occurs—to better enable them to avoid or manage the event (Institute of Medicine, 1989).

A large number of studies indicate that prevention works. For example, an issue of the *Journal of Community Psychology* (Lorion & Ross, 1992) included a series of articles that clearly demonstrated that prevention services for high-risk youth can reduce alcohol, tobacco, and other drug use as well as related social problems. The American Psychological Association published a well-documented listing of successful prevention programs (Price, Cowen, & Lorion, 1988). An outstanding longitudinal study was reported by Botvin (1993), in which he outlined a successful six-year follow-up of life-skills training.

CONCLUSION

Providing prevention services at any point along a continuum reduces demand while reducing costs for subsequent services. It also reduces related costs, such as accidents, illness, death, and crime. It is most cost effective to provide services as early as possible. However, budget priorities continue to emphasize law enforcement and treatment over prevention.

For prevention to play an appropriate role in responding to the problem of drug use and abuse, the federal, state, and local governments need to establish standards and ensure that the best practices in prevention and education are provided to all ages. The major obstacle remaining is the lack of means to train professionals and volunteers in what is known and to assist them in implementing the best practices. Unfortunately, most of the very limited government monies available for prevention of substance abuse are allocated to a flowthrough blockgrant mechanism or to the development of new models—without a follow-up system of disseminating or replicating what is already known about effective prevention.

(SEE ALSO: *Adolescents and Drug Use; Disease Concept of Alcoholism and Drug Abuse; Families and Drug Use; Homelessness, Alcohol, and Other Drugs; Parents Movement; Partnership for a Drug-Free America; Prevention*)

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EIGHTEENTH AMENDMENT *See* Alcohol: History of Drinking; Temperance Movement; Women's Christian Temperance Union

ELIMINATION OF THE DRUG ADDICTION AND ALCOHOLISM CATEGORY IN SOCIAL SECURITY DISABILITY PROGRAMS Since 1950, the federal government of the United States has provided income support by welfare or social insurance mechanisms to individuals with work disabilities unrelated to military service. Currently, the Social Security Administration operates two programs for the disabled: Social Security Disability Insurance (DI) and Supplemental Security Income (SSI). The differences between them reflect a fundamental schism in the American welfare state, which is divided into "tracks" along the line of labor force attachment. As it name implies, DI is an "insurance-like" program: Workers make payroll deductions that over time qualify them for benefits based on average lifetime earnings should they ever become disabled. SSI, on the other hand, is a "welfare" program designed for individuals with little history of employment and few resources. Whereas income and wealth are no bar to the receipt of DI, SSI is "means- tested." Excluding (mainly) the value of a home and an automobile, no SSI recipient can have assets valued at more than $2000 (or $3000 in cases where two beneficiaries are married). Some people collect both SSI and DI (they get "concurrent benefits") because although they qualify for DI, their benefit level is so low that it is augmented by SSI.

Typical of income maintenance schemes in liberal welfare states, the American system emphasizes economic returns to work, and thus social insurance offers more substantial benefits than welfare. In March 1999, the average monthly benefit for DI recipients was $773, whereas the federal minimum SSI benefit for sighted individuals under 65 years old and living in their own households was $500 per month. Some states (notably Alaska, California, and Connecticut) augment the federal minimum with a state supplement, but even in states the value of SSI is substantially less than the average DI payment.

For both SSI and DI, statue defines disability as "the inability to engage in any substantial gainful activity by reason of any medically determinable physical or mental impairment which can be expected to result in death or has lasted or can be expected to last for a continuous period of not less than 12 months." The rules and procedures used to determine if a case falls within this definition are also the same for both programs. Substantial gainful activity is defined as the performance of significant physical or mental activities for remuneration of profit at the level of $700 per month. Since a series of federal court rulings in 1993 and 1994 (codified by statue in 1994), illegal activities such as prostitution and drug dealing have been included in its meaning. Thus, an addict supporting a $700 per month heroin habit through prostitution, for example, may on this evidence be ruled able to work.

GROWTH OF THE DRUG ADDICTION AND ALCOHOLISM IMPAIRMENT CATEGORY

From the advent of SSI in January 1974, until March 1996, drug addiction and alcoholism (DA&A) were treated as potentially disabling impairments. The DI program adopted the more liberal SSI addiction standard in 1975. In Social Security lingo, beneficiaries who qualified on this basis were known as "DA&As." In the SSI program, DA&As were obliged to be in treatment and to have a "representative payee," a third party who received their checks and managed their funds. "DA&As in the DI program were not subject to such requirements until 1994. This disparity reflected the historical reference tendency for American income maintenance programs to combine material aid and moral surveillance in welfare programs (Welfare Policy and Substance Abuse in the United States), but to treat the beneficiaries of the "insurance-like" programs as though they were the recipients of an insurance benefit for which they had paid premiums in full.

Because there were no practical consequences of DA&A classification for DI recipients, the Social Security Administration had no accurate count of them until 1995. In the SSI program there were fewer than 10,000 DA&As as late as the end of 1986. By mid-1996, however, there were almost 166,000 SSI DA&As (including concurrent beneficiaries) and almost 43,000 DA&As on DI—a total of about 209,000. Overall, the two disability pro-

grams grew substantially during this period, but the DA&A category swelled disproportionately.

Most of the growth in the DA&A category occurred after 1989. Some of it was artifactual, stemming from the Social Security Administration's more accurate identification of DA&A cases after 1991. However, most of the growth was real and seems to have resulted from four additional factors, the precise contributions of which cannot be specified. First, federal circuit court decisions during the mid-1980s removed substantial technical obstacles to claimants seeking benefits on the grounds of addiction. Second, in the wake of these decisions many state and county governments set out to transfer to SSI (a program funded almost entirely by the federal government) recipients of General Assistance, a welfare program supported entirely with state and local funds. To promote this process, some states (like Illinois) contracted with private non-profit legal advocates to support applications and appeals. This very effective "cost-shifting" strategy was also appealing in view of the spiraling costs of medical care that were overwhelming many public hospital systems supported substantially by state and local tax revenues. As SSI beneficiaries automatically qualified for Medicaid (a federally supported, means-tested medical assistance program) in 39 states and the District of Columbia, and as DI recipients qualified for Medicare (Medicaid's non-means-tested counterpart) after a waiting period, this represented a second important source of savings for state and local governments. When the DA&A SSI population is disaggregated by state, it is clear that California, Michigan, Illinois, and a few others made much higher per capita use of the DA&A category than did other states. For example, by 1996 Oregon had as many DA&As on SSI as Texas, a state with several times the adult population of Oregon.

The last two contributors to the growth in the DA&A rolls are related to a famous Reagan-era controversy concerning the Social Security disability programs. During the early 1980s, responding in part to a Carter Administration initiative and also drawing on a similar tactic applied during his governorship of California between 1967 and 1974, President Reagan's Social Security administrators launched a roll-cutting campaign that relied on "continuing disability reviews" (CDRs). As a result, over 500,000 people lost federal disability benefits, a large percentage of them people with mental illness. Subsequent backlash from the courts and Congress restored many to the rolls, further liberalized eligibility criteria, and all but paralyzed the CDR process for years to come. As a result of perennially backlogged CDRs, many DA&As who regained their ability to work remained on the rolls, particularly as the economic conditions of the late 1980s and early 1990s provided few opportunities for poor, unskilled, ill-educated people. In part as the result of this episode, and in part due to the dramatic rise in homelessness during the 1980s (HOMELESSNESS, ALCOHOL AND OTHER DRUGS ENTRY), the Social Security Administration was charged with increasing its outreach efforts, especially among homeless people. This brought more DA&As into the application process.

CONTROVERSY AND DEMISE

Throughout the history of the DA&A program, the Social Security Administration relegated it to the Agency's backwaters. With no specific appropriations from Congress to ensure that DA&As received treatment or were separated from the rolls for failing to participate, and with no resources to thoroughly investigate the relationship of representative payees to beneficiaries, the Agency allowed the program to drift. However, it attracted a great deal of critical and unwanted attention as it began to grow rapidly. Beginning in 1991, the program was the subject of unflattering reports from federal watchdog agencies and a mounting number of highly publicized incidents involving DA&As using benefits to purchase drugs and signing up representative payees (like bartenders) with little fiduciary interest in them. The more scandalous claims about the program were largely unfounded, but the DA&A program was repugnant to many legislators and representatives of the alcohol and drug treatment community who saw it to be "enabling" addiction. Moreover, the program's rapid growth, and the Social Security Administration's apparent inability to curb it, lent credence to the claim that it was an entitlement program "out of control" in an era of bipartisan fiscal retrenchment.

In August 1994, after Congressional hearings and national media coverage (almost exclusively negative), Congress limited DA&A benefits to three years, reiterated the necessity to participate in treatment, and made DI DA&As subject to treat-

ment and representative payee requirements for the first time. Although Social Security Administration made no efforts to defend the DA&A program, it worked very hard to implement treatment referral and monitoring arrangements in all of the states. But as it did so, the November 1994 elections shifted control of the House of Representatives to conservative Republicans who were hostile to the DA&A program. As house welfare reform legislation shaped up during 1995, it became clear the DA&A program would be terminated.

On March 29, 1996 Congress eliminated the DA&A category in the Social Security disability programs, the first time any qualifying impairment had been legislated out of existence. The benefits of 209,000 recipients of SSI and DI were to cease after 1996 unless they applied for redetermination and were reclassified on the basis of other impairments (mental illness, for example). Only 34 percent had been reclassified to the rolls by the end of 1997.

CONCLUSION

In retrospect, the demise of the DA&A program seems to have been over-determined. It was at once culturally problematic and thus deprived of a unified constituency; extremely difficult to administer (and thus distinctly unloved by the Social Security Administration); and as a result of its administrative problems, susceptible to discrediting scandal. The program left behind a legacy of mandatory treatment and representative payee provisions that may become common features of state and local welfare reform measures, but no observers see any chance of its resurrection at the federal level in the foreseeable future.

(SEE ALSO: *Welfare Policy and Substance Abuse in the United States; Homelessness, Alcohol, And Other Drugs*)

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EMPLOYEE ASSISTANCE PROGRAMS (EAPS) An Employee Assistance Program (EAP) consists of employer-sponsored services intended to aid employees with personal problems that may adversely affect their job performance. Initially developed to address alcohol-related problems, over the last fifteen years EAPs have emerged as a common response to the problems of ALCOHOL and drug abuse in the workplace. In addition, they provide a variety of services to help employees and their families resolve health, emotional, marital, family, financial, or legal concerns.

While the exact mix of services provided depends on a number of variables, such as size and type of company, EAPs generally offer, at a minimum, confidential client counseling, problem assessment, and treatment referral. A comprehensive EAP offers

1. assessment and referral—EAPs conduct psychosocial assessments to guide decisions to refer clients to treatment and the choice among treatment alternatives
2. treatment follow-up—client follow-up and reintegration into the workplace is an essential EAP function
3. supervisor, management, and union representative training—training provides the information needed on how and when to use the program and how to best assist employees who use it
4. employee education—information on a broad range of problems and how to use the EAP.

The delivery of EAP services may take several forms, depending on such factors as the organization's size and structure. Large companies and organizations, unions, and employee groups often operate their own programs. These services are most often housed within the human resources or medical departments. Smaller organizations, or organizations with dispersed worksites may find it more advantageous to contract with an independent EAP provider located outside the company. A newer trend among small employers is the development of consortium EAP arrangements in which a number

of small employers contract with an external provider to provide EAP services.

In the 1980s and 1990s, the number of EAPs grew dramatically. The Employee Assistance Professionals Association estimates that by the 1990s, 20,000 EAPs were in place in organizations throughout the United States. The Department of Labor's Bureau of Labor Statistics reported that nearly 12 percent of the nonagricultural establishments they sampled offered EAP services. Further, they found that of those sampled, the probability of an establishment offering EAP services increased as a function of establishment size, ranging from 79 percent of employers with over 250 employees, to 9 percent of employers with fewer than 50 employees.

Rapid growth in the number of EAP progams has led to heightened scrutiny concerning their cost effectiveness; in the current economic climate, EAP programs will experience increased pressure to conduct evaluation studies that provide empirical evidence of their efficacy. More research is needed to identify and improve the most essential program components and to aid in tailoring programs to fit specific needs.

Costs incurred in providing EAP services vary widely, but their presence has been clearly tied to overall savings in a number of areas. For example, the McDonnell Douglas Corporation of St. Louis found that employees utilizing their EAP services betwen 1985 and 1988 for an initial assessment before being referred to treatment had 44 percent fewer lost work days, 81 percent lower termination rates, and lower total four-year medical claims per person than employees seeking treatment for chemical dependence without first consulting the EAP.

For many companies, the approach taken to minimize the impact of drugs in the workplace incorporates a number of additional elements that complement EAPs and constitute a comprehensive strategy. These include a clearly stated formal policy prohibiting drug use, consequences for violating the policy, and alternative strategies to deter drug use.

The Employee Assistance Professionals Association may be consulted for further information: Suite 1001, 4601 North Fairfax Drive, Arlington, VA 22203.

(SEE ALSO: *Drug Testing Methods and Clinical Interpretations of Test Results; Industry and Workplace, Drug Use in; Military, Drug and Alcohol Abuse in the U.S.; Productivity*)

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ENDORPHINS Endorphins are a group of peptides with potent ANALGESIC properties that occur naturally in the brain. The word *endorphin* is a contraction for the words *end*ogenous and *morphine*; it was coined by narcotics researchers in 1975 as the preferred term for a then hypothetical natural substance capable of action at RECEPTORS for OPIATES (such as HEROIN). The underlying hypothesis was that an endorphin NEUROTRANSMITTER utilized the receptors at which morphine and related drugs exerted their actions. After extensive and intensely competitive research by many groups, three distinct types of such endogenous opioid peptides were found (*peptides* are segments of linked amino acids that can act as neurotransmitters). By 1999, additional peptides able to act at opioid receptors as well as to regulate pain sensitivity through nonopioid receptors had been identified.

Each type of opioid peptide gives rise to one or more opioid peptide prohormones, which are then modified by enzymes in tissues to convert the larger inactive peptides into smaller active ones. For example, the pro-opiomelanocortin prohormone is synthesized in the corticotropes in the anterior pituitary gland and separately in hypothalmic and medullary neurons is cleaved in those cells to β-endorphin, a 31 amino-acid peptide with the greatest intrinsic opioid activity. Each active natural opioid peptide contains the tetrapeptide tyrosine-glycine-glycine-phenylalanine at its amino terminus. The fifth amino acid is either methionine (resulting in the so-called Met^5 enkephalin) or leucine (resulting in leu-enkephalin). Opioid peptides derived from plants—for example, caseimorphin—have also been described. The opioid peptides, of which the proenkephalin- and prodynorphin-derived peptides are most widespread, are found in specific neurons in the brain.

(SEE ALSO: *Enkephalin; Opiates/Opioids*)

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ENFORCEMENT STRATEGIES AND TACTICS *See* Drug Interdiction

ENKEPHALIN Enkephalin is either of two pentapeptides (containing five amino acids) with OPIATE and ANALGESIC (painkilling) activity, occurring naturally in the brain, with a marked affinity for opiate receptors. ENDORPHIN was initially the name for all opioid-like NEUROTRANSMITTERS in the brain; the research team of Hans Kosterlitz and John Hughes gave their own name, enkephalin (a variant of *en-cephal* ["of the brain"]), to the two opioid pentapeptides that they had purified from ox brains (ca. 1977). They confirmed their discovery by showing that the effects of synthetic peptides were the same in bioassays using opiate RECEPTORS and that both Met^{5}enkephalin and Leu^{5}enkephalin were authentic endogenous opioid peptides.

(SEE ALSO: *Opiates/Opioids*)

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ENZYME-MULTIPLIED IMMUNOASSAY *See* Drug Testing Methods and Clinical Interpretations of Test Results

EPIDEMICS OF DRUG ABUSE Hearing the word *epidemic*, one often thinks first of the flu, measles, the ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS), or some other contagious disease spreading through a community. In epidemics with person-to-person spread of infection and disease, people become infected and fall victim to the disease, and in the process they come into contact with other people, who in turn get the infection and disease. Often, what is being spread from person to person is not the disease itself, but rather an agent of the disease—for example, one of the viruses that accounts for influenza, the measles virus, or the human immunodeficiency virus (HIV) that causes AIDS.

In EPIDEMIOLOGY (the study of epidemics), it is not the agent, the person-to-person spread of a disease, or the intentional or unintentional nature of acquiring the infection or disease that defines an epidemic. Instead, an epidemic is defined as an unusual occurrence of an infection, disease, or other health hazard in a population. The contrast between "usual" and "unusual" most often is determined by looking at the number of cases that have been occurring within the population over time. If the number of cases occurring in the population this month (or year) is notably greater than the number of cases that occurred in the population during each of the prior months (or years), then it is legitimate to talk of a growing epidemic.

An epidemic may be most obvious when the number of cases goes from zero to a much greater number in a relatively short span of time. For example, before the middle 1970s, the U.S. population apparently had no cases of HIV infection or AIDS. For those years, the usual number of cases per year was zero. Since then, the country has seen a mounting number of HIV infections and AIDS cases each year, and it has become a raging epidemic. Compared to the previous usual number of cases per year, the United States faces an unusual occurrence of disease in the form of thousands of cases per year.

The same concept can be applied on a smaller scale. In the mid-1990s there still are small cities and communities where apparently no one in the population has yet acquired the HIV infection. Health officers who watch over these populations may speak legitimately of an HIV epidemic once the number of cases occurring in the population begins to mount, and there is no need to wait until there are hundreds or thousands of cases before describing the epidemic situation. This is because epidemics are not defined by the absolute number of cases that are occurring. In the early 1990s, there was an epidemic outbreak of hantavirus infection

and hantavirus-related deaths in the southwest United States. Because the usual number of hantavirus-related deaths in this region was zero, the situation was declared to be an epidemic well before 100 cases had occurred. Sometimes an epidemic that is limited to a certain place or time will be called an *outbreak*, but this distinction is not a technical one.

There are also epidemics even when no person-to-person spread is involved. For example, in the middle of the twentieth century, there was an epidemic of infant blindness due to retrolental fibroplasia, induced when premature infants were kept in incubators with excessively high concentrations of oxygen. These very high concentrations of oxygen were not a result of machine failure. Instead, the number of cases of retrolental fibroplasia and associated blindness kept growing as ever more hospitals raised the oxygen concentration within incubators in a misguided effort to increase survival of the infants by enriching their oxygen supply. Later, clinical and epidemiologic studies showed that this effort to save lives actually led to the increased occurrence of blindness.

Sometimes people object to the usage of the term *epidemic* as applied to drug dependence because it is believed that people bring drug problems down upon themselves by their careless behavior. Epidemiologists, however, typically do not recognize the distinction between "careless" and "careful" behavior when it comes to epidemics. For this reason, they have no trouble speaking about epidemics of syphilis and AIDS, which in some degree are linked to unprotected sexual behavior, something that many would regard as careless behavior.

In summary, the evenhanded application of the concept of epidemic makes it clearly legitimate to speak of an epidemic of smoking-related lung cancer or emphysema, an epidemic of liver cirrhosis due to drinking of alcoholic beverages, an epidemic of leukemia induced by ionizing radiation, an epidemic of mental retardation due to rubella (German measles) infection during gestation, an epidemic of motor vehicle crashes, and an epidemic of deaths by homicide, as well as epidemics of drug use and drug abuse. In order to use the term *epidemic* to describe the health-related experience of a nation, state, or community, it is necessary to demonstrate an *unusual* occurrence of the condition in the population during some specified span of time, relative to the number or rate of cases that occurred in the population during the immediately prior time spans. There is no need to limit usage of the term to infectious diseases with known agents such as rubella or HIV: nor is there a need to limit its usage to diseases spread by person-to-person contact or to be concerned whether the spread of the disease involves careful or careless behavior.

EPIDEMICS IN THE UNITED STATES

An unusual occurrence of drug use or an unusual occurrence of problems connected with drug use can be referred to as epidemics of drug use and drug abuse. In the mid-1990s in the United States, there were multiple indications that the nation had gone through its second major epidemic of COCAINE use and now was in the end-stages of that epidemic.

The first U.S. epidemic of cocaine use started in the late nineteenth century and early twentieth century when cocaine was marketed widely in a variety of forms, including Coca-Cola, Vin Mariana (a wine containing cocaine), and other cocaine products sold without a doctor's prescription. That epidemic subsided, in part because of increased federal and state restrictions on importation and marketing of cocaine, as well as new labeling requirements for patent medicines and other over-the-counter products.

From 1920 through the early 1960s, cocaine use in the United States was not a usual occurrence outside of relatively small circles of HEROIN users, movie and television stars, jazz musicians, and others who came into contact with illicit suppliers of the drug. In the early 1970s, when the federal government began supporting a series of national and state surveys of illicit drug use, cocaine use was found so rarely that it was difficult to get a reliable impression of the characteristics of the cocaine users—there were too few of them in the survey samples.

By studying the series of survey reports from 1972 through the mid-1990s, it is possible to plot the growth of this second U.S. epidemic of cocaine use from what had been typically low levels of use to increasingly greater numbers of cocaine users. The peak years of the epidemic use seem to have been in the late 1970s, which were followed by declining numbers of cocaine users in subsequent years, notwithstanding a small rally in the mid-1980s in connection with the emergence of crack-cocaine smoking.

Although the number of active cocaine users in the U.S. population has dropped back toward the levels observed in the early-to-middle 1970s, it seems that an epidemic of cocaine dependence is still very much in evidence, if the definition of cocaine dependence is meant to encompass very frequent cocaine use as well as the cocaine dependence syndrome described in the more formal terms of clinical research. That is, as the epidemic of cocaine *use* subsided in the late 1980s and early 1990s, there was no parallel falling off in the numbers of daily or other frequent cocaine users, and there was no clear drop in the number of people actively affected by cocaine dependence. Indeed, in the mid-1990s, the number of active cases of cocaine dependence in the population seems to be greater than it ever has been in the nation's history. Thus, it can be said that the epidemic of cocaine dependence is not yet over, for there continues to be an unusually large number of cocaine-dependence cases in the population. There is not yet enough evidence to say whether fewer newly occurring cases of cocaine dependence are developing in the U.S. population. Once it can be shown that the new occurrence of cases has fallen off, it can then be said with more confidence that the nation has entered a declining phase in this most recent epidemic of cocaine dependence.

With their attention focused upon a declining number of cocaine *users* in the early 1990s, the American public and politicans seemed to turn their attention away from the nation's cocaine problems. At the same time, the level of support for treatment of drug dependence dropped from relatively high levels of expenditures in the mid-1980s, even though the number of people suffering from cocaine dependence had remained about the same as it was during the late 1980s. This set of circumstances underscores the political importance of drawing a distinction between epidemics of drug use versus epidemics of *drug dependence or drug-related problems*. It is likely that many Americans equated declines in the number of cocaine users with declines in the number of cocaine-dependent persons: they were not aware that the epidemic of cocaine dependence continued even as the epidemic of cocaine use was subsiding dramatically.

Coincident with decline of cocaine use within the United States, several other drugs have been the subject of increased attention and use, including drugs whose past popularity has re-emerged in recent years. This comeback of older drugs might be due to newer cohorts of drug users with no experience of friends suffering the adverse consequences associated with the drug, or possibly due to a change in either the availability, purity, or administration of the drug which would make its use more attractive, accessible, or reinforcing. Two examples of this re-emergence are methamphetamine and heroin.

Methamphetamine, a subgroup of amphetamines, was widely used in the 1960s and 1970s. Also known as "speed," "crank," "meth," "zip," and "ice," the medical and nonmedical uses of methamphetamine have included appetite suppression for weight loss, staying awake, and recreation. The stimulant effect is similar to that of cocaine, but with longer duration.

Methamphetamine use has appeared in outbreak and epidemic form in Asia, the Pacific Islands, and primarily southwestern parts of the United States since the middle of the 20th century, often in the form of "ice" smoking (i.e., inhalation of volatile fumes). In the early 1990s less than two percent of the population over the age of twelve had tried methamphetamine, according to national estimates. This number increased fifty percent in the later part of the decade and now remains relatively steady as we enter the 21st century. Among teenagers, the number of methamphetamine users doubled during the1990s. Emergency room admissions associated with methamphetamine use increased nearly 350 percent from the early to the middle of the 1990s; admissions to treatment increased nearly four hundred percent from the early to the late part of the decade. Outbreaks of "ice" smoking have spread northward and eastward from the southwestern United States, suggesting an epidemic pattern in the United States in the 1990s, still persisting in the year of publication.

Prevalence estimates of heroin use had been relatively consistent during the 1980s, but early in the 1990s the purity of the drug increased dramatically, as did its availability. The heightened purity allowed for modes of administration other than injection, such as snorting and "smoking" (inhalation of volatile fumes), opening a door to heroin use for the drug users who otherwise might abstain due to an aversion toward injection.

Initiation of heroin use among youths in the mid-1990s was at its highest level in nearly 30 years. From the mid-1990s to the end of the dec-

ade, the proportion of heroin users using needles remained unchanged while the proportion sniffing or snorting increased from 50 percent to 75 percent. Much of the new heroin use is within the population under age 25. Heroin use started to increase in the early 1990s and continued through the end of the decade. It now seems to have stabilized.

OTHER PAST DRUG EPIDEMICS

An epidemic during the third century B.C. of "hanshi" use at the end of the Han dynasty in China and the spread of tea drinking prior to 900 B.C., might be the earliest documented epidemics of PSYCHOACTIVE DRUG use in the world, not counting outbreaks of excessive ALCOHOL use (see ASIA, DRUG USE IN).

In the 1600s, in Europe, there were epidemics of CHOCOLATE (cocoa) consumption, TOBACCO consumption, and COFFEE consumption. These epidemics followed shortly after colonization of the Americas by Europeans and were sustained by ever-increasing supplies of these products shipped from the cash-poor colonies.

During the nineteenth century, many Europeans became enthusiastic about the inhalation of ether, an intoxicating volatile substance that was investigated for its medical uses by John Snow, one of the fathers of modern epidemiology. Although definitive statistics are not available, it appears that nonmedical inhalation of ether spread through Ireland in an epidemic fashion during the nineteenth century, as did inhalation of NITROUS OXIDE (laughing gas) in the United States. Also during the nineteenth century, China and several other countries experienced epidemics of OPIUM consumption, especially opium smoking. In part, an increased spread of opium smoking in the Americas prompted passage of antiopium legislation, which ultimately produced international agreements that curbed the supply and distribution of opium and opium products worldwide.

It has been said that the international agreements on these drugs were less effective than the public-health and punitive actions taken within countries to curb opium smoking. For example, harsh jail sentences were imposed for violation of city, state, and federal laws concerning opium, and a tradition of executing "drug criminals" was started in some countries. In Communist China, according to some stories, capital punishment of drug dealers and drug users account for the virtual disappearance of drug problems in that country. The truth of these stories cannot be known.

About the same time that the international agreements on opium and opium products were passed, the United States experienced an increase in tobacco smoking, ultimately with peak population levels of tobacco smoking occurring during World War II and the following years, before declines occurred in conjunction with the surgeon general's 1962 report on smoking and health and other publicity about the health hazards of smoking. When one considers the social climate of the 1990s, a time when tobacco smoking was not at all a socially approved drug-use practice, it may be difficult to imagine that during World War II Lucky Strikes and other cigarettes were passed out to soldiers as part of their daily food rations. This turned out to be an effective way to sustain the epidemic of tobacco smoking, but one cannot be sure whether the tobacco industry's intent was primarily to boost the morale of soldiers or to create and build market strength for tobacco cigarettes. Someone interested in the history of epidemiology might be able to sort this issue out, if industry records from that time were opened for inspection.

A more definitive case can be built for the marketing strategies that have been used to increase and build market strength for smokeless tobacco products such as snuff. There was a tremendous increase in the youthful usage of smokeless tobacco between 1970 and 1985. This increase has been traced to deliberate marketing strategies, including formulation of relatively low-cost, "unit dose" supplies of tobacco snuff that had been flavored to increase palatability.

While tobacco consumption was increasing worldwide, Japan's population was affected by an epidemic of METHAMPHETAMINE use during and especially after World War II; later distribution of this drug was seen throughout other countries of the world, including the Scandinavian nations and the United States. At one point in the 1950s, it was estimated that 2 percent of Japan's population had taken methamphetamines nonmedically. It also has been said that especially harsh jail sentences and other criminal penalties accounted for the termination of the amphetamine epidemic in Japan, but as noted in regard to capital punishment and prior Asian drug epidemics, there is no good evidence on

this issue. Between 1945 and 1965, other countries saw amphetamine epidemics come and go without the implementation of especially harsh criminal penalties.

The prevalence of nonmedical STIMULANT use in the 1950s did not reach the 2 percent level in the United States as it had in Japan, but it was sufficiently widespread to yield congressional hearings that focused especially upon AMPHETAMINE use by long-distance truckers (e.g., those who used the drug to promote vigilance and stamina for lengthy trips) and by homemakers (e.g., those who took amphetamines to curb their appetite or because of their mood-altering effects). In part, these epidemics should be understood in relation to the relatively widespread availability of amphetamines in a context of limited regulation of supplies and distribution. These epidemics resulted in legislation and social action to reduce the supply and control the distribution of the amphetamine drugs. In the United States, two especially relevant pieces of federal legislation were the Drug Abuse Control Amendments of 1965 and the CONTROLLED SUBSTANCES ACT of 1970; these laws were directed at controlling the use of the amphetamines as well as the use of other drugs.

The usage of marijuana and the psychedelic drugs (e.g., LSD) grew during the 1960s and seems to have peaked during the 1970s. In the 1990s, there were conflicting reports of increasing consumption of these drugs, especially LSD. By some accounts, the nation entered a new phase of LSD usage. It appears, however, that this nationwide increase was not detectable in population estimates from the NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE, and it is possible that the apparent nationwide epidemic actually remains quite limited in scope.

Several noteworthy developments occurred in relation to HEROIN and the OPIOID drugs during the late 1960s and early 1970s. One important clinical and epidemiological research group based at the University of Chicago developed important innovative strategies for community-level intervention directed at outbreaks of heroin use and heroin dependence. An important element in the group's intervention plan was to employ outreach workers, including staff in recovery from heroin dependence, who would spend enough time on the street corners to identify both new and old users of heroin and to help them get into treatment and stay in treatment. In addition, in Britain, Richard de Alarcon adapted classical methods of epidemiologic research to study the diffusion of injecting drug use (especially injecting heroin use) as an epidemic phenomenon, by plotting the person-to-person spread of the epidemic over time and across the cities of that country.

In 1971, President Richard M. Nixon declared a "war on drugs" following a period of increased heroin use in the United States: he did this partially in association with the return of Vietnam veterans, many of whom had become users of heroin and other opioid drugs during their overseas tours of duty. This epidemic of the late 1960s and early 1970s was documented most readily by examining statistics on clients entering treatment for heroin dependence, including the lag of several years that separated users initial injection of heroin to their first admission for treatment. Despite the war on drugs, a decline in heroin use in the early 1970s was followed by another smaller epidemic of heroin use or dependence during the mid-1970s, followed by apparent decreases in the occurrence of heroin dependence during the late 1970s and early 1980s. The early decrease appears to have coincided with the decrease in importation of heroin to the United States from supplier countries such as Turkey and the mid-1970s increase with the emergence of Mexico and Southeast Asia as suppliers of illicit opiates.

When heroin is the drug of choice and heroin availability declines, users often take other drugs that provide the same functions—either opiate drugs derived from the opium poppy such as morphine or synthetic opioids derived in the chemistry lab and not requiring cultivation products from poppy fields. One example of a synthetic opioid is the so-called China White, which spread through the United States, especially on the West Coast. The number of overdoses linked to China White and related synthetic opioid drugs seemed to increase until the mid-1980s. Since then, there have been declines in the incidence of this type of overdose, possibly because of the increased supplies and street-level purity of poppy-derived heroin.

In addition to the cocaine epidemics already mentioned, there was a cocaine epidemic in the late twentieth century, which might have been sustained by the introduction of CRACK-cocaine, another unit-dose formulation of a psychoactive drug that reduces cost to a level that can be afforded (at least, initially) by many people. Other articles in

this encyclopedia discuss reasons that crack-cocaine smoking might have helped sustain the epidemic of cocaine use, including differences in the pharmacologic, pharmacokinetic, and reinforcement profiles of crack-smoking versus nasal insufflation of cocaine hydrochloride powder. In this context, it is interesting to note that the epidemics of crack smoking and cocaine use ended when they did, during a period of widespread availability of cocaine in a low-cost formulation. In epidemiologic terms, this development carries three very important implications. First, given widespread availability, many Americans had opportunities to smoke crack or take cocaine powder and did not do so. In some important way, these were Americans who were not susceptible to widespread media publicity and other conditions that otherwise might have promoted the use of crack or other forms of cocaine.

Second, for many Americans who tried crack or cocaine powder, the use of these drugs did not compete well with alternative behaviors that were as readily available to them in their home and community environments. They found that there were other, more reinforcing ways with which to occupy their daily lives. This signifies that within the population, for those who have used cocaine, there are differences in the users' susceptibility to becoming cocaine dependent.

Third, within the American population, the balance of these several kinds of susceptibility must have changed over the course of the 1980s. For example, during many other epidemics of contagious disease, as the balance of susceptibility changes, the people who are more susceptible become surrounded by people who are less susceptible. Sometimes, the balance of susceptibility changes without any active and organized public health intervention, as in the case of a typical influenza epidemic in an elementary school population. Sometimes, the balance of susceptibility is changed quite deliberately by organized public-health action, as in the successful worldwide effort to eradicate deadly smallpox by making sure that susceptible persons were immunized against smallpox, and by making sure that infected individuals were surrounded by those who were not susceptible by virtue of either immunization or past infection.

In the case of a drug epidemic, as the more susceptible individuals in the population start to become surrounded by people who will not or do not take the drug, it must be increasingly difficult for them to come into contact with the drug at an individual level, even when the drug supply is great at the societal levels. Furthermore, as the balance of the several kinds of susceptibility changes within the population, there must be an evolution of the social-influence processes that promote the spread of drug use from person to person: Fewer people are being pressured by peers to use the drug; fewer people are talking about the drug in favorable terms; more people are talking about how they had a chance to use it, but it just didn't seem worth it; more people are talking about how they have used the drug but it just didn't do very much for them.

This sort of process must have taken place with regard to the cocaine epidemic for the balance of susceptibility to have changed within the population; otherwise, the epidemic of cocaine use would have persisted. Because we do not have an effective biological vaccine that would reduce susceptibility to cocaine use the way the smallpox vaccine reduced susceptibility to smallpox infection, this change in the balance of susceptibility had to have been caused by something else. *Before* the epidemic of cocaine use had started to decline, the social demographer K. Singh hypothesized that it would decline simply because of demographic changes in the U.S. population caused by a declining birth rate fifteen to twenty-five years earlier. Singh apparently reasoned that, numerically, there would be fewer and fewer people aged fifteen to twenty-five, and this by itself would change the balance of susceptibility in the population because the developmental period from age fifteen to twenty-five is one that is at especially high risk for starting illicit drug use.

Later, and *after* the epidemic of cocaine use had started to decline, two other main hypotheses emerged. One of these took note of the demographic changes to which Singh had pointed but also drew on three other interrelated epidemiologic observations, namely that (1) cocaine use almost always starts after MARIJUANA use has started; (2) a history of marijuana use probably is the strongest indicator of susceptibility for trying cocaine; and (3) most marijuana users try cocaine once or a few times but do not go on to become dependent upon it (i.e., they are in the second kind of susceptibility group already mentioned). These three epidemiologic observations were also linked with an observation from ethnographic research: When a young

person is presented with an opportunity to try marijuana or cocaine, it very often is a slightly older person with a history of marijuana use who presents the opportunity. It might thus have happened that the cocaine epidemic had stopped growing and had started to end once the supply of cocaine had increased to a level where a large proportion of former and current marijuana users had been presented with an opportunity to use cocaine. When these marijuana users either declined to use cocaine or tried and then stopped using cocaine, they then no longer could serve as sources of diffusion to younger persons. That is, the change in balance of susceptibility within the population was related to the number of individuals who previously had tried marijuana and to whether they had completed the normative passage of (1) declining to use cocaine when it was offered to them or (2) trying cocaine a few times without becoming dependent upon it, thereby ceasing to be part of the vanguard of cocaine experimenters who in the glow of their first cocaine experiences would enthusiastically be offering cocaine to others.

According to the other main post-epidemic hypothesis, trends in the perceived danger or risk of harm associated with taking cocaine affected trends in cocaine use. Particularly after basketball star Len Bias died after smoking crack, more young people reported that they perceived there to be substantial risks of harm associated with taking cocaine. Concurrently, there were declines in reported levels of cocaine use. For a number of years, as surveys showed more and more young people reporting that they perceived cocaine use to be dangerous, the levels of cocaine use declined even further, despite increasing or stable levels of cocaine availability. These trends gave rise to the optimistic observation that perhaps it was the increases in perceived dangerousness of cocaine use that accounted for the declines in cocaine use. If such an observation were true, society might be able to stop or curb future epidemics by educating youths to perceive the harmfulness of drug use.

DRUG EPIDEMICS IN THE FUTURE

Singh's prediction based on an analysis of demographic changes in the population and the two main hypotheses that emerged after the epidemic of cocaine use had started to decline have historical importance. Although it was not possible to test these hypotheses about the 1975-to-1994 epidemic of cocaine use in the United States in any rigorous fashion, and it cannot be known for certain that any of them is correct, they may help in the plans for coping with future epidemics of drug use and drug dependence; they also offer pointers what kind of societal response might be needed if a rising line is perceived in the plotted curves of new epidemics. Nonetheless, until a more certain knowledge is acquired about the dynamics of epidemics of drug use, it will be premature for politicians or anyone else to ride to glory on the descending line of these curves. There is enough knowledge to take action, but not enough to say what specific combinations of public-health actions will be effective.

The array of public-health actions to stop or curb future drug epidemics have not yet been exhausted. In the 1970s, Dr. Jerome H. Jaffe and other experts suggested developing prevention strategies that would be based on concepts of reducing susceptibility to drug dependence. This might sound like science fiction, but recent new developments in molecular biology, immunology, pharmacology, and neuroscience have made a viable strategy of this type more plausible.

A relatively sharp increase in the use of steroids among youths towards the end of the 1990s suggests that investigation into its use, especially among males, might identify an emerging epidemic. Similarly, "club drugs" such as MDMA ("ecstasy"), Rohypnol, ketamine, and others, have shown increases in use and availability throughout the 1990s, primarily among youths and young adults.

Because of the novelty of these types of drugs, surveys designed to estimate the number of users have difficulty keeping up with their emergence in isolated outbreaks until use has persisted long enough and has become sufficiently prevalent to warrant inclusion in the survey assessments. For this reason, there are no definitive sources of epidemiological evidence on epidemics of drug use, akin to the evidence available for notifiable diseases such as syphilis and HIV infection. Readers interested in local area outbreaks and epidemics will find useful and sometimes definitive evidence in the periodic reports of Community Epidemiology Work Groups established by the United States National Institute on Drug Abuse, and its counterpart institutions in other countries.

In seeking to understand the future of drug epidemics in society, it will be necessary to complete more thorough studies of some predicted epidemics that did not materialize. For example, following the 1990/91 Persian Gulf war and 1992 posting of U.S. troops to Somalia in East Africa, it was said that the United States would suffer a khat-cathinone epidemic as soon as the veterans returned with the experience of seeing khat used by the people of the Middle East and Somalia—and when cathinone (one of khat's active ingredients) was extracted or synthesized by underground chemists for distribution. So far, however, the prediction of the nationwide KHAT-cathinone epidemics has been wrong. There have been isolated epidemics in a few communities but apparently no widespread use, and it is not altogether clear what curbed the spread to other communities.

As of the early 21st century, many countries have conducted epidemiological surveys to estimate the number of drug users in their populations, and some countries maintain substantial surveillance efforts to assess whether and when drug epidemics are occurring. No country, however, has made a substantial investment in the empirical study of drug epidemics. Most of the hypotheses and theories about drug epidemics remain untested against epidemiologic evidence, including a recently stated and fairly elaborate theory that incorporates what might be the necessary conditions for the expansion, the maintenance, and the decline of drug epidemics. It must thus be said that the present stage of applying epidemiology to the study of drug epidemics is a fairly primitive one.

(SEE ALSO: *Adjunctive Drug Taking; Alcohol; History of Drinking; Amphetamine Epidemics; Education and Prevention; Epidemiology of Drug Abuse; High School Senior Survey; Opioids and Opioid Control; Substance Abuse and AIDS Prevention Movement; U.S. Government Agencies; Vulnerability as Cause of Substance Abuse*)

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EPIDEMIOLOGY OF DRUG ABUSE One of the best ways to introduce an article on the epidemiology of drug use and drug dependence is to ask some basic questions that epidemiologic studies can answer but laboratory and clinical studies cannot. Here are some examples:

In the late 1990s in the United States, about how many ages 12 to 17 had used cocaine at least once?

In the late 1990s, within which U.S. population subgroups were active cocaine users most likely to be found?

Within the United States in the early 1990s, among those aged 15 to 24 who had used cocaine, what proportion had become dependent on it?

In the early 1990s, which age group within the U.S. population was most likely to have experimented with cocaine, and which age group was most likely to have developed cocaine dependence?

For a young adult living in the United States, what is the risk of developing the problem of alcohol abuse or dependence between one year and the next?

Is the risk for alcohol dependence greater for some young adults than for others?

Which subgroups of young adults are at especially high risk for alcohol dependence?

Are these same subgroups of young adults at especially high risk of becoming dependent on psychoactive drugs such as marijuana or cocaine?

To answer questions of this type, it is necessary to step outside the laboratory and clinical settings where drug users receive treatment. This step can be taken during the course of epidemiologic surveys that seek information about all aspects of the population's drug experience; the surveys take into account not only the relatively modest numbers of drug users who have received counseling and treatment, but also those who never have received any kind of health care or social services. The answers to these questions, based on epidemiologic surveys conducted in the United States between 1980 and the present, are as follows:

In the late 1990s, among those aged 12 to 17 in the United States, an estimated 496,000 to 682,000 had used cocaine at least once. As a proportion, this amounted to about 2.5 percent of those 12 to 17 in the United States at that time.

Within the United States in the late 1900s, young adult men aged 18 to 29 were more likely to be active cocaine users than any other population subgroup categorized by age and sex. For example, slightly more than 2.5 percent of men 18 to 25 were active cocaine users, as compared with 1.4 percent of men 26 to 34, 1.3 percent of women aged 18 to 25, and 0.9 percent of women aged 26 to 34.

Within the United States in the early 1990s, among those aged 15 to 24 who had used cocaine, an estimated 25 percent had become dependent on it. That is, for every four who had experimented with cocaine, one had become dependent on it.

Within the United States in the early 1990s, people of the 25 to 34-year age group were most likely to have experimented with cocaine; within this age group, about 30 percent of men had tried cocaine at least once, and about 21 percent of women had tried cocaine at least once. Cocaine dependence also was most prevalent in this age group: it affected about 4 percent of all persons aged 25 to 34. Among cocaine users aged 25 to 34, an estimated 16 percent had become dependent on it.

For those 18 to 29 living in the United States, the best available estimate for the risk of developing alcohol abuse or dependence between one year and the next is about 2 to 4 percent.

The risk of succumbing to alcohol abuse or dependence for males aged 18 to 29 is an estimated 6 percent per year, as compared with about 1 percent per year for females aged 18 to 29.

Males between the ages of 18 and 25 are at especially high risk of succumbing to alcohol abuse or dependence.

These same subgroups of young adults are at especially high risk of becoming dependent on psychoactive drugs such as marijuana or cocaine. When all the abuse or dependence syndromes attributable to nonmedical use of these drugs are considered, the estimated risk for males aged 18 to 29 of developing clinically recognizable drug problem is estimated at 4.4 percent per year; for females aged 18 to 20, it is about 1.6 percent.

There is, of course, good reason to wonder whether epidemiologic surveys of drug use and drug dependence have sufficient validity to be trusted. On the one hand, especially among young people, there may be a tendency to exaggerate drug taking, and to falsify survey responses in the direction of more drug taking than has really occurred. On the other hand, some people may be hesitant to disclose their histories of drug taking or drug problems; they might not agree to participate in the survey, or they might falsify their answers in the

direction of less drug taking or fewer problems than have actually occurred.

There fortunately is a body of methodologic research that provides some general assurance about the accuracy of estimates in epidemiologic surveys. Accuracy of the survey results seems to be enhanced considerably when special care is taken to guarantee confidentiality of responses, to protect the privacy of the survey respondents, and to develop trust and rapport before asking survey questions about sensitive behavior, alcohol and drug problems, or illegal activities. In particular, except in poorly conducted surveys of very young respondents, there seems to be very little exaggeration of drug involvement, and older adolescents and adults rarely report drug use unless it actually has happened. Moreover, the accuracy of the estimates does not seem to be distorted too much when the surveys concentrate on household residents and do not extend their samples to include homeless or imprisoned segments of the population. Even though homeless people and prisoners often have significant and special needs for alcohol- and other drug-dependent treatment services that society cannot ignore without peril, the number of homeless and incarcerated persons is small relative to the considerably larger number of persons living in households.

It also is important to note the relatively large size of the survey estimates obtained in these epidemiologic surveys. For example, in 1998, as part of the HIGH SCHOOL SENIOR SURVEY (Monitoring the Future), almost 16,000 high school seniors were asked to fill out confidential questionnaires about their use of such drugs as marijuana and cocaine; more than 38 percent reported having taken these drugs illegally, 80 percent reported consuming alcoholic beverages, and more than 60 percent reported having consumed alcohol to the point of getting drunk. In 1998, more than 25,500 American household residents aged 12 years and older participated in a U.S. government-sponsored NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE and were asked to answer an interviewer's questions about the use of these drugs; illegal drug taking was reported by an estimated 21 percent of those 12 to 17 years, 48 percent of those 18 to 25, 51 percent of those 26 to 34, and 32 percent of older adults. Furthermore, between 1990 and 1992, almost 9,000 Americans aged 15 to 54 completed confidential interviews as part of a U.S. government-sponsored National Comorbidity Survey. According to this survey, one in three tobacco smokers had tobacco problems, signs, and symptoms consistent with their having become dependent on tobacco and one in seven drinkers had alcohol problems, signs, and symptoms consistent with their having developed the clinical syndrome of alcohol dependence. Among those who reported use of marijuana, heroin, or other controlled substances, one in seven reported drug problems, signs, and symptoms consistent with their having become dependent on these drugs. These survey-based estimates are already high enough to provoke social concern. They would be even higher if corrections were to be made to account for respondents who were hesitant to report either their consumption of these drugs or the problems associated with drug use that they had.

DRUG-SPECIFIC ESTIMATES FOR THE U.S. POPULATION

It may be useful if, bearing in mind these potential limitations in the survey methods, one considers each broad drug class one by one, in order to convey the relative frequency of use of tobacco, alcohol, and other drugs in the United States, and to identify population subgroups within which drug use or drug dependence is most common. (From this point on, estimates based on the 1999 survey of high school seniors are labeled MF estimates; those from the 1998 National Household Survey on Drug Abuse are labeled NHSDA estimates; and those from the 1990–1992 National Comorbidity Survey are labeled NCS estimates.) In view of recent attention to the CAFFEINE-dependence syndrome and other health hazards of drinking COFFEE or TEA or consuming other caffeinated products, estimates concerning the use of caffeine and caffeine dependence might seem warranted. There is not yet a stable base of epidemiologic data on caffeine use and caffeine dependence, however; these remain topics that ought to be examined in future epidemiologic studies.

Tobacco Smoking in the Late 1990s. Monitoring the Future (MF) estimates show that about 65 percent of high school seniors have smoked TOBACCO cigarettes at least once. An estimated 35 percent of high school seniors smoked tobacco cigarettes at least once during the month

prior to the survey, and 23 percent had become daily tobacco smokers.

According to the National Household Survey on Drug Abuse (NHSDA), which included household residents age 12 years and older, an estimated 68 to 71 percent smoked tobacco cigarettes at least once, for a total of about 149,021,000 to 155,515,000 smokers. An estimated 29 to 32 percent had smoked in the year prior to the survey, for a total of 64,012,000 to 69,522,000 recently active smokers; most of these had smoked in the month prior to the survey (57,811,000–63,072,000).

There was an important age and sex-related variation in these estimates. For example, among adults past age 34, males were more likely than females to have been recent tobacco smokers (26.9% versus 23.4%). Among those 18 to 25, within the limits of survey error, there essentially were no differences between the sexes in prevalence of smoking, and both estimates were in a range from 37.5 to 45.3 percent. Among those 12 to 17, there also were no statistically reliable differences between the sexes, and the estimated proportions were between 17 and 21 percent; although estimates from earlier years show the proportion of girls smoking in this age group to be numerically greater than that for boys of the same age (NHSDA estimates).

Using data from the National Comorbidity Survey of Americans aged 15 to 54, it has been possible to estimate the proportion of tobacco smokers and other drug users who have developed drug-dependence syndromes, as defined in relation to a set of diagnostic criteria for drug dependence that were developed by the American Psychiatric Association in 1987. Before the diagnoses of drug dependence are made, the survey must produce evidence that drug users experienced signs or symptoms of dependence such as going through withdrawal or taking drugs to avoid withdrawal symptoms. Applied to the tobacco smokers identified in the Naitonal Comorbidity Survey, these diagnostic methods indicated that almost one-third of tobacco smokers in the survey population had developed tobacco dependence. That is, for every three tobacco smokers, one had developed tobacco dependence and was found to have met the American Psychiatric Association's diagnostic criteria for dependence on this drug. Of the more than 70 percent of respondents who had smoked tobacco at least once, a truly remarkable proportion of about 24 percent was found to have a history of currently active or former tobacco dependence (NCS estimates).

Smokeless Tobacco Use in the Late 1990s. An estimated 23 percent of high school seniors had tried smokeless tobacco at least once, and about 8.4 percent had used it during the month prior to the survey (MF estimates). Household survey estimates indicate somewhat lower values, except among males aged 18 to 25. For example, among 12- to 17-year-olds, an estimated 8.9 percent had tried smokeless tobacco, and just over 1 percent had used it in the month prior to the survey. By comparison, slightly more than 24 percent of 18- to 25-year-olds had tried smokeless tobacco; corresponding estimates for 26- to 34-year-olds and those over age 34 were 23.4 and 15.6 percent, respectively. Males aged 18 to 25 were also more likely to be recent smokeless tobacco users; more than 10 percent had used it during the month prior to the survey, while an additional 16 percent had used it at some time before the past month (NHSDA estimates).

Alcohol Use in the Late 1990s. An estimated 80 percent of high school seniors have consumed ALCOHOL at least once. About 74 percent had consumed alcoholic beverages in the year prior to the survey, and 51 percent had done so during the month prior to the survey. About 3.4 percent had become daily drinkers (MF estimates).

An estimated 62.3 percent of high school seniors had been drunk at least once—almost 53 percent during the year prior to the survey and almost 33 percent during the month prior to the survey. About 3.4 percent reported having become daily drinkers (MF estimates).

Among household residents aged 12 and older, an estimated 80 to 82 percent have consumed alcoholic beverages; this represents from 174,928,000 to 179,975,000 individuals. During the month prior to the survey, an estimated 51 percent had consumed alcohol. As might be expected, the prevalence values for 18- to 25-year-olds were somewhat higher than they were for the high school seniors, especially in relation to recent drinking: Almost 60 percent of the 18- to 25-year-olds had consumed alcoholic beverages during the month prior to the survey. The values for 12- to 17-year-olds were lower: About 37 percent in this age group had tried alcoholic beverages at least once, and about 19 percent had consumed alcohol during the month prior to the survey (NHSDA estimates).

An estimated 22.4 percent of respondents of all age groups from 12 years upward reported drinking at least once per week or more during the year prior to the survey. Corresponding estimates for respondents aged 12 to 17, 18 to 25, 26 to 34, and 35 + were 4.6, 24.5, 23.8, and 24.6 percent, respectively (NHSDA estimates).

Alcohol dependence was found to have affected 15 percent of those who had consumed alcoholic beverages: Out of every six or seven persons who had tried alcohol, about one had become dependent on alcohol. In relation to the total survey population that included drinkers as well as abstainers, an estimated 14 percent were found to qualify for the diagnosis of drug dependence, according to the American Psychiatric Association's criteria (NCS estimates).

Other Illicit Drug Use in the Late 1990s. When controlled substances such as MARIJUANA, cocaine, and heroin, as well as INHALANT drugs, were considered, it was found that an estimated 55 percent of respondents had used these drugs on at least once occasion, 42 percent during the year prior to the survey. About 26 percent had taken one or more of these drugs during the month prior to the survey (MF estimates).

The National Household Survey on Drug Abuse reported that an estimated 34 to 37 percent of the population aged 12 and older had engaged in illicit drug use at lease once: this amounts to about 75 to 81 million drug takers. The number of recently active drug takers was lower; they represented 6 to 7 percent of the population (NHSDA estimates).

According to the National Comorbidity Survey estimates, out of every seven persons who had tried marijuana, cocaine, or other controlled substances and inhalant drugs, one had developed drug dependence (14.7%). In light of the fact that about 51 percent of this survey population of 15- to 54-year-olds reported a history of illicit drug use, the resulting estimate for the prevalence of dependence on controlled substances was 7.5 percent. That is, in the total population of individuals (including both drug users and never users), about one in fourteen had fulfilled the criteria for drug dependence (NCS estimates).

Cannabis Use in the Late 1990s. An estimated 50 percent of high school seniors had tried marijuana or HASHISH (*Cannabis*) on at least one occasion, and about 38 percent had smoked cannabis during the year prior to the survey. An estimated 23 percent had smoked cannabis during the month prior to the survey, and an estimated 6 percent reported daily cannabis use (MF estimates).

Within the age ranges of 12 to 17 and among persons aged 35 and older, there are many individuals who have not yet started to use illicit drugs such as cannabis, as well as many others who never will start to use these drugs. As a result, one might expect lower prevalence values in these age groups as compared to the values for other age ranges. In fact, this is precisely what the national survey estimates indicate. Overall, an estimated 32 to 34 percent of respondents reported having tried cannabis, but among 12- to 17-year-olds the estimate was only 18.9 percent, and among those aged 35 years and older it was 29.4 percent. Prevalence of cannabis use was most common among 26- to 34-year-olds (47.9%) and among 18- to 25-year-olds (44.6%). This also was true for recent cannabis use during the month prior to the survey: There was a prevalence of 5.0 percent for the population overall, 8.3 percent for 12- to 17-year-olds, 13.8 percent for 18- to 25-year-olds, 5.5 percent for 26- to 34-year-olds, and 2.5 percent for older adults (NHSDA estimates).

Among cannabis users, about 9 percent were found to have developed cannabis dependence. Among *all* 15- to 54-year-olds (including both users and never users), 4.2 percent had become dependent on cannabis (NCS estimates).

Inhalant Use in the Late 1990s. INHALANTS had been used by an estimated 15 percent of high school seniors—about 6 percent within the year prior to the survey and about 2 percent during the month prior to the survey. Very few respondents (well under 1 percent) reported daily inhalant use (MF estimates).

The National Household Survey on Drug Abuse indicated that about 5.8 percent of its survey population had tried inhalants at least once; about 1 percent had done so during the year prior to the survey, and from 0.3 to 0.4 percent had used these drugs during the month prior to the survey. It was found, when considering age and sex, that the subgroup most likely to have used inhalant drugs during the month prior to the survey was that of males aged 18 to 25; in this group, 1.9 percent reported recently active inhalant use (NHSDA estimates).

An estimated 2.3 to 5.1 percent of the inhalant users have been found to qualify for the diagnosis

of dependence on inhalant drugs. Translated into an overall prevalence estimate for both users and nonusers, this amounts to about 0.3 percent prevalence of inhalant dependence in the total survey population (NCS estimates).

Use of Psychedelic Drugs in the Late 1990s. PSYCHEDELIC drugs (primarily LYSERGIC ACID DIETHYMIDE, or LSD) had been used by an estimated 14 percent of high school seniors. Almost two-thirds of these users (9.4%) had used them in the year prior to the survey, and about one-quarter (3.5%) had used them during the month prior to the survey. PHENCYCLIDINE (PCP) users were in the minority within this group of drug users; only 3.4 percent of the high school seniors had ever tried PCP (MF estimates).

Among persons aged 12 years and older, from 9.1 to 10.7 percent of individuals had tried psychedelic drugs such as LSD, but for the most part these drug experiences were not recent: Only 0.5 to 0.9 percent reported taking psychedelic drugs during the month prior to the survey. Peak prevalence values for recent use of the psychedelic drugs were observed in the years of adolescence and early adulthood; only for 12- to 17-year-olds and 18- to 25-year-olds did these values exceed a threshold of 1 percent (1.8 and 2.7%, respectively); otherwise, they were at the 0.4 percent level or lower (NHSDA estimates).

About 5 percent of the users of psychedelic drugs were found to qualify for the diagnosis of a dependence syndrome, defined in relation to the American Psychiatric Association criteria. Thus, about 0.5 percent of the survey population of 15- to 54-year-olds had become dependent on psychedelic drugs.

Cocaine Use in the Late 1990s. Among high school seniors, an estimated 9.8 percent had tried cocaine; within this group of COCAINE users, roughly one-half had tried CRACK-cocaine. About 6 percent of high school seniors had used cocaine (including crack) during the year prior to the survey, and just over 2.6 percent had used it in the month prior to the survey. In the MF sample of about 16,000 high school seniors, daily cocaine smoking was too rare to estimate precisely (MF estimates).

An estimated 10 to 11 percent of the National Household Survey's population reported having tried cocaine or crack smoking (or both) at least once. The corresponding value for 12- to 17-year-olds was only 2.2 percent, and there was age-related variation: 10.0 percent of the 18- to 25-year-olds had taken cocaine (including crack); 17.1 percent of the 26- to 34-year-olds had done so, and the prevalence estimate for older adults was 10.4 percent. Translated into absolute numbers, an estimated 21 to 25 million Americans aged 12 and older had tried cocaine or crack smoking. Recent use was substantially less common: Only 0.7 to 1.0 percent of the survey population reported having used these drugs during the month prior to the survey; this represented about 1.4 to 2.1 million recently active cocaine users in the survey population.

By the early 1990s, the second American epidemic of cocaine use had peaked and waned. Crack smoking had sustained the epidemic for a time, but in the early 1990s it became clear that crack smoking had not diffused broadly through the U.S. population. The relatively low prevalence values for crack smoking among high school seniors was reflected in the National Household Survey on Drug Abuse, which found that only 1.8 to 2.3 percent of its survey population had tried crack smoking; this amounted to 3.9 to 5.1 million individuals. The age groups with most crack-smoking experience were the 18- to 25-year-olds, with a prevalence value of 2.7 percent, and the 26- to 34-year-olds, with a prevalence value of 3.9 percent. Prevalence of crack smoking during the month prior to the 1998 survey was uniformly under 1 percent for all age and sex groups under study (NHSDA estimates).

For every six individuals who had tried cocaine at least once, one had developed cocaine dependence. That is, among these cocaine users, an estimated 15.2 to 18.2 percent had become sufficiently dependent upon cocaine to qualify for the American Psychiatric Association diagnosis. In relation to all persons in the survey population, whether they had tried cocaine or not, an estimated 2.7 percent qualified for the diagnosis of cocaine dependence (NCS estimates).

Use of Non-Cocaine Stimulants in the Late 1990s. The nonmedical use of stimulants other than cocaine (such as AMPHETAMINES) was actually more prevalent than cocaine use among high school seniors. An estimated 16.3 percent of high school seniors had taken these stimulant drugs without any doctor's orders; 10 percent had done so in the year prior to the survey, and 4.5 percent had done so during the month prior to the survey. Metham-

phetamine or "ice" smoking reemerged among youth in the 1990s. Among high school seniors, 4.8 percent had ever tried "ice," 1.9 percent had done so in the year prior to the survey, and 0.8 percent had used during the prior month (MF estimates).

For reasons not well understood, the Monitoring the Future sample of high school seniors yields prevalence estimates for non-cocaine stimulant usage that are considerably larger than corresponding estimates from the national household survey. Overall, the household survey population estimate for nonmedical use of these stimulant drugs was 4.4 percent, and the age group with the highest prevalence value was that made up of 26- to 34-year-olds, at 5.1 percent. Nonetheless, within the survey population, recent use of the stimulant drugs was found to be 3.2 to 4.9 percent for the 18- to 25-year-olds, the age group whose level of use most resembled that of the high school seniors (NHSDA estimates).

Slightly more than 11 percent of the persons who had used these stimulant drugs were found to have become dependent on them. This number of stimulant-dependence cases represents about 1.7 percent of all persons in the survey population aged 15 to 54 (NCS estimates).

Use of Anxiolytic, Sedative, and Hypnotic Drugs in the Late 1990s. About 9 percent of high school seniors had used tranquilizers (anxiolytic) or SEDATIVE-HYPNOTIC (e.g., BARBITURATE) drugs without a doctor's orders. About 5.8 percent had done so during the year prior to the survey, and 2.5 percent had done so during the month prior to the survey (MF estimates).

About 3 to 4 percent of the national household survey population reported nonmedical use of tranquilizers or anxiolytic drugs, while 2 to 3 percent reported nonmedical use of sedative-hypnotic drugs without a doctor's orders. For tranquilizers, this amounted to 6.8 to 8.8 millions of nonmedical users. For sedative-hypnotics, the total was 4.0 to 5.4 millions of nonmedical users. The estimated number of recently active users was less substantial; they represented less than 0.5 percent of the survey population for tranquilizers (under 1 million nonmedical users) and for the sedative-hypnotics (under 500,000 nonmedical users).

Grouping the users of the tranquilizer or anxiolytic drugs together with the users of the sedative and hypnotic drugs, the National Comorbidity Survey team found that about 9 percent of these drug users had become dependent on them. In considering this prevalence value, it is important to note that in this survey nonmedical drug use was defined to include not only use of the drug to get high, but also taking more of the drug than was prescribed or in ways not consistent with accepted medical practice. Overall, the prevalence of dependence on these drugs was at a level of 1.2 percent in the survey population (NCS estimates).

EPIDEMIOLOGY OF DRUG USE AND DRUG DEPENDENCE OUTSIDE THE UNITED STATES

Each year, the United States allocates more resources to epidemiologic surveys of drug use than does any other country in the world. For this reason, it has been possible to assemble a wealth of epidemiologic survey data on the prevalence of drug use and drug dependence within the United States. Other countries also have conducted surveys of this type and have produced valuable evidence about their experience with tobacco, alcohol, and other drugs. (See the bibliography for some references that can be consulted to gain more information about the results of these surveys.)

OTHER ASPECTS OF EPIDEMIOLOGY AS APPLIED TO DRUG USE AND DRUG DEPENDENCE

A broad range of research questions must be answered in order to gain a complete understanding of the epidemiology of drug use and drug dependence. The focus in this article has been on *quantity*: How many people in the population (or what proportion) have been affected by drug use and by drug dependence? Although many epidemiologists now devote their research careers to surveys that are needed to answer this kind of basic question, more stress ought to be placed on the other central questions for epidemiology, especially when the answers to these questions can guide society toward effective strategies for prevention of drug use and drug dependence. These questions are:

Where in the population are the affected cases located (in which subgroups, in which places, during which seasons, years, or epochs)? This is a question of *location*.

What accounts for some people becoming affected, whereas others do not become affected? This is a question about CAUSES.

By what processes or sequence of conditions do people become dependent on drugs? This is a question about *mechanisms* and linked sequences of causal conditions.

What can we do to prevent and reduce the suffering? This is a question about *prevention* and *amelioration*.

At its best, epidemiology provides critically important answers to each of these questions, and it works to ensure that new findings are translated rapidly into effective strategies for prevention. This is the future agenda for epidemiologic research on drug use and drug dependence.

(SEE ALSO: *Amphetamine Epidemics: Diagnosis of Drug Abuse; Diagnostic and Statistical Manual; Drug Abuse Warning Network; Epidemics of Drug Abuse; Social Costs of Alcohol and Drug Abuse; Vulnerability as Cause of Substance Abuse*)

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EQUANIL *See* Meprobamate

ETHANOL/ETHYL ALCOHOL *See* Alcohol: Chemistry and Pharmacology

ETHCHLORVYNOL This is a complex alcohol that causes depression of the central nervous system (CNS). It is a SEDATIVE-HYPNOTIC drug typically used on a short-term basis to treat insomnia and is prescribed and sold under the name Placidyl. Because of its depressant effects on the brain, it can impair the mental and/or physical abilities necessary to operate machinery, such as an automobile.

Continued use of ethchlorvynol can result in TOLERANCE AND PHYSICAL DEPENDENCE leading to abuse. Since the risk of abuse is not very great, it is included in Schedule IV of the CONTROLLED SUBSTANCES ACT. Withdrawal signs, not unlike those seen after ALCOHOL (ethanol) or BARBITURATES, occur upon termination of its use in addicts. Ethchlorvynol should never be combined with other CNS depressants, such as ethanol or barbiturates, because their depressant effects are additive. Because of their greater safety, the widespread use of BENZODIAZEPINES as sedative/hypnotics has largely supplanted the use of ethchlorvynol.

(SEE ALSO: *Withdrawal*)

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ETHINAMATE This is a short-acting Sedative-Hypnotic drug typically used to treat insomnia. It is prescribed and sold as Valmid. Structurally, it does not resemble the Barbiturates, but it shares many effects with this class of drugs; the depressant effects of ethinamate are, however, generally milder than those of most barbiturates. Continued and inappropriate use of ethinamate can lead to Tolerance and Physical Dependence, with withdrawal symptoms very similar to those of the barbiturates. Because of their greater safety, the widespread use of Benzodiazepines as sedative/hypnotics has largely supplanted the use of ethinamate.

(See also: *Withdrawal*)

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ETHNIC ISSUES AND CULTURAL RELEVANCE IN TREATMENT Differences exist among ethnic and cultural groups in their use—and abuse—of drugs and alcohol, as well as among risk factors that precede use and responses to treatment. Research suggests that an approach known as cultural congruency—when a patient and counselor share the same ethnic background or gender—can significantly improve the outcome of public health interventions and treatment. Drug and alcohol abuse treatment programs are no exception, and a number of recent studies have shown that careful attention to a special population's variant cultural framework can decrease recidivism and enhance treatment efficacy. The basic conceptual background for these tailored approaches begins with an examination of the cultural values held by the target community. Questions the treatment provider must ask when developing a targeted program include (Amodeo et al., 1997): At what point is the use of alcohol or other drugs considered a problem in this culture? At what point is a user deemed to require treatment? Who is perceived as owning this problem (e.g., the individual, the family, the community)? To what extent is any stigma attached to the problem? Are certain individuals more stigmatized (e.g., women)? This article will outline treatment approaches and considerations both general to the concept of cultural congruency and specific to some major ethnic groups.

ADDICTION: A MULTICULTURAL PROBLEM IN NEED OF MULTICULTURAL SOLUTIONS

Just as addiction is a global, rather than a national or regional, phenomenon, so addiction problems in the United States are multicultural. The whole fabric of successful treatment needs to be woven around cultural realities. In this society, twelve-step fellowships, such as Alcoholics Anonymous (AA), Narcotics Anonymous, and Cocaine Anonymous, are increasingly seen as the primary means to ensuring long-term abstinence and sobriety through addiction recovery.

Outside the United States there is strong professional resistance to both the Disease Concept and twelve-step recovery. In France, for example, where the *toxicomanes*, physicians dealing with chemical dependency, are heavily invested in a psychotherapeutic approach, there is professional denial that twelve-step programs exist or, if they do, are effective with French clients. Several *toxicomanes* maintained that even if they, themselves, championed twelve-step recovery and attempted to refer clients to such programs, the French, with their heritage of individual freedom and idiosyncratic behavior and beliefs, would never abridge their freedom by joining such fellowships as AA.

Health professionals in such wine-producing and -consuming countries as Italy, Spain, and France also express concern over the issue of addicts needing to abstain from all psychoactive sub-

stances. Wine, they maintain, is a food, and should not be included in such a blanket prohibition.

It has been suggested that twelve-step fellowships and their success provide credibility to addiction treatment as the bridge between active addiction and active recovery. While this may be increasingly true for the mainstream of white, European-American cultures, it may be less true for other cultures.

Countering the Perception That Twelve-Step Fellowships Have an Exclusively White, Male, Christian, Middle-Class Focus. From its beginnings, elements within the "group conscience" of AA began working to broaden the scope and flexibility of their fellowship. AA may have had its specific beginnings in the Christian Oxford Movement and the personal interaction between its cofounders, Bill W. and Doctor Bob, but its basic tenets reflect a spectrum of cultural antecedents. Throughout history and within various cultures, attempts have been made to deal with addiction and associated human problems. The most generally successful of these have involved in some way the development of individual spiritual maturity within a supportive environment. In that context, the Twelve Steps developed by AA and adapted by other twelve-step fellowships can be seen as a blueprint for developing spiritual maturity, which is similar in intent to the Buddhist Four Noble Truths and Eightfold Path, the Hindu *Vedas*, and the Zen Oxherding Panels.

Individuals with certain religious backgrounds may have particular problems relating to certain tenets of the Twelve Steps. Many Buddhists, for example, venerate the Buddha as a fully enlightened being to be followed and emulated, but do not see him as a "higher power." Not utilizing a concept of God or a higher power in their cultural background, they see their faith as a philosophy and a way of life rather than as a religion. Points of reference need to be established in order for twelve-step recovery to become meaningful for these individuals.

Culture and Spirituality in Twelve-Step Fellowships. While there are many meetings that have a distinct Christian orientation that goes far beyond joining hands and reciting the Lord's Prayer, there are many others that do not. Definitions of God and a "higher power" can and do include an open range of options. Essentially, a belief in God as represented in any particular religion is unnecessary for the workings of twelve-step recovery. However, belief in a power outside oneself that is capable of bringing one to sanity in terms of one's addiction is necessary, even if this power is characterized as the meeting group.

From a recovery standpoint, addiction can be seen as a disease of self-centered fear that depends on isolation and deeply held convictions regarding the nature and effects of the addicts' drugs of choice; that isolation renders the addict incapable of understanding the disease and its personal effects, which is the basis of denial. So long as the addict attempts to fight the addiction through personal willpower alone, he or she is fighting a losing battle, trapped in emotional gridlock in a state of "white knuckle sobriety," where increasing anxiety from the stress will inevitably result in relapse. The reason for this is that the convictions about use are buried within the individual's spiritual belief system, where they can be reached only if the addict is willing to accept that there is something outside his or her own immediate being that can lead him or her to sanity—a power higher than oneself.

Surrender and Powerlessness. The concept of surrender, given its many war-related connotations of occupation, rape, loss of freedom, and so on, is hard enough for anyone to accept, but it is particularly hard for cultural groups that have, over time, suffered more than their share of occupation, rape, loss of freedom, and so on. African-Americans and Native Americans, for example, may feel that they have been in a state of individual and cultural powerlessness for many generations, and have no desire for further surrender. Native Americans also have difficulties with that aspect of twelve-step recovery because it runs counter to tribal mores of self-reliance and stoicism. Adolescents, although their cultural cohesion is transitory, are in the process of developing their own individuality and are often loath to appear to be giving up something they have so recently gained. Muslims may have the least problem with the concept of surrender. "Islam" literally means "submission to God's will."

In explication, and to some degree expiation, of the term "surrender" as it is used in recovery, members of the community speak in such terms as "joining a winning team," and urge newcomers to "hang out with the winners." In admitting powerlessness over the disease, addicts are in effect gaining the power, through enlisting the support of their higher power and the fellowship itself, to be responsible for their own recovery. A misunder-

standing of this process can lead to an interpretation that people in twelve-step recovery are somehow "copping out" from personal responsibility. The point is that while the addict may not be responsible for having a disease that involves physiological and possibly genetic, psychological, and environmental components, in twelve-step fellowships the addict is most certainly responsible for his or her own recovery.

The African-American Extended Family Program is a good example of how the precepts of twelve-step recovery can be adapted to the needs a specific community. In it, African-American cultural mores and traditions are taken into consideration and made primary to recovery. Culturally, African-Americans strongly value communalism, or a collective identity (Longshore et al., 2000). In many treatment modalities targeted to African-American populations, drug addiction and use are related to slavery. For example, many African-Americans see methadone, a common treatment for opiate addictions, as a type of chemical slavery (Longshore et al., 1998). The HAIGHT ASHBURY FREE CLINICS, Inc. (HAFCI)/Glide Memorial Methodist Church African-American Extended Family Program (AAEFP), described in detail in Reverend Cecil William's book, *No Hiding Place*, represents an important collaboration that has made possible an effective intervention in the inner-city crisis of CRACK-cocaine use.

The key to this intervention has been the adaptation of TWELVE-STEP principals of supported recovery to the AFRICAN-AMERICAN inner-city culture. In the HAFCI/Glide program, the basic practicalities of recovery are utilized in a model that is uniquely meaningful in terms of the African-American experience.

The "Big Book" of ALCOHOLICS ANONYMOUS uses the terms "spiritual experience" and "spiritual awakening," manifesting in many different forms, to describe what happens to bring about a personality change sufficient to induce recovery. While some of these may involve an "immediate and overwhelming God consciousness," most are what William James called an "educational variety" of revelation, developing slowly over time. According to a "Big Book" appendix titled "Spiritual Experience," the core of this process is the tapping of an "unexpected inner resource" by members who identify this resource with "their own conception of a Power greater than themselves."

Many members of the African-American community afflicted with crack-cocaine addiction have been raised in the church. There is a tradition of revelation; many who have been "saved" now believe they are sinners because they have used and sold crack-cocaine to their own people. God has been described in a strict denominational sense. Spiritual awakening in a recovery model within a church program may produce conflict with traditional religious definitions, particularly the third step: "Made a decision to turn our will and our lives over to the care of God *as we understood him*." Religious leaders, such as Reverend Williams, have played a role in presenting a model of recovery theology that helps mobilize the church as a sleeping giant to better respond to the nation's drug epidemic. In his model, Williams employs self-definition within a spirituality of recovery.

In keeping with the IBCA's African-American cultural approach, it was generally agreed that the best site for the new program would be a church. In a Glide conference panel debate on religion and spirituality, Richard Seymour pointed out that under the best of conditions, religion equals spirituality plus culture. This is particularly true in the African-American community, within which the church provides a point of cohesion and a center for both spiritual and community values and, thus, a common ground for positive community activity. For a number of reasons, the clear choice was Glide Memorial Methodist Church in San Francisco's Tenderloin, a neighborhood that, though it includes a number of ethnic minorities, is predominantly African-American, low-income, and hard hit by the onslaught of dealing and abuse of crack-cocaine.

Under the leadership of Reverend Williams, Glide had been providing services for indigent and homeless residents, including addicts, for 25 years. Because of his growing concern over the crack-cocaine problem, Reverend Williams and his wife, Jan Mirikitani, executive director of Glide, attended a twelve-step recovery conference conducted by David Smith and Millicent Buxton. Following this conference, they decided to develop a culturally specific recovery program at Glide Church because of the resistance of people of color to participating in the twelve-step process.

Specific problems of the African-American target population as identified by various studies

(HAFCI, 1990; Jackson, 1995; Longshore et al., 1998 and 2000) include the following:

- Low self-esteem
- Late introduction into recovery
- Focus on short-term abstinence rather than long-term recovery
- Dialect of African-Americans
- Institutionalized racism
- Internalized racism
- A unique, often dysfunctional family structure: many classical African cultures have been matrilineal, and look to the "grandmother" for spiritual direction and values. African-Americans developed a matriarchal family structure to survive during slavery, but this structure has proved unable to address problems of alcohol and other drug addictions. America is based on a patriarchal family structure, the opposite of the African-American model. It is therefore difficult for African-Americans to relate to systems and to address dysfunctional families when their model is not the norm. The most extreme injury is seen in children being taken from mothers by the system.
- Women's meetings: For those who have lost children, the comparison between now and the capture of children in Africa during the slave trade is made. Particular emphasis is placed upon the role of women in the more matriarchal African-American family. For many the most positive role model is a grandmother who passed on the traditions of the family and represents a "higher power."

The first and foremost priority is bringing to intervention and recovery an approach and nature that members of a target culture can identify and live with. Culturally responsive activities need to be identified and developed. Most research to date has been conducted with African-American populations, but the treatment models developed in conjunction with these studies can be transliterated to other ethnic and cultural populations.

Implicit within these modalities is the recognition that treatment is more than the prescribing of medication or the providing of basic and generic counseling based on a homogeneous model of what constitutes addictive disease.

Does establishing culturally congruent treatment produce results? Another example of a treatment intervention designed to be congruent with the cultural values and mores of the group process is the Engagement Project developed by Longshore et al., which is used for the purposes of scientific measurement of the effects of cultural congruency. Treatment began with a traditional African-American meal of fried chicken, ribs, greens, potatoes, and red beans and rice, to establish a culturally-specific framework for the intervention. The participant shared this meal with a counselor and a former drug user, called a "peer." This group then together watched a video featuring still photos, footage, and clips from commercial films about African-Americans. The third and final phase of the intervention consisted of a counseling session to review the participant's commitment to recovery. By situating drug abuse as both an individual problem and a community problem seated in power inequalities between the African-American community and dominant institutions, the intervention proved statistically effective in terms of participants reporting being drug abstinent one year afterwards.

Cultural Characteristics of Other Ethnic Groups. *Asian Americans.* Asian Americans have been traditionally treated as a conglomerate group, a "model minority" whose drug problems have often been overlooked (Nemoto, 1999). However, this is patently not the case, as Japanese Americans, Filipinos, Vietnamese Americans, and Chinese Americans all come from differing cultural backgrounds and retain variant attitudes toward substance abuse, illness, and disease. Some cultural constructs that are shared among most Asian Americans regarding the use of drugs and alcohol are a fear of addiction, fear of injecting drugs, and a strong stigma attached to drug users in the community (Nemoto, 1999). Immigrant Asian Americans are more likely to use drugs than American-born people of Asian descent (Nemoto, 1999); such cultural factors often inform a user's response to treatment. It is necessary that treatment providers be not just bilingual, but also bicultural, in the sense that they are equipped to understand the unique family structure and pressures present in Asian American culture.

Native Americans. The traditionally tribal orientation of Native American society is in stark contrast to dominant institutional norms. For many Native Americans, an effective approach to the treatment of drug and alcohol problems involves a strong

spiritual component. A 1998 report by Christine T. Lowery asserts that four broad concepts comprise an intellectual understanding of "healing the spirit" for Native Americans. These concepts, addressing the concepts of spiritual health and wellness, are:

- Balance and wellness.
- The colonization experience and addiction as a crisis of spirit.
- Issues of abuse (including sexual abuse).
- A time of healing.

Careful consideration of these principles illustrates the unique spiritual perspective Native Americans have on addiction and recovery. The intersection of the concepts outlined above should be the focus for intervention in these communities. For Native Americans, healing is traditionally a multidimensional, spiritual, relational, and intergenerational endeavor.

Hispanic Americans. Studies indicate that there is a positive correlation between length of time in the United States and drug usage among Hispanic Americans (Ma et al., 2000). Moreover, degree of acculturation and immigration status may affect treatment-seeking behaviors (Amodeo, 1997). An illegal immigrant is less likely to seek drug or alcohol treatment intervention because of the perceived threat of deportation.

Acceptance of disease-concept-related treatment and recovery outside the United States has differed from culture to culture, from country to country, in some cases from community to community. In Scandinavia, for a studied example, Finland, Iceland, and SWEDEN have experienced phenomenal multiplication of AA groups since the 1970s, whereas Denmark and Norway have experienced a decline in groups over the same period. With the advent of glasnost, narcologists in the former Soviet Union discovered AA. Since that time, treatment has been increasingly linked with recovery in Russia and other republics.

Overcoming Points of Resistance and Concern. The distance between cultures may seem like a chasm at times, but it is being bridged by such projects as the AAEFP that provide both recovery and a means to developing cultural parity. Society is changing rapidly, and fortunately, recovery has the flexibility to change along with it. Many groups within AA have learned that if there is no meeting that fits their special need, they can form their own meetings. The challenge is to adapt the process of treatment and recovery to all cultures and races, to counter stereotypes that recovery works only with certain groups.

(SEE ALSO: *Chinese Americans, Alcohol and Drug Use among; Ethnicity and Drugs; Hispanics and Drug Use; Rational Recovery; Sobriety; Treatment; Women and Substance Abuse*)

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ETHNICITY AND DRUGS In national statistics for the United States, it is common to see information about different segments of the population. For example, data from the U.S. Census and many national surveys on drug use often are subdivided in relation to four racial groups: (1) white, (2) black, (3) Asian or Pacific Islander, (4) American Indian or Alaska native. In concept, "racial heritage" refers to biologically inherited origins, but most people appreciate that these categories of race are determined more by social ideas and customs than by sharp genetic distinctions among these four groups. Some people even change their racial affiliation as they change their social perceptions.

In some national statistics and survey data, it also is common to see subdivisions in relation to "ethnic heritage," which sometimes refers to a person's country of origin but more generally refers to shared social and cultural characteristics. For example, people with recent or distant family origins in Spain or Portugal, or former colonies of Spain and Portugal (e.g., Mexico, Brazil), are called Iberian, Hispanic, or Latino; in North American statistics, it has been typical to subdivide the racial groups in relation to ethnicity as well: (1) White-not Hispanic, (2) Black-not Hispanic, (3) White-Hispanic, (4) Black-Hispanic, and so on. Here, too, the designation of Hispanic or Latino refers more to a social characteristic than to a specific family-genetic background. For example, American Indians from Mexico may be classified as Hispanic-American on the basis of their Mexican ancestry or as Native American on the basis of their North American Indian ancestry. The utility of these classifications of ethnicity and ethnic heritage depends on the degree to which they reflect sameness of social customs and learned behavior. People who are being compared within different ethnic groups ought to exhibit similarities in social customs and learned behaviors, and sometimes a shared sense of affiliation with that particular group. People across different ethnic groups ought to demonstrate more variation in social customs and learned behaviors than are to be seen among people within these groups.

There are many reasons for national reports to present statistical data on the population classified in relation to racial and ethnic heritage. Anyone reading historical documents for the period during and preceding the nineteenth century will find it difficult to escape a conclusion that these classifications were motivated in part by prejudice and racist thinking. Since the nineteenth century—from the earliest days of the U.S. Census—government officials have been interested in knowing the ethnic origins, as well as the size, of different racial and ethnic groups within the population for various policy and planning purposes.

Despite their somewhat questionable origins and uses, racial and ethnic classifications are important measures of social and historical phenomena in the United States. For example, in the area of public health, when national statistics on liver cirrhosis

are examined, it can be seen that Americans who describe themselves as African-American are more likely to develop liver cirrhosis compared with Americans of predominantly European heritage. This type of information can guide public health action directed at preventing and treating liver cirrhosis. It is a help in targeting early detection and intervention efforts intended to reduce the suffering associated with liver cirrhosis. It may help identify specific environmental conditions such as poor nutrition or infectious diseases that might account for the higher risk of liver cirrhosis in the African-American segment of the population.

National statistics on alcohol and other drug use in relation to racial and ethnic heritage also have helped the nation's policymakers to see that some segments of the population have a greater need than others for alcohol and drug treatment and prevention services. Through block grants and other funding mechanisms, the federal, state, and local governments can provide support for services that target the special population groups with more needs for these services.

Although statistics on ALCOHOL and other drug use in relation to race and ethnic heritage can be used for the benefit of the population, it must be said that this topic has been understudied and the evidence often misrepresented. On the one hand, the topic is understudied in the sense that differences can be observed in alcohol and other drug use across racial and ethnic subgroups of the population, but it is not known whether they are due to differences in inherited predispositions or to other differences. On the other hand, the evidence of racial and ethnic differences in alcohol and drug use can be misrepresented and interpreted prejudicially as data showing one group to be inferior to another.

The complicated nature of this topic can be illustrated by considering liver cirrhosis among African Americans in the United States. In part, the occurrence of liver cirrhosis is determined by long-term heavy drinking of alcoholic beverages, but liver cirrhosis is also caused by prior infections or by autoimmune reactions, and vulnerability to alcohol-related liver cirrhosis is also influenced by cofactors such as poor nutrition. In the United States, African Americans historically have been at great social disadvantage. On average, they are not as wealthy as other Americans, and, in addition, they more often live in poverty, with associated poor nutrition, underutilization of health care services, and compromised health status. Hence, it might be these socioeconomically related conditions that account for the excess occurrence of liver cirrhosis among African Americans rather than any inherited characteristics or personal characteristics related to drinking.

Within the United States, many other racial and ethnic minority groups also live with social disadvantages similar to those endured by African Americans. For this reason, it is easy to misinterpret national statistics on alcohol and drug use among racial and ethnic minority groups if they are taken strictly at face value. Instead, one must look beneath the surface and ask whether social or economic conditions might account for the statistics.

While studying racial and ethnic differences in CRACK smoking and other COCAINE use, some public health scientists have attempted to hold constant the social and neighborhood conditions that also could explain these differences. Once social and neighborhood characteristics had been taken into account, these studies found very little evidence to support the idea that African Americans or Hispanics were more likely to smoke crack or to take cocaine.

Although the importance of social and environmental influences in people's use of alcohol and other drugs has been clearly illustrated, it is important to keep in mind that biological factors may also play a role in determining one's preference for alcohol or particular drugs. For example, Asian Americans, as a group, consume less alcohol than any of the other racial or ethnic groups. Their lower drinking rates have been attributed, in part, to the fact that a majority of Asians possess a particular form of an alcohol-metabolizing enzyme whose action results in unpleasant side effects after drinking alcohol.

It also is interesting to find variation *within* large racial and ethnic groups, because this draws attention to the fact that not all African Americans are alike, nor all Hispanic Americans, Native Americans, Asians, or Pacific Islanders. For example, studying occurrence of alcohol abuse and dependence in different countries of Asia, epidemiologists found that men in South Korea had an extremely high prevalence of these conditions but men in Taiwan an extremely low prevalence. In addition, epidemiologists found more crack smoking among Hispanic Americans whose behavior showed that

they had become acculturated to mainstream customs (e.g., by choosing to speak English rather than Spanish) and less crack smoking among other Hispanic Americans (e.g., those who chose to speak Spanish rather than English). This relationship was more pronounced among Hispanic Americans from Mexico than among those from Cuba, however, and this is an additional indication of variation within the large and growing Hispanic segment of the U.S. population.

Studies conducted on alcohol and other drug use by Native Americans provide another example of the variation that can exist within a large racial group. For instance, there is considerable variation in alcohol and other drug experiences from tribe to tribe, from one part of the country to another, and even from one residential location to another (e.g., boarding school students versus other young people). It becomes difficult, therefore, to summarize the alcohol and drug experiences of Native Americans in a few sentences. For many Native-American young people and adults living in urban environments, and sometimes on reservation lands as well, the use of alcoholic beverages and also INHALANT drugs is associated with several social and health problems. Researchers have speculated that the disintegration of Native-American culture has contributed to high rates of STRESS and that this in turn is related to a disproportionately high use of alcohol among this segment of the American population. These statistics alerted the attention of public health workers and government officials, and through their efforts many programs have been initiated to draw Native Americans with alcohol abuse problems into treatment.

Racial and ethnic patterns of alcohol and other drug use and related problems vary by age, gender, and drug. National surveys of high school seniors conducted since the early 1970s, and more recent surveys that included eighth- and tenth-graders, reveal that some minority youth use less alcohol and other drugs than Caucasian youth. Specifically, Caucasians, Native Americans, and Mexican Americans have the highest frequency of reported alcohol use whereas African Americans and Asian Americans have the lowest. Because these surveys include only in-school youth and not children who have dropped out of school, it may be that the true proportions of alcohol and other drug use have been underestimated.

In general, males report using drugs more frequently than females, and this gender difference cuts across racial and ethnic boundaries. For example, African-American males and Caucasian males are more likely than African-American and Caucasian females to use alcohol. It is also true that people in different age groups vary in relation to their reports of using alcohol and other drugs. When researchers carefully divide different racial and ethnic groups by age, some interesting trends in alcohol-use patterns appear. For Caucasian adults, drinking tends to increase until mid to late life, with older people drinking less as a group than younger adults. African Americans, however, tend to be heavier drinkers later in life and to exhibit more alcohol-related health problems (e.g., cirrhosis, esophageal cancer). For some drugs other than alcohol, a similar picture exists. For example, Caucasians and Hispanic Americans report using cocaine earlier in life whereas African Americans report using it later in life. Cigarette SMOKING is more common among young Caucasians (12–17 years old) than it is among Hispanic Americans or African Americans of the same age; however, a higher proportion of the latter groups report smoking later in life.

It is sometimes difficult to interpret findings that point to differences in drug use between minority and nonminority subgroups within the U.S. population. It must be kept in mind that socially shared environmental conditions (e.g., availability of drugs, neighborhood conditions, economic resources) rather than race or ethnic identity may be underlying patterns of drug use. Other factors such as social status and community norms for coping with life stresses may account for reported racial or ethnic differences in drug use.

Continued research is needed to track patterns of alcohol and other drug use in the population and to find out the mechanisms or the reasons that put some groups at higher risk than others for problematic involvement with alcohol and other drugs. Some of the most current information is limited. For instance, minority intravenous drug users are known to have higher rates of exposure to HIV than Caucasian drug users, but no clear explanation for this observation has been determined. Perhaps learning more about barriers to obtaining treatment for intravenous drug use in certain minority populations will contribute to an understanding of this problem.

Researchers, as well as policymakers, need to be culturally sensitive; that is, they must appreciate the social, cultural, and economic conditions that underlie racial and ethnic differences in alcohol and drug use. It is important to realize that racial and ethnic identification can serve as a source of strength to those who design targeted prevention and intervention programs for certain segments of the population.

(SEE ALSO: *Asia, Drug Use in: Causes of Substance Abuse; Chinese Americans, Drug and Alcohol Use among; Epidemiology of Drug Abuse; Ethnic Issues and Cultural Relevance in Treatment; Families and Drug Use; Injecting Drug Users and HIV; Poverty and Drug Use; Vulnerability as Cause of Drug Abuse; Women and Substance Abuse*)

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ETHNOPHARMACOLOGY This branch of pharmacology studies the use and lore of drugs that have been discovered and developed by sociocultural (or ethnic) groups. It involves the direct observation and report of interactions between the societies and the drugs they have found in their natural environments and the customs that have evolved around such drugs, whether ceremonial, therapeutic, or other. These drugs, usually found in plants (hence similar study by ethnobotanists as well as ethnologists), are described—as are their effects within the customs, beliefs, and histories of a traditional culture or a specific society.

Examples include descriptions of the use of coca leaves (*Erythroxylon coca*) by indigenous populations of Colombia and Peru, for increased strength and endurance in high altitudes; the ceremonial use of PEYOTE (*Lophophora sp.*) by Native Americans of the Southwest and Mexico; and the use of KAVA (*Piper methysticum*) in ceremonial drinks by the indigenous populations of many South Pacific islands.

(SEE ALSO: *Asia, Drug Use in; Dover's Powder; Plants, Drugs from*)

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ETHYL ETHER *See* Inhalants

EUROPE AS TRANSIT AREA FOR ILLICIT DRUGS *See* International Drug Supply Systems

EXCLUSIONARY RULE In legal proceedings, the exclusionary rule prohibits the use of any evidence obtained in contravention of the U.S. Constitution. The rule is frequently invoked when government authorities seize evidence in violation of the Fourth Amendment's prohibition against unlawful searches and seizures. Evidence may be illegally obtained when government officials do not have a warrant to search an individual's premises or the warrant is defective. Law enforcement officers may also lack sufficient probable cause to arrest a person. In addition, the courts may invoke the exclusionary rule when they find a violation of an individual's Fifth Amendment right against self-incrimination or a violation of a defendant's Sixth Amendment right to counsel. Courts often refer to evidence obtained in violation of the Fourth, Fifth, or Sixth Amendment as "tainted" or "the fruit of a poisonous tree."

The U.S. Supreme Court established the exclusionary rule in the early 1900s. It applies to all federal courts through the Fourth Amendment and to all state courts through the Due Process Clause of the Fourteenth Amendment. Before the rule was created, any evidence was admissible in a criminal trial if the judge found it relevant. It made no difference how the police had obtained it. In *Weeks v. United States*, 232 U.S. 383, 34 S.Ct. 341, 58 L.Ed. 652 (1914), the Supreme Court barred the use of evidence secured through a warrantless search of a defendant's house by federal agents. However, for almost 50 years the exclusionary rule only applied to federal courts.

The Supreme Court broadened the rule's coverage in *Mapp v. Ohio*, 367 U.S. 643, 81 S.Ct. 1684, 6 L.Ed.2d 1081 (1961). It held that the due process clause of the Fourteenth Amendment requires states to exclude evidence obtained from an unconstitutional search or seizure. The Court has often cited an individual's right to privacy and the deterrence of unreasonable police conduct as the primary reasons for excluding evidence obtained from an unreasonable search and seizure.

A criminal defendant who claims an unreasonable search and seizure is usually allowed to make the claims in a suppression hearing that is conducted before the trial. At this hearing the judge must determine what evidence will be suppressed, or excluded from trial.

A number of exceptions to the exclusionary rule have emerged to reduce the effects of the doctrine, such as a police officer's "good-faith" belief that an otherwise defective warrant is valid, evidence obtained in "hot pursuit," or evidence seized in "plain view" of the law enforcement officer's sight and reach. There are other exceptions to the exclusionary rule. Evidence seized by private parties is not excluded from trial if the search was not at the direction of law enforcement officers. If a criminal defendant testifies in his or her own defense, illegally seized evidence may be used to discredit the defendant's testimony. Illegally seized evidence can also be used in grand jury proceedings and civil proceedings. However, a grand jury cannot use illegally seized evidence if it was obtained in violation of federal wiretapping statutes.

IMPORTANCE IN DRUG CASES AND ENFORCEMENT

The exclusionary rule prohibits the introduction of constitutionally tainted evidence. The effect of the doctrine has often been the exclusion of evidence that might be used to convict a suspected drug trafficker or abuser. Courts have excluded evidence of drug PARAPHERNALIA or supplies illegally seized, admissions obtained by coercion or without notifying the party of the right to remain silent, and evidence obtained in violation of a defendant's Sixth Amendment right to counsel, such as a lineup identification. The Supreme Court has determined that it is preferable to allow a drug trafficker to go free than to permit law enforcement officers to violate a citizen's constitutionally protected rights.

Two recent Supreme Court cases illustrate the polarities in Fourth Amendment exclusionary rule cases. In *Minnesota v. Carter*, 525 U.S. 83, 119 S.Ct. 469, 142 L.Ed.2d 373(1998), the Court had to balance law enforcement and privacy interests in assessing the reasonableness of a drug search and seizure. The key issue was whether a police officer who looked in an apartment window through a gap in a closed window blind violated the privacy of the drug dealers in the apartment because they had an expectation of privacy that is protected by the Fourth Amendment. The Supreme Court held that the police officer did not violate the Fourth Amendment because the occupants of the apartment did not have an expectation of privacy. Therefore, the drugs that the police officers saw and later seized did not have to be excluded from evidence.

The outcome was much different in *Bond v. U.S.*, ___U.S. ___, 120 S.Ct. 1462, 146 L.Ed.2d 365(2000). In this case, the Court ruled that police cannot squeeze the luggage of bus passengers to try to find illegal drugs. The U.S. Border patrol routinely squeezed carry-on luggage of bus passengers traveling near the Texas-Mexico border. Border patrol officers discovered a brick of methamphetamine after feeling the defendant's soft-sided bag. The Supreme Court noted that the Fourth Amendment provides that a person's "effects" are protected from unreasonable searches and seizures. A traveler's piece of luggage was clearly an "effect" protected by the amendment. It found that a "bus passenger clearly expects that his bag may be handled. He does not expect that other passengers or bus employees will, as a matter of course, feel the bag in an exploratory manner." Because the agent did manipulate the bag, he violated the Fourth Amendment. In addition, the Court ruled that the defendant's expectation of privacy was reasonable. It distinguished prior rulings that defeated exclusionary rule challenges because they were based on visual inspections, not tactile inspections.

(SEE ALSO: *Drug Laws: Prosecution of; Seizures of Drugs*)

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EXECUTIVE OFFICE OF THE PRESIDENT *See* U.S. Government: The Organization of U.S. Drug Policy

EXISTENTIAL MODELS OF ADDICTION *See* Values and Beliefs: Existential Models of Addiction

EXPECTANCIES The beliefs a person has about the effects a drug will have are called *expectancies*. The study of expectancies began with the employment of the experimental balanced-placebo design in alcohol research in the early 1970s (see Marlatt & Rohsenow, 1980, for a review). Research on people ranging from light drinkers to inpatient alcoholics revealed that expectancies are predictive of some of the behaviors exhibited when people use a drug. These studies revealed that both the beliefs an individual has—about whether a drink contains ALCOHOL and the specific outcomes that individual expects from consuming alcohol—are in many cases more predictive of subsequent behavior than the pharmacological effects of the drug.

EXAMPLES OF RESEARCH STUDY

An example of research using balanced-placebo design is as follows: In a simulated bar setting, half the participants in a study are told they will receive a drink containing vodka and tonic, and half are told they will receive a drink containing only tonic. After this expectation is established, half of each group does receive vodka and tonic, while the other half receives only tonic, resulting in four groups: (1) those who expect vodka and tonic and receive vodka and tonic, (2) those who expect vodka and tonic and receive only tonic, (3) those who expect tonic and receive vodka and tonic, and (4) those who expect tonic and receive tonic. Thus, some of the people who expect alcohol receive only tonic, and some who expect only tonic receive a mix containing alcohol.

Behavioral observations following this manipulation reveal that the most powerful predictor of behavior after consuming the assigned drink is not whether the person actually receives alcohol, but whether that person *believes* he or she is drinking alcohol: People who expect alcohol in this experimental situation consume significantly more drink than those who are not expecting alcohol, regardless of whether or not they do receive alcohol in their drink. With the discovery of this phenomenon, even in people who are considered dependent on alcohol, this finding has been interpreted as providing contrasting evidence to the disease model's notion that "loss of control" is caused exclusively by the pharmacological effects of alcohol; the findings introduced the idea that cognitive factors are influential in a person's drug-related behavior. The presence of expectancy effects have also been identified in research on drugs other than alcohol, including TOBACCO and MARIJUANA (Marlatt & Gordon, 1985).

Most of the research on expectancies during the 1970s and 1980s was conducted on college students, with samples ranging from light to heavy social drinkers who were primarily Caucasian. This research has shown that the effect of a person's expectancies depends on whether the behavior involved is socially mediated: Stronger expectancy effects are found for social behaviors (e.g., aggression or sexual arousal) than for nonsocial behaviors (e.g., beliefs concerning motor coordination or memory skills); they are stronger for outcomes that are perceived as positive (e.g., sexual arousal) than as negative (e.g., poor motor coordination).

For socially mediated behaviors, expectancy research has revealed that college students of both sexes show less anxiety in social situations if they believe they have consumed alcohol. In addition, males show heightened sexual arousal when exposed to an erotic environment if they believe they have consumed alcohol (Marlatt & Gordon, 1985). Men and women of college age have also both been found to respond more aggressively when provoked after they believe they have consumed alcohol. Sex differences have been found on the effects of alcohol on anxiety with persons of the opposite sex: Women of college age have shown more anxiety in the company of an unfamiliar man when they believe they have consumed alcohol, while men of college age have shown reduced anxiety when in the company of an unfamiliar female. The results have been interpreted as reflecting gender differences regarding the acceptability of alcohol in social situations with a stranger of the opposite sex.

OTHER STUDIES

Other experimental work has revealed that specific outcomes can vary with the personal beliefs an individual holds regarding alcohol and with the phase of intoxication of an individual (Southwick et al., 1981). Overall, the results based on expectancy research point to the likelihood that people may have established cultural beliefs regarding the effects of alcohol in social situations and that these beliefs play some role in the behavioral effects of alcohol.

Research has also found that expectancies do predict drinking behavior over a one-year period for early adolescents (Christiansen et al., 1989); that expectancies tend to crystallize in people at a young age and that they tend to be resistant to change (Miller, Smith, & Goldman, 1990). Other studies on Caucasian adolescents and young adults have found that those who have mostly positive and only few negative outcome expectancies tend to experience more alcohol-related problems than those whose outcome expectancies are more evenly divided between positive and negative effects (Brown, Christiansen, & Goldman, 1987).

Since the late 1980s, researchers have begun to examine ethnic and racial differences in the expectancy variable. One study of college-age students (Daisy, 1989) revealed that Native-American students had significantly stronger expectancies for the positive social and physical effects of drinking than did Asian-American students. Caucasian students were found to have stronger positive expectancies for social and physical effects than did Asian-American students, but less than did Native-American students. These beliefs concerning the effects of alcohol were also found to be highly associated with the drinking patterns of the study participants: those people whose drinking pattern was considered heavy had stronger beliefs in the above expectancies than individuals who drank less. The study strongly suggests that ethnic differences exist in alcohol-related expectancies, and it confirms that expectancies are related to the amount of alcohol consumed.

The association between expectancies and drinking pattern has been consistent in the research and has therefore become targeted in substance-abuse treatment. Expectancies have been found to influence the way a person copes with high-risk situations after treatment aimed at abstinence (Marlatt & Gordon, 1985; Condiotte & Lichtenstein, 1981). In RELAPSE PREVENTION, positive-outcome expectancies are viewed as the source of urges or cravings for a substance. Treatment according to this perspective therefore includes changing a client's outcome expectancies: If a person believes that drinking will provide immediate relief from stress, then treatment focuses on helping that person consider the long-range implications of drinking—helping the person by adding the negative outcomes of drinking to the anticipated positive results of drinking—and thereby changing the composition of the person's outcome expectancies.

Self-efficacy expectancies, or how effectively one feels he or she can cope with a high-risk situation, are also examined in treatment. If a client lives a stressful lifestyle and believes that only alcohol provides relief from that stress, the therapist helps the client develop and utilize alternative methods for coping with stress. For example, clients can be taught to look forward to meditation or exercise or other positive-reward situations to help cope with stress and to reduce urges and the resulting temptation to drink. Treatment focuses on developing alternative coping strategies for a client's individual high-risk situations, and therefore includes an ongoing assessment of each client's high-risk situations.

Self-efficacy differs from overall motivation to quit or reduce substance use, since perceived control will vary across situations. In research on relapse prevention, self-efficacy has been found to be predictive of the first use of the substance after abstinence-based treatment: Those people who do not believe they can cope with either a specific situation or cope, in general, with the temptation to use a substance are more likely to relapse in the face of a high-risk situation than are people who believe that they are able to maintain their goal of abstinence in the same situation (Condiotte & Lichtenstein, 1981).

The Alcohol Expectancy Questionnaire (AEQ), developed in the late 1980s, became the most commonly used alcohol expectancy instrument. Criticisms of the AEQ led to a conceptual model of drinking expectancy grounded in social learning theory. In this model, people acquire a set of alcohol expectancies regarding how alcohol will affect them during what is called the acquisition phase of the model. The behavioral outcomes of these beliefs

were then hypothesized to be regulated by a process involving Drinking Refusal Self-Efficacy (DRSE).

In 1996, the Drinking Expectancy Profile (DEP) was developed, which had two interrelated subtests, the Drinking Expectancy Questionnaire (DEQ) and the Drinking Refusal Self-Efficacy Questionnaire (DRSEQ). When compared to the AEQ in a study, the DEP showed better predictive ability on the Alcohol Dependence Scale and for quantity of drinking and frequency of drinking in a student sample. Furthermore, the DEQ contained both negative and positive outcome expectancies, which yielded better information on alcohol-related outcomes.

Further research in the 1990s showed that alcohol expectancies can develop independently of the actual drinking experience, developing from vicarious learning before even tasting alcohol. Actual drinking behavior could later reinforce or modify the existing beliefs. Drinking refusal self-efficacy beliefs were also show to develop prior to one's drinking history.

A study in 2000 considered the 1992 Temptation Restraint Inventory (TRI) and DEP as indicators of problem drinking across a range of drinking parameters. It yielded a more comprehensive picture of the complex interrelationship between the variables that make up the individual drinker's motivation for risky and dependent drinking. The results showed that drinking restraint and related control and impaired-control issues were the strongest predictors of alcohol problems. Alcohol expectancies and drinking refusal self-efficacy, while reflecting some of these loss-of-control factors, tended to focus more on choices of whether to drink or not and thus predicted more frequent usage of alcohol. This study suggested that restraint, alcohol expectancy, and self-efficacy measured different cognitive domains (Connor, et al, 2000).

Another study in 2000 looked at psychosocial and behavioral factors as predictors of heavy drinking among adolescents and assessed students' expectancies about drinking. The study found that boys who reported positive drinking expectancies were over seven times more likely to become heavy drinkers than boys who had negative drinking expectancies. In fact, positive alcohol expectancy was the single strongest predictor of later heavy drinking among boys. However, the expectancy variables were not associated with later heavy drinking for the girls in the study (Griffin, et al, 2000).

(SEE ALSO: *Coping and Drug Use; Disease Concept of Alcoholism and Drug Abuse; Ethnicity and Drugs; Prevention; Treatment; Women and Substance Abuse*)

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FAMILIES AND DRUG USE One major debate in the area of families and drug use continues to be whether dysfunctional family life creates drug addiction or whether drug addiction produces dysfunctional families. In other words, are ALCOHOLISM and other drug addictions diseases of individuals or are they products of disorganized families and other social systems? The former is an "individual-focused" view, often held by drug counselors who favor SELF-HELP groups such as AA, Al-Anon, NA, and the like. The latter is a "systemic" view held by professionals who prefer to treat drug addictions by working with families, in order to change family systems into more healthy environments.

Whatever one's position in this debate, almost everyone agrees that the family is the primary socializing agent in society. However, Glick (1988), a senior family demographer, observed that during the past fifty years American families have been undergoing significant transformations. Social acceptance of various forms of families is steadily replacing the older, normative view of a family as comprising only two parents and their children, with the father as a breadwinner and the mother as a homemaker. In the 1960s and 1970s, decades of social protests, Americans witnessed increasing numbers of cohabiting couples, families being maintained by single parents, and many adults living alone. As a result, divorce, single-parenthood, childlessness, and living alone have become more acceptable. Significant transformation has also occurred in gender attitudes, which moved toward greater egalitarianism and resulted in increased percentages of young men and women who perceived fatherhood as a fulfilling experience (Lewis, 1986; Thornton, 1989).

These changes continued to occur until the early 1980s when they began to level off, and by 1987 a quarter of all children under eighteen years of age no longer lived with both of their parents. Eighty-two percent of these children lived with stepfathers, whereas only 18 percent lived with stepmothers. The late eighties and early nineties, however, seem to have been a period of stabilization, during which all trends flattened (Glock, 1988; Thornton, 1989).

No systematic analysis has been conducted to assess the association between these social and demographic changes in the family and trends in drug abuse. If one looks at the statistics closely, however, one sees that the trends in families and in drug use look similar. A dramatic increase in the abuse of all kinds of drugs by all age groups was observed during the early 1970s to early 1980s. These trends in drug use also flattened in the early eighties and, as was observed in 1988, are beginning to drop significantly, especially among youth aged twelve to seventeen years.

This line of reasoning is not meant to suggest that the changes in attitudes toward families and the changes in family structures and forms in the last three decades directly caused the current trends in drug use. It may suggest, however, that the instability of families either allows there to be or

imposes greater stresses upon individuals and society. Similarly, the stabilization of families provides more secure environments for individuals, who may then more effectively cope without the abuse of substances.

There is nevertheless some evidence and much speculation about a reciprocity between an individual's drug addictions and "family illnesses," since the latter often appear to be passed from one generation to another.

Although recent reductions in the use of illicit drugs present a somewhat optimistic picture of the future of American families, the overall number of drug casualties is still grim and the consequences are debilitating. Every year, 100,000 Americans die as the result of drug abuse. That number should increase with the spread of AIDS. Alcohol, nicotine, and illicit drug abuse are number-one health problems, especially among the young. Life expectancy has steadily risen over the past seventy-five years in all age groups except that for youth aged fifteen to twenty-four, who now have a higher death rate because of injuries and disappearances related to drug use. Long-term substance abuse is associated with DEPRESSION, hostility, malnutrition, lower social and intellectual skills, broken relationships, mental illness, economic losses, and growing CRIME rates.

FAMILY PREDICTORS OF DRUG ABUSE

Family factors that predict drug use may be put into three interrelated categories: structural, historical, and interpersonal. The structural factors pertain to family composition, such as single- or two-parent families, the number of children, sibling spacing, and gender composition. Family historical factors specifically refer to intergenerational patterns, such as the extent and influence of drug usage in the family of origin. Finally, interpersonal factors relate to interpersonal dynamics in the family, such as those reflected in the quality of marital relationships or the quality of parent-and-child or sibling relationships.

Family Structural Factors. Three structural factors—parental composition, family size, and birth order—are the most often included variables referred to in drug and family research. Although these factors seem to contribute to the etiology of drug abuse, one needs to look at the findings more critically to try to evaluate the extent of their influence.

The literature on drug abuse is replete with findings that suggest that, compared with traditional nuclear families, disorganized, especially single-parent families are more vulnerable environments for children. These families are associated with an earlier onset and greater degree of drug and alcohol abuse. Information regarding the role of family size and birth order, however, is currently insufficient. According to the data, there are very limited indications that an only child is the least at risk, whereas families with seven or more children are at greater risk for drug abuse. However, there seem to be fewer cases of drug abuse involving first-born children compared with the number of cases involving subsequent, especially last-born, children (Barnes, 1990; Glynn, 1984).

Stanton (1985), Hawkins et al. (1987), and Wells and Rankin (1991) have argued that family structural factors do not contribute much to our understanding of drug-abuse behavior. More important risks for children, they suggest, lie in family processes and the quality of family environments.

Divorce, for example, may be a healthy way of ending a hostile marital relationship. The separation of parents may only be the culmination of hostile relationships, painful negotiations, and the draining of family resources prior to the family breakup. Sessa and Steinberg (1991) argue that the most important impact of divorce on children is how much it disturbs the children's developmental tasks—for example, their autonomy. Most children experience relatively brief adjustment problems following a divorce, but continued development of the adjustment process depends on many more factors, such as the age of the children, the gender, the custodial parent, and the quality of life in the home after the divorce.

Different forms of families may possess varied abilities to exercise certain parenting practices, like monitoring and supervision. Dishion, Patterson, and Reid (1988) found interesting linkages between living in a single-parent family, poor parental monitoring, and greater adolescent involvement with drug-abusing peers. In a supportive family relationship, however, parental composition is not a predictor of adolescent drug use.

Variations in family size may impose certain restrictions and may afford opportunities for the utilization of family resources, such as parental

support and finances. Birth order seems to expose each child to different opportunities for social learning (e.g., in regard to role models) and different behavioral expectations, depending on one's family traditions. It is therefore important to look at family processes and the quality of family environments as well as at the family structure.

Family History Factors. Some well-established evidence indicates that drug use by any member of the family is related to drug use by other family members. In couple relationships, the initiation of a female partner into illicit drug use and her progression toward drug dependency are related to patterns of drug use in the male partner, whereas illicit drug use by the male partner is more independent of spousal drug use (Weiner, Wallen, & Zankowski, 1990).

Parental and sibling drug use have consistently been found to be associated with ADOLESCENT drug-abusing behavior (Hawkins et al., 1986). The transmission of the problem behavior, however, is perceived differently by different scholars. Although there is an increasing fascination with GENETIC explanations, more research is needed to validate genetic assumptions (e.g., Cadoret, 1990; Searles, 1990, 1991). In their view of the literature, Hawkins et al. (1986) concluded that the evidence from behavioral genetic research was limited to male ALCOHOLISM and the lack of convergent evidence from adoption, twin, and biological response studies. Similar criticism has been presented by Searles (1990, 1991), who also argued that only 20 percent of children of alcoholics become alcoholics and that half of all alcoholics do not have a family history of alcoholism. Research on the family clustering of OPIATE and ALCOHOL abusers indicates that a genetic explanation is inadequate when it is considered that the community or environment affects the choice of the substance of dependence.

A systemic (family) approach presents more compelling explanations. Research focusing on the role of parental attitudes and values has revealed a high congruence between parents' and adolescents' perceptions of the use and abuse of drugs (Barnes, 1990). When parents use drugs such as CIGARETTES and alcohol, it indicates to the children that such use is expected (or at least allowed) in the family.

Heavy drug use in the family, especially by parents, also disrupts functional properties of the family system (e.g., care and support, problem solving, etc.), and this, in turn, provides a conducive environment for drug use and abuse by other members of subsequent generations (Steinglass et al., 1987). Dishion and Loeber (1985) argued that parental drug use diminishes parental ability to exert effective monitoring and supervision, thus allowing children to mingle with peers who abuse drugs frequently. Clinical observation also suggests that parental drug use blocks effective communication, alters modes of interpersonal relations, and is associated with all kinds of child abuse (Barnes, 1990; Leonard & Jacob, 1988).

Interpersonal Factors. There are at least two broad dimensions of interpersonal dynamics in the family—support and control—and one facilitating dimension—communication (Barber, 1992; Rollins & Thomas, 1979). The support dimension refers to the positive affective experience associated with relationships, such as acceptance, encouragement, security, and love. The control dimension pertains to the extent to which children's behavior is restricted by the caregiver(s), and this ranges from establishing rules and discipline to varieties of physical coercion (e.g., hitting and yelling). Familial support is regarded as the most robust variable in the prevention of all kinds of delinquent behaviors in children and adolescents (Baumrind, 1991; Gecas & Seff, 1990). Different aspects of support have recently been identified, such as general support, physical affection, companionship, and sustained contact (Gecas & Seff, 1990), all of which are negatively associated with socially unacceptable behaviors. Coombs and Landsverk (1988), for example, found consistent evidence that maintaining a rewarding parent-child relationship deters substance abuse during childhood and adolescence (see also reviews by Glynn, 1984; Hawkins et al., 1986). Parental praise and encouragement, involvement and attachment or perceived closeness, trust, and help with personal problems are all characteristics of the families of abstainers, whereas parental rejection, conflicts, manipulative relations, and overinvolvement are related to the earlier onset and continued use of drugs (Baumrind, 1991; Hawkins et al., 1986).

The control dimension is more complex than the support dimension, since one needs to differentiate between types of control. Baumrind (1987, 1991), for example, distinguished between authoritative and authoritarian controls. The first is characterized by a combination of warmth, supervision, and

opportunity for negotiation; this type of control is associated with positive outcomes. In her study of drug-abusing adolescents, Baumrind found that authoritative control characterized the families of abstainers and soft experimental drug users. Authoritarian control, on the other hand, is based on force, threats, and physical punishment; this is the type of control that characterized the families of dependent drug users. Other studies have revealed that sexual abuse and physical abuse are prevalent in the families of drug abusers.

It has been especially well documented that families with inconsistent or no clearly defined rules also have adolescents who abuse drugs (see Baumrind, 1987; Coombs & Landsverk, 1988; Hawkins et al., 1986; Volk et al., 1989). The constantly changing rules in some families jeopardize parental ability to monitor and supervise children and make it difficult for the children to adapt to family expectations.

In order to function within these two dimensions, families must rely on their communication mechanism. To give support or exert control over others, it is necessary to communicate one's intents. Watzlawick, Beavin, & Jackson (1967) believe that when people communicate, the communication also defines their relationships with other persons. They also believe that to be able to define the relationship, those who communicate should be able to understand each other's perceptions regarding what they talk about and regarding their relationship. In a family where drug use is prevalent, communication is heavily loaded with interpersonal misperception and exchanges of negative affect. Studies also indicate that communication in these families is frequently blocked either by the use of drugs or feelings of not being understood (Hawkins et al., 1986; Jurich et al., 1985; Piercy et al., 1991).

The Family and Other Systems. The peer group and school are two other systems to be considered when the adolescent member of the family who is involved in drug abuse. These systems intervene with their own parenting practices, because they provide much of the environment for learning VALUES, attitudes, and norms as far as expected behaviors are concerned (behaviors that may or may not be expected by the adolescent's family).

It is well known that most new drug users are introduced to drugs by peers and that peers help maintain patterns of use, including greater dependent use. To assess the influence of peers, one should assess the following indicators (Agnew, 1991): (1) time spent with peers, (2) the degree of attachment to peers, and (3) the extent of peer delinquency or drug use.

Although researchers find consistent evidence of the relationship between school DROPOUTS, low performance and underachievement in school, and drug abuse, it is not known when school factors become developmentally salient as possible predictors of drug abuse (Hawkins et al., 1986). Some research indicates that a low grade-point average and dropping out of school are strongly associated with children's involvement with drug-abusing peers. It is clear, on the other hand, that parental involvement in children's schoolwork and activities reduces the changes of a child being seriously involved in drug use.

Hawkins et al. (1987) documented limited evidence with regard to the association of drug use and the social isolation of the family. The 1990 NATIONAL HOUSEHOLD SURVEY indicated that drug users were concentrated within underprivileged families of lower social economic status and within communities of color.

IMPLICATIONS FOR PREVENTION

In the last ten years, those responsible for drug-PREVENTION efforts have discovered that (1) the most effective programs are multilevel programs; (2) it is most cost-effective to target youth aged twelve and younger; (3) the family is the most influential context within which to set programs, especially with drug users who are younger and female; and (4) LIFE-SKILL programs rather than knowledge-oriented programs are most effective in preventing drug abuse.

In the assessment phase, one can determine the risk status of a family by looking at the intergenerational history of drug usage, reported child abuse, the children's academic performance, the degree of parental involvement in schools, and the characteristics of the community in which the family lives (e.g., population density, extent of economic and social deprivation, rates of criminal activity and drug abuse behavior).

In the program development phase, one may well consider issues embedded in (1) individual and family development (Baumrind, 1991; Steinglass et al., 1987), (2) culture and gender (Weiner et al., 1990), and (3) health and economy,

both of which affect the individual and the family (Bush & Iannotti, 1987; Conger et al., 1991). One could also determine how these issues are interconnected in order to come up with the best possible program for specific populations.

In the implementation phase, matching of staff and target group and the ways in which the programs are delivered may affect the outcomes. It may be wise to staff prevention programs delivered in cultures other than the mainstream culture with personnel of similar backgrounds or with those who have an adequate knowledge of that specific culture. Positive and nonthreatening approaches that combine both information and life-skill building are most effective. Parental or significant-other involvement with involvement by the school give programs the most credibility to youth.

FAMILY TREATMENT

As described earlier, dysfunctional family life is one potential contributor to the development of drug addictions in family members. The reciprocal nature of addictions and disorganized families, however, is evident in that not only may dysfunctional families produce addictive behaviors in their members, but these addictions, in turn, may affect the quality of family life, thus negatively impacting the behavior of family members and devitalizing or fracturing family relationships. The most demoralizing aspect of this reciprocity is that drug addictions are often passed from earlier generations to later generations, unless this pattern can be ended by successful treatment or intervention.

Until the mid-1980s, very few drug treatment programs *directly* utilized spouses, parents, or other family members in their treatment of the identified patient. After that time, family therapy became the treatment of choice for most drug abusers, especially in the area of alcoholism treatment. A growing body of research findings has shown that family-centered drug interventions are very effective in getting family members off drugs and keeping them off (Lewis & McAvoy, 1984).

There is evidence, for example, that family groups given systemic family interventions have a higher treatment success rate—that is, decreased drug dependence and less recidivism (Stanton & Todd, 1982). In contrast, if adolescents are treated individually and their family system has not changed, they often return home to resume the same roles and behaviors that had earlier fostered their addictive behaviors.

The inclusion of other family members in an adolescent's drug treatment does add to the complexity of the treatment. Yet this addition often gives a family therapist greater leverage for sustained and successful drug treatment (Lewis & McAvoy, 1984), because of the drug abuser's wish to maintain family love and relationships. Strengthening family relationships may therefore help to reduce or eliminate an individual's addictive behaviors.

Some of the better known interventions currently used in the field of alcoholism treatment are treatments based on family systems. For instance, research has revealed that the spouses of alcoholics often play roles that support their spouse's addiction (through co-dependency). Changes in the spouse's behavior and roles, however, can also contribute to the effective treatment of the spouse's alcoholism (Steinglass et al., 1987).

Systemic family treatment has also been widely utilized in the treatment of adolescents' drug abuse, according to the successful research conducted by Stanton and Todd (1982) with adult heroin addicts. In this programmatic research, one of the best controlled studies of family therapy, the researchers found a significant decrease in the heroin usage of young adults when family-focused therapy was employed.

A longitudinal study of 136 adolescents (Lewis et al., 1991) also documents the relative effectiveness of a family therapy program as compared to a family education program and treatment-as-usual (i.e., individual counseling). In this study, the two brief family-based drug interventions together reduced the drug use of nearly one-half (46%) of the adolescents who received them. This success is thought to be due primarily to the fact that both of these outpatient interventions focused on the systemic treatment of entire family groups. In contrast, the family therapy intervention seemed to have been more effective in significantly reducing adolescent drug use for a greater percentage of the adolescents (54.6% compared with 37.5%). Thus family-based interventions (especially family therapy) can be potent and viable drug-treatment programs.

The best drug treatment, however, may be a combined treatment (Lewis, 1989), in which individual treatment focuses on the teaching of social

skills and strategies for coping with stress, whereas the emphasis of the family treatment component is on increasing the nurturance and parenting skills of other family members. It is at the intersection of these two approaches that much of the current creativity seems to be taking place. Even though their focus and methods may differ, it is good for these two arenas of inquiry to become better known to each other, since each has a wealth of understanding to contribute to the other.

(SEE ALSO: *Adjunctive Drug Taking; Codependence; Conduct Disorder and Drug Use; Conduct Disorder in Children; Ethnic Issues and Cultural Relevance in Treatment; Ethnicity and Drugs; Poverty and Drug Use; Treatment Types; Vulnerability As Cause of Substance Abuse*)

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FAMILIES IN ACTION *See* Prevention Movement

FAMILY VIOLENCE AND SUBSTANCE ABUSE Substance abuse has a profound impact on Americans of all ethnic groups. Many people are concerned about substance abuse, especially because it is believed that it has the major consequence of increasing rates of crimes such as robbery and ''drive-by'' homicides. Yet the physiological, psychological, and social effects of substance abuse extend well beyond acts by individuals against strangers; substance abuse has especially adverse effects on families.

Most individuals' illicit drug use occurs between the ages of eighteen to thirty-five, the childbearing years (National Institute on Drug Abuse, 1993). About 10 million children reside in households that have a substance abuser (Blau et al., 1994), and a minimum of 675,000 children per year are neglected or abused by drug- or alcohol-dependent caretakers (Bays, 1990). At the same time that substance abuse increased, foster care placements increased by 30 percent between 1986 and 1989 (Kelley, 1992).

The extent of spousal abuse by substance abusers is more difficult to document. Although there is much more focus on men as perpetrators and women as victims, women in conjugal relationships do assault their male partners (Halford & Ogarsby, 1993). Recent estimates suggest that annually about 10 percent of married women experience some level of assault (Dutton, 1989) and that between 12 percent to 25 percent experience more serious assault such as being hit or kicked (Andrews & Brown, 1988; Randall, 1990). Physical abuse has been identified as the main reason that between 20 percent and 33 percent af all women seek treatment in emergency rooms (Randall, 1990). Rates for violence against men by their female partners are similar to those reported for violence by men against female partners, but whereas women are believed to commit about 10 percent of murders of nonspouses, they commit 48 percent of murders of husbands and partners (Strauss & Gelles, 1990). Thus, domestic violence by women against men appears much more likely to be lethal when it does occur, whereas domestic violence by men appears more likely to result in severe injuries. Few studies, however, have inquired as to whether either the perpetrator or the victim was a substance abuser or was under the influence of alcohol or drugs at the time of a precipitating incident.

Public awareness of child abuse and neglect has increased dramatically since the mid-1980s, but awareness of spousal abuse has lagged behind. Until recent years, adult victims rarely acknowledged their predicament, attributed signs of physical abuse to other causes, excused perpetrators, and resisted recommendations that they use the legal system to try to deter perpetrators. There are several reasons for reluctance to prosecute. In many instances, wives are dependent on their male partners for economic support, fear loss of their children as a result of custody suits, or conceal abuse to avoid criticism by family, friends, or the community. The still-popular notion that women "deserve" abuse prevails and will only diminish as popular beliefs are replaced with information about the complex circumstances facing abused women.

There are few reliable estimates of abuse of elderly people by family members (Pillemer & Suitor, 1988). Many cases may go unreported. One survey reported that 1.5 million elderly persons in the United States were abused in 1989, but others estimate that the range could be somewhere between 4 percent to 10 percent of the elderly population (Boudreau, 1993). Low rates of spousal abuse (3.3%) have been noted for persons over the age of sixty-five, but only 55 percent of this population is married (Strauss & Gelles, 1990). Since women live longer than men, study of the abuse of elderly people by their children or children's spouses focuses mainly on the abuse of mothers. In relationships between adult children and their parents that have become abusive, predisposing factors include health status, dependency status, social isolation, intergenerational transmission of violent behavior, and external stressors. Anecdotal reports indicate that in 30 percent to 45 percent of cases reported to service providers, perpetrators have mental health or substance abuse problems, but the topic requires more systematic study, especially for rates in the general population.

Most studies of family violence involving children have focused on intergenerational relationships. Much less information is available about abuse among siblings or by other children. For example, research emphasis in studies of childhood sexual abuse has examined characteristics of adult male perpetrators who are stepfathers or other relatives, with sexual abuse by brothers identified as the least frequent occurrence.

SUBSTANCE ABUSE AND FAMILY LIFE

It has been estimated that abuse is associated with psychological disorders in about 20 percent of cases (Stark & Flitcraft, 1988). The family plays an important role in factors relating to the development, maintenance, and treatment of substance abuse. The fundamental significance of families as dynamic systems has been recognized and studied (Wolin et al., 1980). Today, treatment plans for substance abusers typically involve family members or significant others. The disorganizing impact of alcoholism on families is perhaps the addiction that has been best delineated, but information about the impact of other drug use is increasing (Kosten, Rounsaville, & Kleber, 1985; Bernardi, Jones, and Tennant, 1989).

Disrupted family dynamics can occur irrespective of socioeconomic status and ethnic group membership. Research involving a large cross-sectional sample found that offspring of substance abusers were more likely to experience marital instability and psychiatric symptoms, especially if they had experienced physical and sexual abuse (Greenfield et al., 1993), and it has also been found that alcohol abuse often co-occurs with domestic violence (Fagan, Barnett, & Patton, 1988; Dinwiddie, 1992). Construction of "family trees," or genograms, are now in common use as clinical tools to depict the degree to which abuse of various substances has had effects on several generations in a family, the extent that support is available from family members, and the emotional "valence" of kinship relationships (Lex, 1990). Background factors significant for women include childhood violence experiences, violence from a cohabiting partner, and presence of concurrent antisocial and/or borderline personality disorders (Haver, 1987).

Substance abuse and child abuse may co-occur under similar family conditions and dynamics, or substance abuse can lead to child abuse (Kelley, 1992). Mediating factors, such as social support and education, income, alternative sources of nurturing, and parents' own histories of familial substance abuse and histories of neglect and abuse are important. It is likely, however, that when mothers who use drugs or alcohol are primary caregivers, they will be unable to fulfill some aspects of their children's emotional or physical needs (Tracy & Farkas, 1994).

One typical factor in family lives of substance abusers is the absent father, who usually is affected in some way by substance abuse and whose familial role has had to be reallocated among other relatives (Bekir et al., 1993; Hayes & Emshoff 1993). Often this pattern is transmitted from the grandparental generation to the parental generation. Involuntarily or out of necessity, the missing role is frequently assigned to a child, who has to assume responsibilities inappropriate to his or her age and generation (that is, to act as a spouse or parent). Some children recall having had to raise themselves, since their parents neglected to nurture them or abused or scapegoated them or controlled their activities excessively. Children's responses can include acting out through anger, antisocial behavior, and estrangement, or compliance and assumption of housekeeping, care for siblings, and other domestic tasks. In adulthood, resentment because of the burdens of these childhood role reversals can promote depression in individuals and affect their adjustment to adult roles, and it can, in turn, damage their relationships with their own offspring. In some cases, the onset of substance abuse in children occurs at the age or life-cycle stage when a parent began substance abuse. Substance abusers often appear to expect parental unconditional love from their spouses that includes unquestioned acceptance of their substance abuse and irresponsible behavior (Bekir et al., 1993). Unstated expectations and other communication difficulties occur when the moods and behaviors of substance abusers are closely tied to those of family members (McKay et al., 1993). "Low autonomy" (emotionally dependent) substance abusers, however, appear to respond well to treatment if family members provide more nurturing and support. Conversely, male substance abusers whose attitudes and actions are independent and detached from family concerns seem to exhibit a pernicious individualism that is associated with a poor outcome in treatment.

CONSEQUENCES OF ADDICTION IN CHILDREN

Infants exposed to drugs in utero can present problems for caretakers, such as the consequences of prematurity, low birth weight, retarded intrauterine growth, and developmental delays (Blau et al., 1994; Scherling, 1994). Cocaine-exposed infants can be irritable and easily overstimulated, exhibit increased muscle tone, and resist attempts at soothing (Kelley, 1990). There is also a large literature on alcohol effects in utero, which may affect at least 2.6 million infants annually (for review of this literature, see Finnegan & Kandall, 1992). For drug-dependent mothers, these babies sometimes present overwhelming challenges that are often interpreted as "personal" rejection. Mothers' emotions can include guilt about exposure of their child to drugs as well as anger that their efforts at parenting hyperactive babies with feeding difficulties and abnormal sleep patterns seem unsuccessful and only generate more stress. The attachment between mother and child may be disrupted because mothers experience these infants as being highly demanding and ignore and withdraw from them or continue to use drugs. All too often, the consequences of disrupted attachment lead to child neglect and abuse.

PRECIPITATING FACTORS

Alcohol, Drugs, and Aggression. It is popularly believed that alcohol use facilitates the commission of violent acts. Although there is an association between alcohol (and drug) use and aggression, it is not appropriate to attribute all family violence to substance abuse, and substance abuse does not inevitably result in violence (Hayes & Emshoff, 1993; Taylor & Chermack, 1993). Individual, familial and environmental factors are all implicated in family violence. Controlled studies in research laboratories constitute one means of disentangling the important interrelationships of these factors. One series of laboratory experiments that used electric shocks between competitors as a proxy for aggressive behavior (see Taylor & Chermack, 1993) showed that both the quantity of alcohol that has been consumed and the social environment encouraging aggression are two major contributing factors. Results should be interpreted cautiously, since the extent to which controlled laboratory conditions, and the stimulus of a shock, can be generalized to the events in daily domestic life in households with a person who meets the diagnostic criteria for substance dependence or abuse remains to be demonstrated (Leonard & Jacob, 1988).

Experiments were designed to identify factors that could instigate aggression in persons intoxicated with alcohol. In an interactive setting, re-

search subjects were tested while sober and while intoxicated (i.e., about 0.10 blood alcohol level, or the limit for intoxication while driving in many jurisdictions). Since actual violence could not be condoned ethically, the experiment could only give the illusion that a subject would compete with an "opponent" who could signal intention to send a shock of intense magnitude.

Unless their opponents indicated willingness to administer a strong shock, 80 percent of the sober subjects and 40 percent of the intoxicated subjects were reluctant to retaliate by increasing the magnitude of the shock presumably to be received by the opponent. An additional important factor was pressure from bystanders. In another experiment, two accomplices of the experimenter encouraged both sober and intoxicated subjects to use high-magnitude shocks against their opponents. Under this condition, escalation of shock strength occurred for 10 percent of sober subjects and 50 percent of intoxicated subjects. Once escalation had occurred, however, intervention by a third party was generally ineffective. Instead, the strategies best suited to averting aggression in intoxicated persons were to show the opponent to be nonthreatening, to announce a conventional limit on aggressive behavior (in this instance, magnitude of shocks), or to divert attention from aggression to more socially acceptable behaviors. Although intoxicated subjects expected opponents to be more aggressive than did sober subjects, using a video camera to project an image of the sober opponent's behavior diminished the aggressive responses.

Effects of other drugs on aggression also were evaluated by using this type of laboratory experiment. These studies are important because some tranquilizers are prescribed for anxiety and irritable behavior (Ratey & Gordon, 1993). Low doses of marijuana could result in aggressive behavior, but high doses suppressed it. The use of low doses of benzodiazepines increased aggression, but amphetamines did not augment aggression, and these results were contrary to prevailing expectations. Other studies showed that pretreatment with nicotine, dextroamphetamine, or propranolol (which lowers blood pressure) inhibited aggressive behavior. Furthermore, when individuals were evaluated on an aggression rating scale, the nonaggressive group did not respond to provocation while intoxicated with alcohol, but persons in the moderate- and high-aggression groups responded with aggression.

Thus, pharmacological action of drugs, dosage, characteristics of the consumer, and the social factors surrounding drug taking are all important factors contributing to aggressive behavior. Disturbance of higher-order information processing, or reasoning, appears to be the factor that best explains escalation in aggression while intoxicated. Intoxicated subjects were likely to continue aggressive behavior once it had begun, unless they were strongly prompted to engage in self-reflection. Weak suggestions to limit aggressive behavior apparently are not perceived. Having crossed a behavioral boundary may make it easier to continue to do so.

It also should be noted that alcohol and other drugs have a pharmacological effect on sexual arousal and sexual behavior. Among men, alcohol can cause secondary impotence and heroin use can delay ejaculation. There also is evidence to support the notion that cocaine use can increase sexual interest for men and women, and marijuana use has become associated with uninhibited sexual activity. Some women find that heroin use by their partner prolongs intercourse, and once heroin is used as an adjunct to sexual activity, couples are prone to relapse to drug use (Lex, 1990).

Pharmacological effects of alcohol and drugs can also distort communication. For example, large doses of alcohol consumed in short periods of time can result in blackouts, or disrupted short-term memory. A person in a blackout is unlikely to remember what was said and done during the episode. Excessive cocaine consumption can result in suspicion, hostility, and paranoia. A person in a state of withdrawal from alcohol or drugs can be irritable, and oscillation between withdrawal and intoxication distorts communications, thereby leading to inconsistency, unpredictability, and mistrust (Hayes & Emshoff, 1993).

Social Context of Domestic Violence. Many sociologists have assumed that domestic violence is a relatively rare event, and until the 1980s anthropologists had only a limited perspective on the occurrence of family violence in other societies. In a major analysis of data from ninety societies (Levinson, 1987), it was found that wife beating was nearly ubiquitous and predictably associated with social and cultural factors. The frequency of wife beating was analyzed, and societies were clas-

sified according to whether wife beating was absent or rare, occurred in less than half of households occurred in more than half of households, or was present in almost all households. Using these criteria, it was found that wife beating occurred in 84 percent of the societies in the sample. Occurrence of this behavior was best explained by both social acceptance of violence and economic dominance of men. In a restudy by Erchak and Rosenfeld (1994), additional societies were selected for analysis and when wife beating was coded as simply being either present or absent; it was found that that it occurred in 80 percent of the sample. However, social isolation occurred in 47 percent of societies without wife beating, in contrast to occurrence in 94 percent of nonisolated societies. Socially isolated societies were typically smaller, and their members need to be mutually interdependent for the purposes of survival. In comparison, societies where raiding or warfare against outsiders was common—that is, where disputes with outsiders were resolved by physical force—had a wife-beating rate of 85 percent, versus 29 percent for societies without warfare. In societies that strongly emphasized men's role as warriors, rates of wife beating were 94 percent, in contrast to rates of 56 percent in societies lacking these attitudes and behaviors. Neglect or abuse of children co-occurred with wife beating. Other associated values were beliefs about women's inferiority, the lack of value of women's lives, and a widow's ability to choose a new spouse. Additional associated behaviors included tolerance for homosexuality, control of female sexuality, and competition for economic resources. Thus, the current prevailing desire of women for equality between men and women in the United States may be counterproductive and result in more violence, because of increased economic competition between the sexes and increased confusion about appropriate gender-related social behaviors (Erchak & Rosenfeld, 1994).

For impoverished members of minority groups, attributes of the community and neighborhood can adversely affect family life (Wallace, Fullilove, & Wallace, 1992). In a number of urban areas, deterioration of housing, decreases in levels of services such as housing inspections and response by firefighting and arson units, and diminished police presence have permitted the dynamics of urban decay to operate. As buildings deteriorate, are further damaged by vandalism, and are destroyed by fire, the impact is much like the spread of a contagious disease. Adjacent buildings may be affected as landlords abandon housing stock and businesses leave or fail. Whole blocks may be damaged, and, finally, entire districts of a city may deteriorate completely.

The quality of life diminishes accordingly. Abandoned buildings are taken over by substance users and sellers or used for other illicit activities such as prostitution. Adolescents can gain ready access to drugs and alcohol, and their behavior may go unchallenged. As people move away, there remain fewer persons available to notice children's behavior, and more unsupervised locations become available where children can engage in disapproved acts. When an area lacks former types of social control, such as sanctions from neighbors, acts such as smoking tobacco cigarettes may escalate to greater deviance, such as using marijuana or crack cocaine. As a consequence, antisocial behaviors may go unchecked, and feelings of anger and hostility can grow. It should be noted, however, that urban settings are not the only locations in which deviance can increase. Contexts that permit anonymity, including ready accessibility of transportation, can also separate perpetrators from persons who know them or would report deviance to authorities.

Perpetrators of Domestic Violence. Much recent attention has been focused on the psychopathology of both perpetrators and victims. One review (Dinwiddie, 1992) suggested that perpetrators had poor communication skills, higher levels of hostility, and, predictably, less control over their anger. Perpetrators studied for personality problems were more likely to be antisocial, passive-aggressive, or narcissistic. The picture is less clear regarding substance abuse, although men meeting criteria for alcohol abuse or dependence (American Psychiatric Association, 1980) were more likely to hit or throw objects at their wives. Studies of community samples have generally found that perpetrators also meet the criteria for diagnoses of depression and antisocial personality disorder.

In one study, rates of spousal abuse and other problem behaviors were studied in 380 married male relatives of alcoholics (Dinwiddie, 1992). Only 16 percent of the men were self-reported spouse abusers, and 30 percent of these were separated or divorced at the time of the interview, in contrast with 14 percent of the nonabusers. When

effects of single diagnoses were examined, alcoholism was the most commonly diagnosed psychological disorder (87%) and was associated with an almost fourfold increase in likelihood of abuse. Diagnoses of antisocial personality disorder (46%) or major depression (33%) were associated with an almost double increased likelihood of spousal abuse. Only four abusers (7%) had no psychological disorder. Most abusers, however, had more than one diagnosis of psychological disorder. Antisocial personality disorder or depression usually co-occurred with alcoholism. Among nonabusers, 65 percent were alcoholic, 23 percent were drug dependent, 20 percent had major depression, and 31 percent had an antisocial personality disorder. Aggressive childhood behaviors were poor predictors of abuse in adulthood, but as adults 95 percent of all abusers reported having physical fights, about half reported marital infidelity, 23 percent had been divorced one or more times, and 17 percent had made attempts at suicide.

Alcohol problems and marital distress appear to be highly interrelated (Halford & Osgarby, 1993). Drinking outside of the home increases marital dissatisfaction, and marital disputes can provoke a relapse in abstinent alcoholics. Divorce rates for alcoholics are thought to be highest among persons with psychological disorders, and divorce or marital problems diminishes the likelihood that alcohol treatment will succeed for individuals. Treatment efforts directed at increasing marital stability, however, can successfully promote abstinence (McCrady et al., 1979). Accordingly, many therapists who treat people for alcoholism suggest conjoint treatment for alcoholism and marital problems. In contrast, few marital therapists address issues of alcohol abuse (Halford & Osgarby, 1993).

A sample of eighty-four women and fifty-six men seeking marriage counseling were identified in a marriage guidance clinic (Halford & Osgarby, 1993). All subjects were still married and cohabiting. The subjects were mainly in their thirties, had about two children, and had been married about nine years. One-third were involved in second or later marriages. The subjects completed questionnaires that probed for information about amounts of alcohol consumption, occurrence of physical violence, and frequency of disputes about alcohol use. About half of the men, but less than 20 percent of the women, met the criteria for a diagnosis of alcoholism. More than 80 percent of the entire sample reported having repeated arguments about alcohol intake, and almost 70 percent reported the occurrence of physical violence. Men and women taking steps leading to divorce were more likely to report disagreements about alcohol use. Women mentioned male violence as a factor in marital dissatisfaction, but men who had been abusive were more likely to seek divorce. In this sample, alcohol abuse was significantly associated with couples taking steps toward divorce, but few other common sources of marital dissatisfaction, such as allocation of household tasks, communication, finances, use of leisure time, and parenting issues, were reported to any significant extent. At the very least, these data suggest that marital therapists should routinely screen their clients for alcohol intake and alcohol-related problems, and that they should assess the extent to which these factors interact with domestic violence. It also is possible that abuse by a husband signals a desire to terminate the relationship rather than to exert greater control over the wife's behavior within the context of marriage.

Disentangling cause-and-effect sequences between alcohol or drug abuse and family violence is an important and necessary step in understanding factors that promote or maintain any interrelationships. There are several ways of approaching these questions, and researchers with competing theories have attempted to explain the relevant issues (Fagan et al., 1988; Strauss & Gelles, 1990). One theory termed "deviance disavowal" has argued that drinkers are not responsible for their actions while they are intoxicated (McAndrew & Edgerton, 1969). Drunkenness is used as an excuse, and it is possible that some persons seek an intoxicated state so as to be able to engage in violent behaviors (Gelles, 1974). According to another theory, alcohol acts on the central nervous system to create a "disinhibition" that releases aggression. Although this reflects a popular belief about the effects of alcohol, it is the social environment promoting or discouraging aggression that is an important contributing factor (Strauss & Gelles, 1990; Taylor & Chermack, 1993). Social learning theory has been applied to a wide variety of behaviors, and the proponents of this theory argue that social meaning becomes attached to behaviors, such as alcohol use, with the result that people come to expect certain behaviors in association with alcohol. Researchers who support a more focused approach have suggested that drinking and violence become associ-

ated within the family context, and that discussion of drinking behavior acts as a cue or trigger that escalates verbal hostility and culminates in physical aggression (Fagan, Barnett, & Patton, 1988).

Characteristics of Perpetrators and Victims. One study used a Relationship Abuse Questionnaire to assess levels of marital violence among abusive and control subjects, including happily married men, maritally dissatisfied men, and men convicted of a violent offense who had not committed acts of domestic violence (Fagan, Barnett, & Patton, 1988). Men in the marital-violence group were young males from minority groups, with limited education and a high rate of unemployment. All members of these groups had been married for an average of four years, had about two children, and were between one to two years older than their wives. Maritally violent men were more likely to consume whiskey and beer, drink daily, drink at lunch on workdays, and drink at home—after work and in the company of their children or by themselves. In addition, maritally violent men indicated that their female partners also drank, but to a lesser degree than they did. These men in the maritally violent group reported that they drank to "deaden the pain in life," to "cheer up a bad mood," to "relax," to "celebrate special occasions," to "forget worries," "to forget everything," and to allay feeling "tense and nervous." They said their female partners drank to "celebrate special occasions" and to "be sociable." Maritally violent men reported that drinking accompanied abuse about one-third of the time but occurred without drinking occasionally, about one-fourth of the time. Female partners were said to drink on about one-fourth of occasions when abuse occurred. Maritally violent men were most likely to report that in the aftermath of violence they felt "sexy" or "wanted to make love," "tried to stop abuse through reasoning," or "took drugs/had a drink." In sum, these men drank more, drank in many social contexts, perhaps continuously but in low amounts, drank to "escape" unpleasant emotions and events, and had female partners who also drank. Drinking or drug taking could be an outcome, however, rather than the cause of a violent episode. It also should be noted that a violent episode could precipitate sexual activity.

A classic study (Kantor & Strauss, 1989) investigated whether drug or alcohol use by victims increased the likelihood of assault by their partners. Information about violence was obtained from 2,033 married or cohabiting women who responded to the 1985 National Family Violence Survey. Research was stimulated by empirical observations that cultural acceptance of violence was the strongest factor in violence directed at wives. This study was designed to test the hypothesis that victims of violence might in some way precipitate violent episodes. Several studies had indicated that people were more likely to attribute blame for violent episodes to women who had violated the cultural attitude that fosters disapproval of women who are intoxicated and another culturally shaped attitude that excuses intoxicated men from the consequences of their alcohol use, including violence. Specific questions included in the interview asked whether women's alcohol or drug use increased the risk of violence from male partners, whether drinking or drug use by male partners increased the risk of violence, whether intervening variables, such as socioeconomic status, explained the occurrence of violence, and whether minor violence and severe violence had different antecedents.

Events were classified as nonviolent, minor violence (throwing objects, pushing, slapping, or grabbing), and severe violence (kicking, hitting, beating, choking, threatening with knives or guns, or using knives or guns). Subjects also were asked whether they used drugs to the extent of being "high" and alcohol to the extent of being "drunk." Predictably, high rates were obtained for alcohol use. Among nonviolent couples, 16 percent of wives and 31 percent of husbands were reported to use alcohol to the extent of being drunk. In contrast, 36 percent of women and 50 percent of men involved in minor-violence episodes used alcohol, and 46 percent of women and 70 percent of men involved in severe-violence episodes had used alcohol. Correlation of violence with drug use (marijuana) was less than half that of alcohol, but the illegal status of marijuana might have encouraged underreporting. Among nonviolent couples, only 4 percent of wives and 5 percent of husbands were reported to use marijuana. In contrast, 14 percent of women and 18 percent of men involved in minor-violence episodes had used marijuana, and 24 percent of women and 31 percent of men involved in severe-violence episodes had used marijuana. Minor-violence episodes were related to the husband's use of marijuana and to violence in the family of origin of the victim. Drunkenness by the wives and by their

husbands, low income, and the wives' acceptance of male violence were significant factors, but wives' marijuana use was unimportant. Severe-violence episodes showed a more restricted pattern. Violence in the women's families of origin and husbands' drunkenness were somewhat stronger factors than husbands' marijuana use. Income level, wives' acceptance of abuse, and wives' drunkenness or being high did not affect the severity of violence. In this study, pregnancy or employment status were not relevant factors.

Some have argued that pregnancy is a factor in the precipitation or escalation of abuse episodes. A recent study examined the extent of physical abuse in a multiethnic sample of pregnant women (Berenson et al., 1991). Of 501 women using services at a prenatal clinic, about 20 percent reported physical abuse, and of this group, 29 percent had been abused while pregnant. However, only 19 percent had ever sought medical help, thus indicating that emergency-room statistics might seriously underreport the prevalence of physical abuse. Abuse occurred typically within the context of a primary relationship, with 92 percent of women reporting abuse by only one person, usually (83% of the time) a male partner. Women who had been abused were more likely to report having a partner who abused alcohol or drugs. Abused pregnant women had significantly more pregnancies and more living children than other pregnant women. Across ethnic groups, white non-Hispanic women were 3.5 times more likely than Hispanic women and 1.6 times more likely than black women to experience physical abuse. Substance abuse increased risk of abuse for white non-Hispanic women to two times that of non-abused women, but for black women, almost four times. Other characteristics were important. Traditional values, as exemplified by speaking Spanish, appeared to be a protective factor for Hispanic women. Divorced or unemployed black women, however, were at higher risk for abuse than either Hispanic or white women. Thus, alcohol or drug use are important factors in the abuse of pregnant women, but black women appear to be at highest risk for abuse when these factors were involved.

There is no single cluster of characteristics that typify men who abuse women. Some studies, however, have indicated that witnessing violence in the family of origin may have taught men to use violence as a coping mechanism. Others have argued that alcoholic abusers also may have had a family history of alcoholism, thereby blurring the relationships between causes and effects in families of origin. In a study of men in a treatment program for family violence (Hamberger & Hastings, 1991), comparisons of marital adjustment, coping with conflict, and personality characteristics were made among alcoholic and nonalcoholic men in treatment and control subjects drawn from the community. The average age of the men was about thirty-five, and they had similar education levels. Nonalcoholic men were more likely to be employed and less likely to have witnessed violence in their families of origin. Alcoholic men who had abused their wives were more likely to have been abused as children, but parental alcohol abuse and parental alcoholism appeared to have no direct role in provoking violence by adult abusers who were alcoholic. As might be predicted, the alcoholic abusers had significantly higher personality-disorder scores for avoidant (passive-aggressive) behaviors, aggression, and negativism, and lower scores for conformity. Both alcoholic and nonalcoholic abusers had a large number of symptoms of pathology, thus scoring high on scales measuring anxiety, hysteria, and depression. Alcoholic abusers had the highest scores on psychotic thinking, psychotic depression, and borderline behaviors. As predicted, abusers had higher scores for personality disorders, and alcoholic abusers had the highest scores in this regard. Alcoholic abusers had witnessed more violence in their families of origin and had themselves been victimized by abusers in their families of origin. Overall, alcohol abuse was significantly related to psychopathology as well as to the degree of harm conferred by abuse. Unemployment as a factor operated in some unknown way to bring abusers to the attention of authorities, but the effect of socioeconomic status was not included in the characteristics examined in this study. Clearly, alcoholic abusers identified through agencies had more severe problems, thus suggesting that treatment programs should carefully assess referral sources of clients. A finding of co-morbidity with depression, anxiety, borderline behaviors, and thought disorders suggests that a program focused on abuse alone would be less successful than a more comprehensive approach that offered services for severe psychological disorders.

In another line of investigation, researchers examined women's histories of victimization and

their alcohol use together with characteristics of their partners. The reasoning behind this approach was the consideration that when abusive behavior was modeled, excused, or condoned, children would perpetuate these behaviors as being appropriate to gender roles. Thus boys would devalue women and consider abuse a conventional way to deal with conflict, and girls would expect to be devalued and would tolerate abuse. One study investigated these background factors among forty-nine abused women and eighteen male abusers (Bergman & Brismar, 1992). Abusers were not identified through their female partners, since many of the women were afraid to permit contact with them and many of the abusers refused to participate. Abusers were selected from men who had been sentenced to prison for assault and battery of their female partners. The extent of injuries inflicted by the selected men and experienced by the women were comparable as a result of matching reports from the abused women and those from the convicted abusers. It was intriguing to find that both the men and the women reported having been raised without fathers in their families of origins, that about half of the absent fathers were alcoholic, and that most of the mothers were abstainers. As children, about 80 percent of both men and women had witnessed domestic violence in their families. Moreover, 29 percent of the women and 11 percent of the men had experienced sexual abuse as children. As adults, almost all of the women (94%) had experienced previous abuse, and 49 percent had been abused by former partners. About half of the men and one-fourth of the women had used marijuana, 62 percent of the women and 44 percent of the men had used sedative-hypnotic prescription drugs, and 55 percent of the women and 61 percent of the men acknowledged that both partners had been drunk at the time of the precipitating episode of abuse (only 20% of the women and 11% of the men had been sober). Roughly two-thirds of the men and of the women indicated that the abusive incident probably would not have happened in the absence of alcohol. Transgenerational perpetuation of abuse patterns seemed likely, since 25 percent of episodes were witnessed by the children of the women and the rate of the parents' alcohol and drug abuse was high. Thus, information about histories of alcohol and drug abuse as well as exposure to domestic violence should be evaluated for each partner in a couple involved in domestic violence.

Less information is available about drug use (see Miller, 1990). Abuse is not uniformly associated with drug use, however. Psychopharmacological factors have been implicated in domestic violence in the case of some drugs, such as cocaine (Maher & Curtis, 1992), and for economic reasons, such as when a drug abuser resorts to appropriation of family funds to purchase drugs. Systemic violence, related to the hazards of illicit transactions, may spill over into the domestic area if a drug abuser is concerned or suspicious that a partner may be an informer or may be adulterating drugs. Female drug users may find themselves devalued on the basis of both their gender and their behavior, and because some women are involved in prostitution to obtain drugs for themselves or their partners, their risk of exposure to violent behavior is increased substantially. Intoxicated women also may be more verbally aggressive and thus violate the cultural norm that values the "soft-spoken" woman (Miller, 1990).

Studies of alcohol abuse as it is associated with the abuse of women have not been able to identify a sequence of cause and events. More definitive studies are needed, but one informative study of alcohol and drug abuse by eighty-two male perpetrators and victims sought important linkages. The perpetrators were parolees, and data about psychological disorders, substance abuse, modes of conflict resolution, and frequency of violent events were obtained from them and their female partners. About three-quarters of the perpetrators, and a surprising 56 percent of their female partners had alcohol problems, and 73 percent of perpetrators and 40 percent of their partners acknowledged using illegal drugs. Similarly, 78 percent of parolees and 72 percent of their female partners reported perpetrating a moderately violent episode, and 33 percent of parolees and 39 percent of their female partners reported perpetrating a severely violent episode at least once during the three months before the interview. About one-third of the episodes were considered severe, and about three-fourths were considered moderate. Neither alcohol nor drug use was involved independently, but concurrent use contributed significantly to violent events, and the separation of drugs into different classes by pharmacological action did not change the effect of alcohol and drug interaction. When combined, however, cocaine and alcohol had a strong effect on violence. In addition, couples with more substance

abuse-related problems had a higher incidence of violent episodes, but, overall, alcohol problems most strongly increased the likelihood that violence would occur. Additional studies of women with concurrent alcohol and drug abuse problems are needed to clarify the temporal relationships.

TREATMENT FOR ABUSERS

Shame, guilt, and denial are powerful emotions that impede both the recognition of problems and the admission of the need for help. It is popularly believed that perpetrators enter treatment only under coercion and with considerable reluctance. Given the strong association between substance abuse and marital violence in some individuals, questions arise as to whether treatment of alcohol or drug abuse alone will concomitantly diminish violent acts. Behavioral marital therapy teaches improved communication skills and has been used to improve the marital relationships of patients as their drinking abates (O'Farrell & Murphy, 1995). This treatment modality, however, does not directly address the problem of violence. A comparison was made between eighty-eight couples with a newly abstinent husband and a nonalcoholic control sample of eighty-eight couples undergoing marital therapy. The study covered the year before treatment and the year after it. Acts of domestic violence occurred between four to six times more frequently during the year before treatment. Rates for violent episodes during the year after treatment remained elevated for both men and their wives, and they were higher than the rates among control couples. In instances of relapse, rates were higher than those for couples who had not relapsed. In turn, rates for couples who had not relapsed were comparable to those for controls. Consequently, effective treatment for alcoholism appears to reduce the frequency of domestic violence, although a study that uses a control group of conjugal pairs not receiving behavioral marital therapy is needed for conclusive results. The cause-and-effect relationships between the release of emotions and relapse still need to be disentangled, however, since the former may provoke the latter or have an additive effect.

Another study examined rates of violent acts among seventy-four persons who completed a treatment program for spousal-abuse abatement and thirty-two who relapsed from this program. Men were referred by themselves or the courts, but neither source of referral nor amount of criminal activity had an effect on outcome. Alcohol problems persisted in 32 percent of the men who completed this program successfully, but 56 percent of recidivists had persistent alcohol problems. Recidivists also had higher levels of drug abuse and less empathy as measured on standardized scales. Recidivists also were found to be significantly more narcissistic (self-centered) and gregarious. These findings suggest that alcohol and drug abuse must be addressed when they occur among perpetrators of domestic violence.

COMMENTARY

Numerous studies that use standardized criteria generally support the prediction that substance abuse and domestic violence co-occur in the majority of violent episodes. Roughly one-fourth to one-fifth of episodes, however, occur without substance abuse as a possible co-factor or precipitant. Some additional studies suggest that verbal hostility can escalate domestic conflict to domestic violence (Lindman et al., 1992), but some episodes of verbal hostility may stem from response to life stress and others may be a result of social learning. In other instances, conflict over a child's or a partner's alcohol or drug consumption may prompt the substance abuser to "protect" the behavior through vehement denial, thereby leading to an escalation of hostility that spirals out of control.

Although any suggestion that women's behaviors might contribute to abuse may seem to take the currently unacceptable position of blaming the victim, there is some evidence that women who express aggression verbally may have had abusive families of origin, and that alcohol abuse may have played a role in fostering a climate of tension and hostility within their households (Gomberg, 1993; Hayes & Emshoff, 1993). This pattern may emerge when women who feel devalued have no behavioral alternative through which to express their frustration. Unfortunately, many potentially interesting and informative laboratory experiments that investigate aggressive behaviors are conducted with undergraduate college students and thus may not disclose important information about effects that stem from income level, social class, educational level, or ethnicity.

Data from alcoholic and drug-abusing women in treatment suggest that younger women may be more verbally aggressive, thus reflecting society-wide changes in gender-role behavior. Other data (Miller, Downs, & Testa, 1993) reveal that women who were victimized as children are more likely to develop alcohol and drug problems in adolescence and adult life. In contrast to women with other psychological disorders, women who require substance-abuse treatment recall more abuse during their childhood. Some contribution to this outcome could be diminished self-esteem and increased alienation from typical childhood socialization processes, as well as limited development of social skills for negotiation and compromise.

It is also possible that the contexts of substance-abuse treatment generate a social expectation that a client must have a family history of substance abuse as well as a background that includes emotional, physical, or sexual abuse. It is clear that additional research is needed and that subject samples need to be drawn from different sources, with different prevalence rates of various types of violence. Longitudinal research that would follow a cohort of children through adolescence, young adulthood, and marital life might hold sorely needed answers. Lacking the answers obtained from definitive research, it is reasonable to continue to screen abuse victims and perpetrators for substance-abuse problems, and to screen substance abusers for perpetration of or victimization through family violence. Because both substance abuse and family violence engender denial that anything is wrong, careful assessment is a prerequisite for effective prevention, intervention, and treatment.

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BARBARA LEX

FERMENTATION Fermentation is a natural metabolic process that produces energy by breaking down carbohydrates (such as sugars) in the absence of oxygen. It occurs in many microorganisms (such as yeasts), and the end product can be either ethyl alcohol (ethanol) or lactic acid; energy is typically given off in the form of heat. The chemical reaction of this process was first described in 1810 by the French chemist Joseph Louis Gay-Lussac. Fermentation is important to the production of many foods and beverages, the most popular of which are bread, butter, cheese, beer, and wine.

Figure 1
Grapes

Fermented foods first occurred naturally, when stored or forgotten caches were found to be altered but edible. In ancient times, wheat and barley were domesticated, farmed, stored, and used to make breads and porridges—some of which fermented and formed brews. Since that time, the process of fermentation has been used worldwide. Industrial means provide huge quantities of fermented foods, as well as alcohol, which is obtained by DISTILLATION from fermented juices of fruits, grains, vegetables, and other plants.

(SEE ALSO: *Beers and Brews*)

SCOTT E. LUKAS

FETAL ALCOHOL SYNDROME Fetal alcohol syndrome (FAS) is a constellation of behavioral, growth, and facial abnormalities resulting from prenatal alcohol exposure. Diagnosis is made by a specially trained physician and is based on the following criteria: growth deficiency; a pattern of distinct and specific facial abnormalities; and central nervous system (CNS) damage. In other cases, where there are no related physical findings, but a pattern of cognitive and behavioral deficits exist concurrent with confirmed prenatal alcohol exposure, a diagnosis of static encephalopathy may be given. Due to confusion, this term and fetal alcohol related conditions (FARC) are used in the place of fetal alcohol effects (FAE). The characteristics listed above and discussed later in this entry must occur in conjunction with confirmed maternal alcohol consumption. Racial, genetic, and familial influences must also be considered when such a diagnosis is made.

HISTORY

The term fetal alcohol syndrome was first used in 1973 to describe the physical problems seen in the offspring of alcoholic women. There have been admonitions against women drinking during PREGNANCY for literally thousands of years—in biblical verses and in the writing of the ancient Greeks. The physical and social implications of women drinking during pregnancy first became highly noticeable during the gin epidemic of the 1750s. At that time, gin became a cheap and easily accessible beverage among low-income women. It was noted that there was a correlation between women who were consuming large amounts of gin and problems among their offspring.

A formal study was conducted in the 1890s by an English physician named Sullivan. He identified the offspring of 120 female "drunkards" in the Liverpool jail and compared them to the children of their nondrinking female relatives. From this study, Sullivan noted a perinatal mortality rate that was two and one half times higher in the offspring of the female alcoholics.

In 1968, Dr. Paul Lemoine published a study on the children of women alcoholics in a French medical journal. This article did not receive much attention until the landmark articles published in the *Lancet* by Jones, Smith, Ulleland, and Streissguth in 1973. Since 1973, more than five thousand articles have been published detailing the effects of prenatal alcohol exposure from birth through middle age. There can be no doubt that alcohol is a powerful teratogen (causative agent in fetal malformations) with lifelong after-effects (sequelae).

DISTRIBUTION

The prevalence of FAS ranges widely from community to community and is determined by the number of women consuming alcohol in any particular community. It is estimated that FAS is now the leading cause of mental retardation in the United States, surpassing Down's syndrome and spina bifida. The prevalence estimates for FAS range from 1 in 600 to 1 in 750 births. However, few prevalence studies have been conducted and many experts have differing views as to the accuracy of the prevalence figures available. New Centers for Disease Control (CDC) studies suggest that drinking during pregnancy is actually on the increase, despite public-health information designed to prevent FAS. This trend may lead to a higher number of babies born with FAS/FARC.

PHYSICAL EFFECTS

Scientific research indicates the likelihood that there is no level of alcohol consumption guaranteed free from risk for any period during pregnancy. Individuals react very differently to alcohol and it is difficult, if not impossible, to predict which women will produce a child with FAS. The exception to this is the woman who has already given birth to a child with FAS or FAE. If this woman continues to drink at the same or an increased level, it is highly likely that her subsequent pregnancy will be affected to the same or a greater degree.

Drinking alcohol during pregnancy produces different effects, depending on *when* the alcohol is consumed. During the first trimester, there is a chance of major physical abnormalities and central nervous system (CNS) damage. During the second trimester, alcohol consumption leads to an increased rate of spontaneous abortion and CNS damage, as well as more subtle physical abnormalities. During the third trimester, alcohol consumption can lead to pre- and postnatal growth retardation and CNS damage. These characteristics are detailed below.

As was mentioned above, three major indices are used in diagnosing FAS. First are the common facial abnormalities: These include short palpebral (eye-slit) fissures; a long smooth philtrum (upper lip groove); and thin upper lip. Other common physical problems associated with prenatal alcohol use include cardiac (heart) malformations and defects; pectus excavatum (hollow at the lower part of the chest due to backward displacement of xiphoid cartilage); clinodactyly and camptodactyly (permanent curving or deflection of one or more fingers); fusion of the radius and ulna at the elbow; scoliosis (lateral curvature of the spine); kidney malformations; and cleft lip and palate.

Growth deficiency in FAS is noted in three parameters: height, weight, and head circumference. Many of the prepubescent patients experience growth retardation; they are generally short and skinny in appearance. Significant changes in weight are noted as the female patients enter puberty; although the growth deficiency remains in height and head circumference across the lifespan, the girls frequently gain weight and appear plump. The male patients seem to remain fairly short and slender until their late twenties or thirties.

CNS damage is frequently manifested in cognitive and memory deficits, sleep disturbances, developmental delays, hyperactivity/distractibility, a short attention span, an inability to understand cause and effect, lower levels of academic achievement, impulsivity, and difficulty in abstracting. The difficulties noted in infancy and early childhood are often precursors to psychosocial deficits in later life.

PSYCHOSOCIAL AND EDUCATIONAL ISSUES

Ages Birth to 5 Years. Diagnosis of alcohol-related birth defects is possible at birth but many physicians are either not trained to identify FAS or do not consider it a possibility. Perinatal behavioral manifestations of FAS include the following: poor habituation, an exaggerated startle response, poor sleep/wake cycle, poor sucking response, and hyperactivity. Failure to thrive, alcohol withdrawal, and cardiac difficulties have become medical concerns frequently noted in this patient population.

Developmental delays in walking, talking, and toilet training are often observed. Concerns such as hyperactivity, irritability, difficulty in following directions, and the inability to adapt to changes are commonly reported. The damage done the brain makes it problematic for children with FAS to learn in a timely and consistent fashion. The more abstract the task, the more apparent this learning gap becomes, particularly as the child enters adolescence and then adulthood.

Recommended interventions at this age focus on the family as well as the child. Many children with FAS are removed from the care of the biological mother owing to abuse, neglect, and/or premature maternal death. Newborns and infants with FAS often have trouble feeding; when this is coupled with a mother who may be deeply involved in substance abuse(s) and not attentive to the needs of her infant, it can lead to medical crises. Therefore, it is necessary to provide the following services and interventions:

- Monitoring of health and medical concerns
- Safe, stable, structured residential placement with services provided to the mother, father, patient, and other family members, such as substance-abuse treatment and parenting training
- Directions given to the caregivers in a simple, concrete fashion, one at a time; directions given to the child in similar fashion
- Adaptation of the external environment to fit the child's level of ability to handle stimulation
- Setting by caregivers of appropriate goals and expectations for their child
- Respite care and ongoing support for caregivers

Ages 6 to 11 Years. Some of the problems noted earlier, primarily health issues, become less severe as others become more severe—with greater implications for negative social functioning. These are hyperactivity, impulsivity, memory deficits, inappropriate sexual behavior, difficulty predicting and/or understanding the consequences of behavior, difficulties in abstracting abilities, and poor comprehension of social rules and expectations. Children with FAS may show decreasing ability to function in school as they get older. The abstracting deficits become more apparent when the child reaches the third and fourth grades and is expected to perform multiplication and division. A summation of suggested interventions at this stage include the following:

- Safe, stable, structured residential placement
- Establishment of reasonable expectations and goals
- Clear physical/behavioral limits and boundaries
- Establishment of reasonable expectations and goals
- Listing of chores and expectations in writing
- Structuring of leisure time and activities
- Education of parents, caregivers, and the patient regarding age-appropriate sexual and social development
- Appropriate educational placement that focuses on an activity-based curriculum, development of communication skills, development of appropriate behavior, and basic academic skills embedded with functional skills

Ages 12 to 17 Years. Children with FAS have the same emotional needs as others this age. Adolescents with FAS may exhibit cognitive deficits, impulsivity, low motivation, lying, stealing, DEPRESSION, suicidal thoughts and attempts, and significant limitations in their adaptive behavior skills. Other concerns include faulty logic, pregnancy/fathering a child, and the loss of residential placement. Social deficits noted encompass financial/sexual exploitation and substance abuse. It is frequently difficult for people with FAS to articulate their feelings and needs. This is commonly the time when they reach their intellectual ceiling.

Despite these problems and deficits, adolescents with FAS should not be infantilized. In addition, this is commonly the time where they reach their academic ceiling. The following are some suggested interventions to help them reach their social, emotional, and adaptive potential:

- Changing the focus from academic to vocational and daily-living skills training
- Structuring of leisure time and activities, such as involvement in organized sports and social activities
- Education of the patients, parents, and caregivers regarding sexual development and the need for birth control or protection against sexual exploitation and sexually transmitted diseases (STDs)
- Planning for future vocational training and placements, financial needs, and residential placement
- Increasing responsibility based on the patient's skills, abilities, and interests

Ages 18 through Adulthood. The problems, deficits, and difficulties seen prior to the age of 18 are precursors to those seen in young adulthood

and middle age. An additional problem experienced by people with FAS is the increased expectations placed on them by others. Not only can people with FAS often not meet these expectations but their impulsivity and poor judgment have more serious consequences than during their younger years. Issues such as poor comprehension of social rules and expectations, aggressive and unpredictable behavior, and depression coupled with impulsivity, may lead to suicide attempts, antisocial behavior, hospitalization, and/or incarceration.

Other concerns noted in adults with FAS include social isolation and withdrawal; difficulties in finding and sustaining employment; poor financial management; problems accessing and paying for medical treatment or child care; and a need for help with social/sexual exploitation and unwanted pregnancy. The hyperactivity and distractibility seen in small children with FAS/FARC manifest in the adult not being able to learn job skills or to meet the requirements of many jobs. The following is a brief outline intended to help adults with FAS deal with problematic issues in a productive fashion:

- A guardianship for or systematic help with whatever funds may be received, since arithmetic skills in this population seldom exceed the third grade
- Subsidized residential placements to help ensure physical safety
- Medical coupons for care, along with birth-control planning
- Homebuilders or community housing to help them live as independently as possible
- Child-care and parenting classes, as needed
- Education to others about FAS, including its limitations and skills, to foster acceptance
- Long-term residential/vocational/psychosocial support for both the patient and/or caregivers

SUMMARY

FAS is a preventable birth defect; once it exists it has life-long consequences. Special programs involving planning for future vocational, educational, and residential needs should be implemented as early in childhood as possible. Education on the harmful effects of alcohol use, focusing on young women and men of childbearing years, is critical to help prevent, or at least reduce, this significant public-health problem.

(SEE ALSO: *Addicted Babies; Alcohol: History of Drinking; Attention Deficit Disorder; Conduct Disorder in Children; Fetus, Effects of Drugs on the; Pregnancy and Drug Dependence: Opioids and Cocaine*)

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ROBIN A. LADUE

FETUS, EFFECTS OF DRUGS ON THE

The pregnant drug-dependent woman subjects her developing infant to a host of problems. When assessing the effects of drugs, especially illicit drugs, on newborn infants (neonates) and young children, two factors must be considered: (1) the duration and concentration of the drug exposure on the developing fetus, and (2) any preexisting medical complications in the mother. These factors are interactive and together will influence, in varying ways, the eventual capabilities of the child. Therefore, the long-term outcome of children exposed to drugs during fetal development should be assessed.

EFFECTS ON THE NEWBORN

A pregnant drug-dependent woman puts her developing fetus at risk for a number of diseases, including hepatitis, ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS), tuberculosis, and sexually transmitted diseases (STDs). A number of these diseases may be acquired through needle sharing. Mothers who are infected with these diseases are likely to deliver prematurely.

In pregnant women who inject HEROIN, the placenta, for example, shows microscopic evidence of oxygen deprivation. The infants are small for their gestational age, with all their organs affected. In heroin-dependent women, a significant portion of the medical complications seen in their newborns is due to prematurity and low birthweight. Such complications include immature lungs, difficulties in breathing at birth, brain hemorrhage, low sugar and calcium levels, infections, and jaundice.

Women on METHADONE MAINTENANCE (an oral NARCOTIC used for the treatment of heroin addiction) are likely to give birth to normal- or almost normal-sized babies. Because they are in treatment, the complications in their infants are not as severe and generally reflect: (1) the amount of prenatal care the mother has received; (2) whether the mother has suffered any complications, including hypertension or infection; and (3) most importantly, any multiple drug use that may have produced an unstable intrauterine environment for the fetus, perhaps complicated by WITHDRAWALS and/or OVERDOSE.

Multiple drug use may cause a series of withdrawals, when the pregnant woman cannot obtain the drug she needs. This series of extreme physical conditions in the pregnant woman can severely affect the oxygen and nutrients that feed the developing fetus, causing various birth defects, depending on when in each trimester the withdrawals occur. If the mother overdoses, a decreased oxygen supply to the fetus can cause aspiration pneumonia—if the mother survives the overdose to give birth.

Laboratory and animal studies have shown that narcotics (OPIOIDS) may have an inhibitory effect on enzymes that influence oxygen metabolism. They also alter the passage of oxygen and nutrients to the fetus by constricting the umbilical vessels and decreasing the amount of oxygen delivered to the developing fetal brain. Such metabolic side effects may cause a derangement in the acid/base balance (acidosis). In contrast, increased maturation of organ systems and certain enzymes have been seen in heroin-exposed infants, including maturation of the lungs, tissue-oxygen unloading, sweat glands, and liver enzymes. The stressful life of the pregnant woman probably contributes to this enhanced maturation in heroin-exposed infants.

The genetic risks to the offspring of addicts on heroin *and* methadone include an increase in the frequency of chromosome abnormalities; infants exposed predominantly to methadone in utero do not. The adverse environmental factors that may contribute to the abnormal findings in heroin-exposed infants may be less prominent in methadone mothers, since drug addiction is compounded by poor maternal nutrition, extreme STRESS, infectious disease, and a lack of early and consistent prenatal care. However, in the absence of specific clinical abnormalities, it is impossible to isolate ei-

ther methadone or heroin as agents linked to GENETIC damage.

Given the obstetrical and medical complications, the lack of prenatal care, and the prematurity of the infants at delivery, it is not surprising that the death rate for ADDICTED BABIES is higher than for infants born to nonaddicts.

NEONATAL OPIOID WITHDRAWAL SYNDROME

This syndrome is described as a generalized disorder, characterized by signs and symptoms of central nervous system hyperirritability, gastrointestinal dysfunction, respiratory distress, and autonomic nervous system symptoms that include yawning, sneezing, mottling, and fever. At birth, these infants develop tremorous movements, which progress in severity. High-pitched crying, increased muscle tone, irritability, and exaggerated infant reflexes are common. Sucking of fists or thumbs is common, yet when feedings are administered, the infants have extreme difficulty and regurgitate frequently—because of an uncoordinated and ineffectual sucking reflex. The infants may develop loose stools and are therefore susceptible to dehydration and electrolyte imbalance. At birth, the blood levels of the drug(s) used by the mother begin to fall, so the newborn continues to metabolize and excrete the drug, and withdrawal signs occur when critically low levels have been reached.

Whether born to heroin-addicted or methadone-dependent women, most infants seem physically and behaviorally normal. The onset of their withdrawal may begin shortly after birth to two weeks of age, but most develop symptoms within seventy-two hours of birth. If the mother has been on heroin alone, 80 percent of the infants will develop clinical signs of withdrawal between four and twenty-four hours of age. If the mother has been on methadone alone, the baby's symptoms usually appear by forty-eight to seventy-two hours.

In summary, various studies have shown that the time of onset of withdrawal in the individual infant will depend on: the type and amount of drug used by the mother; the timing of her dose before delivery; the character of her labor; the type and amount of anesthesia and pain medication given during labor; and the maturity, nutrition, and presence or absence of systemic diseases in the infant.

Studies indicate that more full-term infants require treatment for withdrawal than do preterm infants. Withdrawal severity appears to correlate with gestational age; less mature infants show fewer symptoms. Decreased symptoms in preterm infants may be due to either (1) developmental immaturity of the preterm nervous system, or (2) reduced total drug exposure because of short gestations.

The most severe withdrawal occurs in infants whose mothers have taken large amounts of drugs for a long time. Usually, the closer to delivery a mother takes heroin, the greater the delay in the onset of withdrawal and the more severe the symptoms in her baby. The duration of symptoms may be anywhere from six days to eight weeks. The maturity of the infant's own metabolic and excretory mechanisms plays an important role. Although the infants are discharged from the hospital after drug therapy is stopped, some symptoms such as irritability, poor feeding, inability to sleep regularly, and sweating may persist for three to four months.

Not all infants born to drug-dependent mothers show withdrawal symptoms, but investigators have reported that between 60 and 90 percent of infants do show symptoms. Since biochemical and physiological processes governing withdrawal are still not fully understood, and since multiple drugs are often used by the mothers in an erratic fashion—with vague or inaccurate maternal histories provided—it is not surprising to find varying descriptions and experiences in reports from different centers. Seizures, a severe outcome in withdrawing infants, are rare in narcotic-exposed infants. One report found that 5.9 percent of 302 newborns exposed to narcotics during pregnancy had seizures that were attributed to withdrawal. Other reports found even rarer occurrences of seizures.

Drug-exposed infants show an uncoordinated and ineffectual sucking reflex as a major manifestation of withdrawal. Regurgitation, projectile vomiting, and loose stools may complicate the illness further. Dehydration, due to poor intake and coupled with excessive losses from the gastrointestinal tract, may occur, causing malnutrition, weight loss, subsequent electrolyte imbalance, shock, coma, and death. Neonatal withdrawal carries a risk of neonatal death when these complications are untreated. The infant's respiratory system is also affected during withdrawal: excessive secretions, nasal stuffiness, and rapid respirations are sometime

accompanied by difficulty breathing, blue fingertips and lips, and cessation of breathing. Severe respiratory distress occurs most often when the infant regurgitates, aspirates, and develops aspiration pneumonia.

The increased sensitivity to recognition, the accuracy of clinical and laboratory diagnosis, and treatment have essentially eliminated neonatal mortality attributed to withdrawal per se.

ASSESSMENT AND MANAGEMENT OF NEONATAL OPIOID ABSTINENCE

With proper management, the neonate's prognosis for recovery from the acute phase of withdrawal is good. If symptoms of withdrawal appear, simple nonspecific measures should be instituted, such as gentle, infrequent handling, swaddling, and demand feeding. Careful attention to fluid-electrolyte balance and calorie support is essential in opioid-exposed infants undergoing withdrawal, since they display uncoordinated sucking, feed poorly, often develop vomiting and diarrhea, and have increased water losses due to rapid respirations and sweating.

Indications for specific treatment, dosage schedules, and duration of treatment courses have varied widely. As a general guide, if, in spite of nonspecific measures, babies have difficulty feeding, diarrhea, marked tremors, irritability even when undisturbed, or cry continuously, they should be given medication to relieve discomfort and prevent dehydration and other complications. The dosages must be carefully regulated so that symptoms are minimized without excessive sedation. Several drugs appear to be effective in treating neonatal narcotic withdrawal, but there has been little controlled comparison of their safety and effectiveness. Drugs such as PAREGORIC or tincture of OPIUM are effective in treating narcotic withdrawal symptoms in the infant, and PHENOBARBITAL is useful, but less so when opioid exposure has occurred in high doses.

NEUROBEHAVIOR IN THE NEWBORN

The Brazelton Neonatal Assessment Scale has been used extensively for evaluating newborn behavior. This instrument assesses reaction to stimuli such as a light or a bell, responsivity to animate and inanimate stimuli (face, voice, bell, rattle), state (sleep to alertness to crying), the requirements of state change (such as irritability and consolability), and neurological and motor development. When using this scale in evaluating drug-exposed infants, it was noted that they were less able than nondrug-exposed infants to be maintained in an alert state and less able to orient to auditory and visual stimuli, most pronounced at forty-eight hours of age. Drug-exposed infants were as capable of self-quieting and responding to soothing intervention as normal neonates, although they were substantially more irritable. These findings have important implications for caregivers's perceptions of infants and thus may have long-term impact on the development of infant-caregiver interaction patterns.

Abnormalities in the interaction of drug-dependent mothers and their infants, on measures of social engagement, have been shown. Abnormal interaction was explained by less positive maternal attachment, as well as difficult infant behavior, which impedes social involvement. Many of these interactive abnormalities reverted to normal by four months of age, but the need for "parenting training" is obvious.

OPIOIDS AND SUDDEN INFANT DEATH SYNDROME (CRIB DEATH)

Sudden infant death syndrome (SIDS) is defined as the sudden and unexpected death of an infant between one week and one year of age, whose death remains unexplained after a complete autopsy examination, full history, and a death-site investigation. Compared to an incidence of approximately 1.5 per 1,000 live births in the general population, narcotic-exposed infants appear to have an increased risk of SIDS. Other high-risk factors for SIDS, such as low socioeconomic status, low birthweight, young maternal age, black racial category, and maternal smoking are all overrepresented in the drug-using groups that are studied. In a most extensive study, New York City SIDS rates were calculated in 1.2 million births from 1979 to 1989. Maternal opiate use, after control for high-risk variables, increased the risk of SIDS by three to four times that of the general population.

LONG-TERM OUTCOME OF CHILDREN WHO HAVE UNDERGONE IN UTERO EXPOSURE TO OPIOIDS

Despite the fact that a drug-exposed newborn may seem free of physical, behavioral, or neurolog-

ical deficits at the time of birth, the effects of pharmacological agents (used or abused) may not become apparent for many months or years. Although heroin abuse during pregnancy has been recognized for more than forty years, and methadone treatment has been employed for more than twenty years, follow-up of opioid-exposed infants is still fragmentary. The difficulties encountered in long-term follow-up of this population include an inability to fully document a mother's drug intake, separation of the drug effects from high-risk obstetric variables, problems in maintaining a cohesive group of infants for study, and the need to separate drug effects from those of parenting and the home environment.

The easiest part of caring for the neonate is actually over when drug therapy has been discontinued and the infant is physically well. The most difficult parts then begin—the care involved in discharge planning and assuring optimal growth and development throughout infancy and childhood. Because there is no standard for the disposition of these infants, some may be released to their mothers, some to relatives, and others placed in the custody of a state agency. Still others may be voluntarily released by the mother to private agencies for temporary or permanent placement.

In the United States, pressure recommending separation of infants from their addicted mothers has been growing. This solution may not be practical in cities where social services and courts are already understaffed and overworked. Decent foster care is expensive and hard to find. Pediatricians basically feel that the mother-infant association should not be dissolved except in extreme situations. Aside from intensive drug rehabilitation and medical treatment, these women need extensive educational and job training—to become the productive citizens and loving mothers who will positively socialize their children. Supportive therapies such as outpatient care or residential treatment may help eliminate some of the medical and social problems experienced by drug-dependent women and their children.

Most of the children evaluated for long-term development have been exposed to methadone. Evaluations have occurred at various intervals—at six, twelve, eighteen, and twenty-four months; then at three, four, and five years of age. Testing procedures utilized have been the Gesell Developmental Schedule, the Bayley Scales of Infant Development, the McCarthy Scales of Infant Abilities, and the Stanford-Binet and the Wechsler Preschool and Primary Scale of Intelligence. Infants have shown overall developmental scores in the normal range but a decrease in scores at about two years of age—which suggests that environment may confound long-term infant outcome: low socioeconomic groups suffer from this factor particularly, because of poor language stimulation and development.

The developmental scores in these early years, although useful in identifying areas of strength and weakness, may not predict subsequent intellectual achievement. More and more studies have proposed multiple-factor models to assess infant outcome following intrauterine drug exposure. One such postnatal influence involves maternal–infant interaction. Drug-exposed infants are often irritable, have decreased rhythmic movements, and may display increased muscle tone (tensing) when handled. Such behaviors may be interpreted by the mother as "rejecting" behavior, leading to inappropriate maternal caretaking and possible neglect of the infant. Studies of mother—infant interactions show that: (1) infants born to narcotic-addicted women show deficient social responsiveness after birth; (2) this deficient mother—infant interaction persists until the infants' treatment for withdrawal is completed; and (3) maternal drug dosage may affect that interaction.

Based on available data, at five years of age, children born to women maintained on methadone, in contrast to heroin-exposed babies, appear to function within the normal range of their mental development. In addition, no differences in language and perceptual skills were observed between them and children of mothers not involved with drugs and of comparable backgrounds. Difficulty in following large cohorts of drug-exposed infants has led to the study of very limited samples, however.

Positive and reinforcing environmental influences can significantly improve drug-exposed infant development. Women who show a caring concern for their infants are most likely to pursue follow-up pediatric care and cooperate in neurobehavioral follow-up studies. Lacking a large data base, there is an obvious need for comprehensive studies assessing the development of large populations of drug-exposed infants.

COCAINE

The effects of maternal medical and obstetrical complications seen in opioid-exposed infants are similar to those of COCAINE exposure—although cocaine is a stimulant, not a depressant drug (like the opioids). The infants are frequently small in weight, length, and head circumference as a result of preterm birth and/or retardation of fetal growth. The effects of blood-vessel constriction, a characteristic pharmacologic effect of cocaine, is one of the main reasons for adverse effects—since it results in lack of oxygen and nutrients to the fetus. This predisposes the infant to growth problems, brain hemorrhage, abnormal organ development, and crib death.

The many studies on cocaine effects in the newborn need further clarification because of inadequate sample size, research methodology, and actual drug intake; these include studies that have evaluated brain hemorrhage, structural abnormalities, crib death, and long-term development. Although cocaine-exposed infants have been reported to have some irritability and perform poorly on neurobehavioral tests in the first few days of life, no evidence shows that they have a withdrawal syndrome as described previously in infants exposed to opioids. The symptoms have been related to a cocaine toxicity reaction rather than to a withdrawal syndrome. Infants with opioid *and* cocaine exposure, as compared to opioid exposure alone, have had milder symptoms. This may be a result of interactions between the depressant *and* stimulant properties of these drugs. No treatment has been found necessary to alleviate the symptoms of infants exposed to cocaine, whereas opioid-exposed infants may need treatment in about 40 to 50 percent of cases.

Although a number of reports in medical literature have described babies who have structural abnormalities related to cocaine exposure, an equal number of studies have found no increased incidence of abnormalities. The abnormalities reported have been those of the urinary tract, intestines, and extremities—all of which are related to the vascular disruption caused by cocaine's ability to constrict blood vessels. The most recent review of the clinical studies describing abnormalities in cocaine-exposed infants shows a very low incidence of occurrence.

Studies evaluating cocaine's effects on the occurrence of SIDS (crib death) have shown diverse results. Although inadequate methodologies and small numbers have accounted for these differences, cocaine-exposed infants have also experienced most of the factors that predispose any child to SIDS. These include low birthweight, POVERTY, neonatal complications, minority ethnicity, low maternal age, and maternal cigarette smoking. When these factors are controlled in the research, cocaine exposure accounts for only a very modest increase in the rate of SIDS.

As with all drugs of abuse, cocaine has properties that permit it to be transmitted through the breast milk. Since a significant portion of drug-using women in the United States may be HIV-positive, until the role of breast feeding in HIV transmission is clarified, breast feeding should be discouraged.

Recent reports indicate that cocaine exposure may even occur in young infants after they leave the hospital. The evidence for the postulated route of cocaine toxicity (passive inhalation of smoked cocaine—"crack") is circumstantial, and the range of occurrences in reported series is 2 to 4 percent. Symptoms involve abnormal neurologic findings, including seizures, drowsiness, and unsteady gait.

Much concern has been voiced regarding the ultimate neurobehavioral outcome of infants following intrauterine exposure to cocaine. Based on multiple-risk factors, it appears reasonable to voice these concerns. Commonly, the parents may be of poor socioeconomic status and culturally deprived. The mother may be poorly nourished, may carry medical and sexually transmitted diseases, including AIDS, and may receive little or no prenatal care. After birth, neurologic and neurobehavioral abnormalities may be present in the infant. Stimulation for intellectual growth may be lacking because of prolonged hospital stays, infrequent and inappropriate parental contact, placement in a group-care facility, or discharge to a home in which intellectual nurturing is lacking.

Follow-up studies of large numbers of cocaine-exposed babies are lacking as of the early 1990s. The lay press has reported anecdotal experiences with the first cohort of three- to five-year-old children born of the crack epidemic. Such cocaine-exposed babies have been characterized as showing significant deficits in environmental interactions during play groups and in nursery schools. These

babies have also been described as showing less representational play, decreased fantasy play and curious exploration, and lesser quality of play. Others have described these children as "joyless"—unable to fully participate in either structured or unstructured situations, with attention deficits and flat apathetic moods. Developmental evaluations show, however, that the majority of children who were exposed to cocaine in utero and who now have stable environments score in the normal range.

NICOTINE

Prenatal exposure to smoking has been linked with a number of impairments to the fetus, including impairments to memory, learning, cognition, and perception. Such impairments may result from chronic fetal hypoxia, a loss of oxygen to the cells that may impair normal development of the central nervous system. Maternal smoking during pregnancy also affects the respiratory system of a fetus, and newborns of smokers tend to have reductions in expiratory flows. It may also alter the developing lung and result in respiratory illness in the infant.

Low birth weight is another factor commonly associated with prenatal exposure to smoking, and even passive smoking—that is, from the father or another person in the vicinity of the mother—seems to affect an infant's weight. Some studies have shown an average decrease in birth weight of about 200 grams in newborns whose mothers smoked throughout pregnancy. The risk of a low-birth-weight infant has also been estimated to be two to four times greater for mothers who smoke. In general, women who stop smoking in pregnancy prevent the full effects of low birth weight associated with smoking, and studies have shown that the earlier a woman stops smoking during pregnancy, the lower the risk of a low-birth-weight baby. An infant's birth weight also appears to be "dose dependent," with heavy smokers being at the greatest risk for low-birth-weight babies.

Behavioral studies have also been conducted with children exposed to prenatal smoking. Some research has also shown that a child whose mother smoked during pregnancy is at increased risk of becoming a smoker. Because smoking activates neurotransmitters in the brain, including dopamine, which is involved in reinforcing the effects of addictive drugs, researchers have speculated that nicotine may have an effect on the developing dopamine system of the fetus and put the child at greater risk of addictive behavior in later life.

Prenatal exposure to cigarette smoking may affect a growing fetus in several ways. Carbon monoxide and high doses of nicotine obtained during inhalation of tobacco smoke can interfere with the oxygen supply to the fetus. Nicotine readily crosses the placenta, and it likely causes vasoconstriction of the umbilical arteries and impedes placental blood flow. Carbon monoxide can bind with hemoglobin to reduce the capacity of the blood to transport oxygen. These factors, combined, likely account for the developmental delays commonly seen in the fetuses and infants of smoking mothers.

One of the most striking risks associated with prenatal smoking is that of Sudden Infant Death Syndrome (SIDS). A higher mortality rate exists for infants whose mothers have smoked compared to those who have not. Maternal smoking during pregnancy has also been cited as a major risk factor in almost every epidemiologic study of SIDS. The risk of sudden infant death syndrome is greater among infants exposed to both prenatal and postnatal smoking compared to those only exposed to postnatal smoking. The increase in SIDS risk also appears to be related to the "dose" of passive-smoke exposure—the greater the exposure to smoke both before and after birth, the higher the risk of SIDS. The link between cigarette-smoke exposure and SIDS is not fully understood.

(SEE ALSO: *Complications: Route of Administration; Fetal Alcohol Syndrome; Pregnancy and Drug Dependence: Opioids*)

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FINANCIAL ANALYSIS IN ENFORCEMENT *See* Drug Laws: Financial Analysis in Enforcement

FLY AGARIC A poisonous mushroom of Eurasia (*Amanita muscaria*), having typically a bright red cap with white dots. A preparation, consisting primarily of the dried mushroom, is ingested by the people of Siberia as a HALLUCINOGEN. Intoxication by ingestion of several mushrooms moistened with milk or fruit juice leads to a progression of symptoms—beginning with tremors, continuing through a period of visual hallucination that may be interpreted as having religious significance, and finally ending in deep sleep. A similar preparation

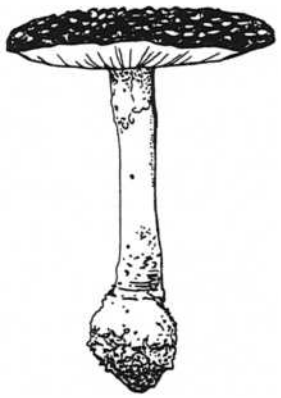

Figure 1
Fly Agaric

may be identified with the deified intoxicant *soma* of the ancient Hindus. In some cultures, the urine of intoxicated individuals is ingested by others to induce intoxication, since the active components of the preparation pass unmetabolized through the body.

The active components found in fly agaric are ibotenic acid and several of its metabolites. The predominant metabolite is muscimol, which has agonist properties at a subset of receptors recognizing the NEUROTRANSMITTER GABA. Ibotenic acid itself has agonist properties at certain excitatory amino acid receptors and has been shown to be neurotoxic.

(SEE ALSO: *Plants, Drugs from*)

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FOOD AND DRUG LAWS *See* U.S. Government: The Organization of U.S. Drug Policy

FOREIGN POLICY AND DRUGS Drug control is a relative newcomer to the list of global issues that are now an integral part of U.S. foreign policy. While arms control and human rights were already important international issues in the

1970s, drug control lagged behind. In 1971–1972 some members of Congress tried to use foreign-aid restrictions to stop the entry of Turkish HEROIN, but the government did not want to risk hurting relations with an important defense ally over heroin, which was not considered a mainstream drug. The U.S. government found a compromise through diplomatic efforts, which led to the Turkish government severely limiting the cultivation of OPIUM POPPIES (from which heroin is made) and changing the way in which poppies were processed into legitimate medicinal opium. Parallel diplomatic negotiations with MEXICO resulted in cooperation on MARIJUANA eradication efforts. On the international front, the U.S. government pressed hard for the ratification of the 1971 United Nations Convention on Psychotropic Drugs and created the United Nations Fund for Drug Abuse Control (UNFDAC), the predecessor of today's United Nations Drug Control Program (UNDCP). During the rest of the decade, however, drug control gradually declined as a key U.S. foreign policy objective.

Drug control only gained full diplomatic legitimacy in the 1980s when COCAINE use became widespread among entertainers, athletes, and stockbrokers. The government's inability to stop the EPIDEMIC at home prompted Congress to take the issue abroad.

In 1986, in the first of a series of comprehensive international antidrug laws (the Anti-Drug Abuse Act of 1986), Congress placed the burden of halting drug flows on the governments of the drug-producing countries. Using a traditional carrot-and-stick approach, the law required the major drug-producing and TRANSIT COUNTRIES to cooperate fully with the United States in drug matters in order to receive American foreign aid. Half of all assistance was withheld every year until the president certified that the country concerned had met the criteria for receiving aid. Subsequent laws have expanded the requirement, obliging the major drug-producing and transit countries also to comply with the 1988 United Nations Convention Against Illicit Traffic in Narcotics Drugs and Psychotropic Substances. Countries that do not comply not only lose U.S. assistance but incur U.S. opposition to loans from the World Bank and other international financial institutions. For many countries in the developing world, losing access to these loans is an even greater hardship than losing U.S. assistance. Though the certification process has raised tensions with some foreign governments, by 2000 it had become an accepted part of U.S. foreign policy. However, critics noted that the U.S. has recertified countries such as Mexico and Columbia, even when political corruption in these nations has seriously undercut narcotics enforcement efforts.

In earning its diplomatic legitimacy, drug control has had to overcome the same obstacles encountered by other global issues, such as human rights or nuclear nonproliferation. The U.S. foreign-policy establishment favors strategic issues affecting vital U.S. national-security or trade interests over law enforcement or scientific endeavor. It has been reluctant to allow multilateral "functional" questions to affect traditional bilateral negotiations. Congress, however, has left no doubt that it intends to keep drug control high on the list of U.S. foreign-policy issues. By denying virtually all forms of aid—excluding humanitarian and drug-control assistance—to countries that refuse to cooperate, Congress has devised an effective form of leverage over drug countries. Since the law also allows the president to waive sanctions when clearly stated national interests are at stake, Congress has made it difficult for foreign-policy agencies to evade their drug-control responsibilities.

RESPONSIBLE AGENCIES

The U.S. Department of State is responsible for formulating international drug policy. Its Bureau for International Narcotics and Law Enforcement Affairs oversees the annual certification process and prepares an annual report. Since 1989, formal coordination authority has rested with the White House Office of National Drug Control Policy (ONDCP) and the National Security Council. Drug control programs, however, involve a broad spectrum of government agencies including the Central Intelligence Agency, the Department of Defense, the U.S. CUSTOMS SERVICE, the Coast Guard, the Department of Treasury, the Justice Department, the DRUG ENFORCEMENT ADMINISTRATION, and the Department of Health and Human Services. A small percentage of the U.S. drug-control budget is spent on international programs. The bulk of the money goes to domestic law enforcement, drug treatment, and public education.

THE REALITIES OF DRUG CONTROL

As presidential administrations have discovered, an effective drug policy is easier to design than to carry out. The drug issue is a typical chicken-and-egg problem. Does supply drive demand or vice versa? The drug-consuming countries traditionally blame the suppliers for drug epidemics, while drug-producing countries allege that without foreign demand, local farmers would not be growing the drug crop at all. Planners must therefore strike the right balance between reducing drug supply and demand. In theory, eliminating drug cultivation in the source countries is the most economical solution, since it keeps drugs from entering the system and acquiring any value as a finished product. Few SOURCE-COUNTRY governments—all of which are in developing nations—will, however, deprive farmers of a livelihood without substantial compensation from abroad. And the price they seek is usually more than the U.S. government is prepared to pay.

THE NATURE OF THE THREAT

Today's illegal drug trade is one of the most lucrative, and therefore powerful, criminal enterprises in history. Drugs generate profits on a scale without historical precedent—especially given their abundance and low production costs. Such financial resources, which are well beyond those of most national budgets, give drug traffickers the means to buy sophisticated arms, aircraft, and electronic and technical equipment available to few countries. More importantly, illegal drug revenues allow trafficking organizations to buy themselves protection at almost every level of government in the drug-producing and drug-transit countries, where drug-related corruption remains the single largest obstacle to effective control programs.

As for the drugs themselves, there is a superabundance. Opium is in especially great supply. In Southeast Asia, Myanmar (formerly Burma) could supply the world's needs several times over with 257.5 metric tons annually. Estimates of heroin consumption in the Unites States range only between 6 and 20 metric tons, less than 10 percent of Myanmar's potential output. In South America, coca production dropped in the 1990s, yet it is enough to satisfy world demand twice over. This surplus is so large that the drug trade easily absorbs losses inflicted by drug-control authorities and still makes enormous profits.

Traffickers have the option of expanding cultivation of drug crops into new areas. For example, although coca plants are currently confined to Latin America, coca once flourished in Indonesia and could do so again if market conditions were right. Opium poppy cultivation is spreading into nontraditional areas, including South America. Gambling on the resurgence of expanding heroin use in the 1990s, South American cocaine-trafficking organizations have been diversifying into opium poppy cultivation. Without active government anti-drug programs, production will grow until the new expanding market is saturated.

CURRENT POLICY

The U.S. government's first priority is to stop the flow of cocaine, which still poses the most immediate threat to potential drug users. Because of rising heroin use promoted by the new, cheaper Latin American producers, the United States must also focus on opium-producing countries. The United States goal is to limit the cultivation of drug crops to the amount necessary for international medical applications. Since all the cocaine that enters the United States comes from coca plantations in Peru, Bolivia, and Colombia, the U.S. government has active drug-control programs in the three countries. During the 1990s, the U.S. has assisted Bolivia and Peru in their efforts to reduce coca cultivation. While these efforts have dramatically reduced production, drug traffickers increased coca production in Colombia. This resulted in increased political corruption and political destabilization. In 2000, the U.S. approved a $1.3 billion emergency assistance package to Colombia to help the Colombian government. The aid package contains money for police and military training, administration of justice programs, and economic development programs. The U.S. has also increased its military assistance to Latin America to help fight narcotics trafficking, yet many critics question the effectiveness of this approach. Others have expressed concern that direct U.S. military involvement may be requested by Colombia, which could lead to problems similar to those encountered by the U.S. in Southeast Asia in the 1960s and 1970s.

Opium control is more difficult than coca suppression, since most of the world's opium poppy

grows in countries where the United States has minimal diplomatic influence (Myanmar, Afghanistan, Laos, Iran, etc.). There also appears to be increasingly important opium poppy cultivation in China, Vietnam, and the Central Asian countries. Left unchecked, this opium expansion will make effective heroin control virtually impossible in drug-consuming countries, as Europe is already aware.

AN INTERNATIONAL APPROACH

Since bilateral programs seldom provide solutions to global problems, the United States has been an active proponent of collective action under the 1988 UN Convention. This latest agreement covers not only the traditional aspects of drug production and trafficking, but requires signatories to control drug-processing chemicals and outlaw drug-money laundering. The MONEY-LAUNDERING provisions are critical innovations, since they target the enormous international cash flows that sustain the drug trade. As astronomical as drug profits may be, drug money is useless unless it can enter the international banking system. The major industrialized countries are therefore pressing for uniform laws and regulations to exclude drug money in all key financial centers. If honestly implemented, strict money-laundering controls, along with better use of existing programs to suppress drug supply and decrease consumption, offer the hope of reducing the drug trade from an international threat to a manageable concern.

(SEE ALSO: *Crop-Control Policies; Drug Interdiction; Drug Laws: Financial Analysis in Enforcement; Golden Triangle as Drug Source; International Drug Supply Systems; Opioids and Opioid Control: History; Terrorism and Drugs; U.S. Government Agencies*)

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FORFEITURE LAWS *See* Legal Regulation of Drugs and Alcohol; Mandatory Sentencing

FREEBASING The illicit practice of smoking COCAINE is generally referred to as freebasing. The hydrochloride form of cocaine (powder) is highly soluble in water and, therefore, is efficiently absorbed by the mucous membranes when taken intranasally (snorted) or via blood when injected intravenously (shot up). This form of cocaine is, however, destroyed when it is heated to the temperatures required for smoking it. Therefore, the cocaine alkaloid, called "CRACK" or "freebase," is the form that is smoked. Although not always differentiated, freebase actually refers to cocaine in the base state with all the adulterants removed (Inciardi, 1991). Cocaine hydrochloride is combined with an alkaline substance, such as sodium hydroxide or ammonia, to remove the hydrochloride. The *free* cocaine *base* is then dissolved in ether, and pure cocaine-base crystals are formed. It has been estimated that approximately 560 milligrams of cocaine freebase can be extracted from one gram of street cocaine hydrochloride (Siegel, 1982). Cocaine freebase has a melting point of 208°F (98°C) and is volatile at temperatures above 194°F (90°C), therefore providing an active drug for smoking. Crack, in contrast, although also in the base state and used for smoking (or freebasing), does not have the adulterants of the street cocaine removed. Cocaine base is soluble in alcohol, acetone, oils, and ether—but is almost insoluble in water.

Cocaine freebase is usually smoked in a water pipe containing fine mesh screens, which trap the heated cocaine as it melts. A temperature of 200°F (93°C) is the most efficient. Although the amount of cocaine absorbed by the smoker varies—depending on the kind of pipe used, the temperature of the heat source, and the inhalation pattern of the user—under optimal conditions approximately 30 to 35 percent of the cocaine placed on the mesh screen is absorbed by the smoker.

COMPARISON OF COCAINE AND METHAMPHETAMINE SMOKING

Vapor inhalation of the (+) isomer of methamphetamine hydrochloride, colloquially known as *ice* has several differences when compared to vapor inhalation of cocaine freebase. Although both methamphetamine and cocaine freebase have their origin as a salt, cocaine hydrochloride must be pretreated with an alkaline substance to remove the hydrochloride, thus creating the freebase of cocaine that can be heated and inhaled as vapor. In contrast, methamphetamine hydrochloride can be heated and inhaled without adulterating the original compound.

When heated, cocaine freebase has a melting temperature of 208°F while methamphetamine hydrochloride melts at 268°F. Once the appropriate melting temperature is met for each substance, vapors will form and can be inhaled. Significant amounts of cocaine freebase vapor are lost through pyrolysis (chemical change caused by heat) and little condensation appears on the water pipe, suggesting decreased amounts of inhaled vapor. Methamphetamine hydrochloride, however, condenses as a crystalline solid on the cooler areas of the glass pipe. It is thought that this same phenomenon occurs in the mouth and throat of the user, leading to rapid methamphetamine absorption through the lungs as well as delayed absorption through the oral mucosa.

These differences in drug absorption have been demonstrated by comparisons of plasma levels of cocaine and methamphetamine after smoking the individual substances. Plasma levels of cocaine peak and decline rapidly, with a half-life of approximately forty-five to sixty minutes. Methamphetamine plasma levels also rise rapidly, but the half-life is approximately eight to twelve hours. The delayed absorption of methamphetamine from the oral mucosa is thought to play a role in the extended half-life. Differences in the metabolism of cocaine and methamphetamine also contribute to the disparity in plasma half-life. Cocaine is quickly degraded to inactive metabolites by plasma esterases (enzymes) and cleared from the bloodstream. Methamphetamine is eliminated by enzymes with limited plasma distribution and limited activity and, unlike cocaine, is converted to active metabolites that prolong the action of the drug. These active metabolites can accumulate, and repeated smoking of methamphetamine and its active metabolites can lead to dangerous levels of methamphetamine in the plasma.

In summary, differences between cocaine freebase vapor inhalation and methamphetamine hydrochloride inhalation include method of preparing the substance, melting temperature, metabolism, and length of plasma half-life. These differences can have important clinical implications. For example, methamphetamine can cause paranoid symptoms that last considerably longer than those ordinarily seen after cocaine smoking. Distinguishing between drug-induced paranoia and other causes of paranoia thus requires a different length of drug-free observation depending on which drug was inhaled. Understanding the differences between cocaine freebase inhalation and methamphetamine inhalation, particularly the difference in duration of action of the two drugs, can be important in the evaluation and management of patients with stimulant abuse.

Although in use since the mid-1970s, freebasing cocaine became popular in the United States in the early 1980s. The popularity of this route of administration was responsible for the rise in U.S. cocaine use during the mid-1980s. When cocaine is smoked, it is rapidly absorbed and reaches the brain within a few seconds. Thus, users get a substantial immediate rush and an almost instant "high," comparable to that after intravenous cocaine. This is in contrast to intranasal use of cocaine, which engenders a high with a much slower onset. Freebasing is thus a convenient way of taking cocaine, with the possibility of repeated and substantial doses. Since the likelihood of abuse is related to the rapidity with which a drug reaches the brain, smoking cocaine makes it more likely that use will lead to abuse than does snorting the drug. Despite losses of more than half of the cocaine when it is smoked, sufficient cocaine rapidly

reaches the brain, providing an intense drug effect—which users repeat, often to toxicity. The danger of freebasing, in addition to the inherent danger of cocaine use, lies in what some users perceive to be the greater social acceptability of a route of administration that requires minimal PARAPHERNALIA and can achieve toxic levels of cocaine with relative ease.

(SEE ALSO: *Amphetamine Epidemics; Coca Paste; Complications: Cardiovascular System; Methamphetamine; Pharmacokinetics*)

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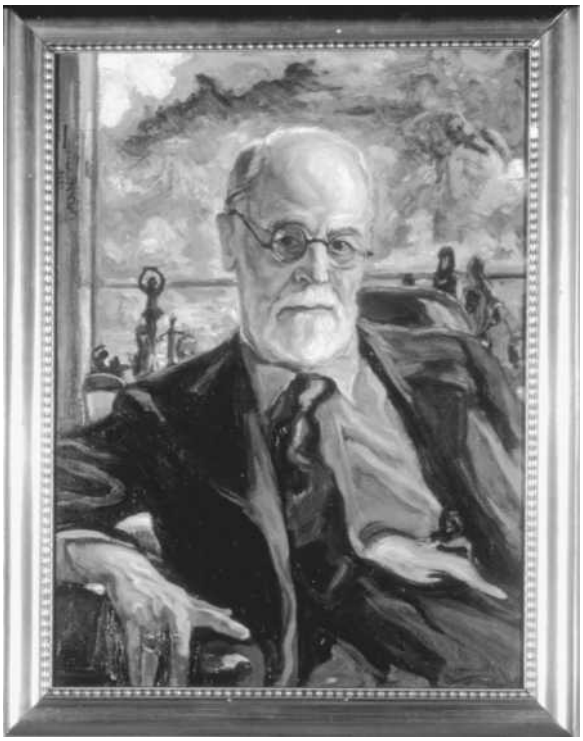
FREE WILL *See* Disease Concept of Alcoholism and Drug Abuse; Values and Beliefs: Existential Models of Addiction

FRENCH CONNECTION *See* Drug Interdiction; International Drug Supply Systems

FREUD AND COCAINE Sigmund Freud (1856–1939), Austrian neurologist and founder of



Sigmund Freud performed a number of cocaine experiments on himself and reported the results in his Contribution to Knowledge of the Effects of Cocaine. (The Library of Congress)

PSYCHOANALYSIS, became interested in COCAINE in the early 1880s. At the time he was in his late twenties and was a medical house officer at the Vienna hospital called the Allgemeine Krankenhaus. He was able both to gain access to the literature about cocaine and, at some expense, to the substance itself (which was not illegal at that time). There had been articles in the American medical literature describing cocaine used in the treatment of various ills and for drug dependencies as almost a panacea. The ability of cocaine to fend off fatigue and enhance mood also came to Freud's attention. He was particularly taken by suggestions that cocaine might be an adjunct to, or even a cure for, ALCOHOL or OPIOID dependencies. His interest was heightened because one of his close teachers and friends, Ernst von Fleischl-Marxow, had become an opiate addict. Using cocaine, Freud treated him with almost disastrous results. At the time, there was no opprobrium attached to the use of cocaine and relatively little concern about any adverse effects.

Freud performed a number of cocaine experiments on himself and reported the results in his experimental paper, "Contribution to Knowledge of the Effects of Cocaine." These were reasonable studies that provided useful data about the physiological and psychological effects of cocaine. Biographies of Freud, such as Ernest Jones's *The Life and Work of Sigmund Freud,* have tended to disparage his experimental paper and other works on cocaine. Although his work was done on himself and was limited in its scope, it has been confirmed in modern replications. Freud was initially skeptical about the possible "addictive" properties of cocaine in normal individuals, but later, in the face of evidence and criticism, he was less vehement on the subject. He became, in later life, very sensitive to criticism of his earlier views on cocaine.

From 1884 to 1887 Freud wrote four papers concerning cocaine, including a definitive review ("Über Coca") in 1884. He obviously felt comfortable in both taking cocaine and writing about it in his letters. He mentions and discusses his use of and dreams about cocaine in the *Interpretation of Dreams* (1889). The true extent and duration of his self-experiments is not known, since access to his correspondence has been severely restricted.

Freud is sometimes credited with the discovery of local anesthesia because of his proposal in his cocaine review paper that the substance could be used for this purpose. He also claims suggesting the idea to both Koenigstein and Carl Koller prior to their experiments in ophthalmology, which led to the initial papers on local or topical anesthesia. There is a semantic problem in understanding these claims. Almost all investigators of cocaine had noticed the numbing properties of the drug when placed on the tongue. The idea that this property had a practical use in ophthalmological surgery does belong to Carl Koller, a friend and colleague of Freud's, who did the proper experiments and published them promptly. The controversy about the discovery between Koller and Koenigstein with Freud's mediation is well covered in the article by Hortense Koller Becker, "Carl Koller and Cocaine," in *Psychoanalytic Quarterly*.

Extreme viewpoints that attribute Freud's behavior and writings to the influence of the toxic effects of cocaine are unsubstantiated by evidence. Clearly, he used cocaine as a psychotropic agent on himself and this experience led to his faith in its relative safety. Despite this, there is no real support for a viewpoint that he was an addict or that his thought was markedly affected by his drug usage. The combined notoriety of both Freud and cocaine has led to speculative exaggerations that make better newspaper headlines than history.

(SEE ALSO: *Abuse Liability of Drugs; Epidemics of Drug Abuse; Pharmacotherapy*)

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ROBERT BYCK

G

GABA *See* Gamma-Aminobutyric Acid

GAMBLING ADDICTION: ASSESSMENT With the legalization and spread of gambling across North America over the last twenty years of the twentieth century, problem gambling emerged from out of the shadows into the mainstream of serious personal and social problems.

BEGINNING OF TREATMENT

In the United States, the first organized program to deal with problem gambling occurred in 1957 with the founding of Gamblers Anonymous, a self-help/mutual support program. The first professional treatment program for compulsive gamblers was begun in 1972 by a psychiatrist, Robert Custer, in an inpatient alcohol program in a Veterans Administration hospital. The first state funded treatment program for compulsive gamblers began in Maryland in 1978.

ASSESSMENT AND TERMINOLOGY

Gamblers Anonymous developed 20 screening questions to help individuals decide whether they are compulsive gamblers. This questionnaire was the primary instrument utilized by professionals until 1980, when the mental health establishment recognized a gambling problem as a psychiatric disorder, naming it pathological gambling. Diagnostic criteria for this disorder were specified in the Diagnostic and Statistical Manual (DSM III) used by mental health and addiction clinicians (American Psychiatric Association, 1980). The most widely used term in society referring to this disorder is still compulsive gambling, while the terms addictive, chronic and disordered gambling are also currently in use. The term *problem* gambling is used generically to refer to an unspecified level of severity and is also used in an assessment context to refer to a gambling problem of mild to moderate severity, encompassing those at risk for developing pathological gambling.

DSM IV DIAGNOSIS

The diagnostic criteria were modified in DSM III-R (American Psychiatric Association, 1987) and in DSM IV (American Psychiatric Association, 1994). The diagnostic criteria for pathological gambling in DSM IV are provided below.

A. Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following:
   1. is preoccupied with gambling, e.g., preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble

2. needs to gamble with increasing amounts of money in order to achieve the desired excitement
3. has repeated unsuccessful efforts to control, cut back, or stop gambling
4. is restless or irritable when attempting to cut down or stop gambling
5. gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)
6. after losing money gambling, often returns another day to get even ("chasing" one's losses)
7. lies to family members, therapist, or others to conceal the extent of involvement with gambling
8. has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling
9. has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling
10. relies on others to provide money to relieve a desperate financial situation caused by gambling

B. The gambling behavior is not better accounted for by a Manic Episode.

Key features in these criteria include: obsessive preoccupation (including craving); progressive inability to control all aspects of gambling; and continuation of gambling despite increasing negative consequences of gambling.

To assist certified clinicians who are not experts in pathological gambling in making a reliable diagnosis of pathological gambling, several DSM IV based structured interviews have been developed but no validation studies have been reported.

It would be clinically useful to include in the future revision of DSM the less severe category of gambling abuse to parallel the current substance abuse diagnostic categories in DSM IV.

DEVELOPMENT OF SCREENING INSTRUMENTS

Pathological gambling is a progressive disorder with very serious life consequences at the later stages. Early identification is especially important because of the devastating individual, family and social impacts of high rates of bankruptcy, suicide, and crime and other individual and societal problems related to pathological gambling (Blaszczynski, et al., 1989; Lesieur, 1998; Phillips, et al., 1997).

The first valid and reliable screening instrument for pathological gambling was the South Oaks Gambling Screen (SOGS) developed in 1987 and still the primary instrument in the field for clinical screening and prevalence research (Lesieur & Blume, 1987). As the SOGS has twenty items, there is a need for a briefer screening instrument which is rapidly scorable. Screening instruments which have been developed to assess problem and pathological gambling in youth are the MAGS (Shaffer, et al., 1994), DSM IV-J (Fisher, 1992) and SOGS-RA (Winters, et al., 1993). Self-report instruments are useful for self-screening and initial professional screening but are not to be used for diagnostic purposes.

ASSESSMENT OF THE FAMILY SYSTEM

In addition to conducting an assessment of the gambler in the clinical context, assessment of other key family members is important for the following reasons (Steinberg, 1993):

- Identification of current and imminent crises.
- Orientation of family members to the treatment setting in preparation for potential involvement in the process.
- Gaining the perspective of significant others provides a more accurate picture of the nature and extent of the gambler's problem.
- Observation of family dynamics provides a clearer understanding of family deficits and strengths.
- Opening an avenue of communication with family members provides earlier detection of signs of relapse.
- It increases the likelihood of help for the family even if the gambler drops out of treatment.
- The impact of the gambling on children in the family can be better determined.

PROGRESSION OF THE DISORDER

Assessment of problem gamblers in less advanced stages is more difficult. Increased public awareness of the signs of pathological gambling

coupled with more human services professionals receiving training in the disorder is resulting in detection of a gambling disorder early in its progression. Instruments that identify the degree of current problem and risk for developing pathological gambling are still needed.

Custer and Milt (1985) identified in clinical practice three stages in the progression of a gambling disorder (for almost exclusively male action gamblers).

Winning stage: Characterized by an initial large win.

Losing stage: Losses are chased with increased gambling until a major problem occurs which is temporarily resolved by a financial bailout, followed by a higher level of gambling and increased crises.

Desperation stage: The gambler further withdraws from family and work responsibilities into gambling, often resulting in criminal and suicidal behavior. Help may or may not be sought.

Hopelessness stage: Rosenthal added the fourth stage for some gamblers who no longer care and continue to gamble without hope of winning. Custer's (1985) chart below depicts the progression and recovery cycle for those who seek help.

PATHOLOGICAL GAMBLING, SUBSTANCE DEPENDENCE, AND OTHER CO-MORBID DISORDERS

While pathological gambling is classified as an impulse disorder, it is increasingly viewed as part of the family of addictions. In fact, the criteria for pathological gambling in DSM III-R were modeled after the criteria for psychoactive substance dependence in DSM III-R. The DSM IV criteria for problem gambling blend DSM III and DSM III-R criteria. There is increasing clinical research evidence for sequential and simultaneous dual addictions involving gambling and substances e.g., alcohol, cocaine, tobacco (Lesieur & Blume, 1996). Brain chemistry research and preliminary genetic research have both pointed to biochemical and etiological commonalities for pathological gambling and substance dependence. While not as extensively researched, relationships have also been found between pathological gambling and food, sex, and work addictions. Co-morbidity has also been found between pathological gambling and other psychiatric disorders, including clinical depression and other mood disorders, anxiety, attention deficit hyperactivity disorder (ADHD), and personality disorders (Blaszczynski & Steel, 1998; Carlton, et al., 1987; McCormick, et al., 1984).

A theory is developing which places pathological gambling in a compulsive-impulsive spectrum with problem gambling as one of the impulse (ego syntonic) disorders at one end of the spectrum and obsessive-compulsive disorders (ego dystonic) at the other end (Cartwright, et al., 1998). Different degrees of impulsivity and compulsivity are experienced by pathological gamblers, depending upon the stage of the development of the disorder with impulsivity primarily at the early stage and growing compulsion at the later stage.

MULTIPLE CONTRIBUTING CAUSATIVE FACTORS

As the twenty first century begins, there is not widespread agreement as to the exact cause(s) of pathological gambling. However, as with many other disorders, a broad model is emerging which includes four major areas of risk factors for developing this disorder: biological, social, psychological, and spiritual (Rugle, 1993).

Biological. Genetic research in the late 1990s has provided preliminary evidence of a genetic link among pathological gambling and other addictive and impulse control disorders (Comings, 1998). This is mediated by neurotransmitters which control impulsivity, emotion and the experience of pleasure. Advances in brain imaging in the late 1990s began to identify areas of deficit in brain functioning which are related to deficits of behavior functioning (e.g. attention deficit hyperactivity disorder [ADHD] (Cartwright, et al., 1998)).

Social/Environmental. Research has provided evidence that early environmental factors in the home such as exposure to a parents excessive gambling or abuse is linked to a later gambling problem. Further, it is likely that trauma in adulthood, including losses later in life, increase vulnerability to developing a gambling problem. Such environmental factors as proximity to gambling, widespread gambling advertisements and the absence of significant education about responsible gambling and the warning signs of problem gam-

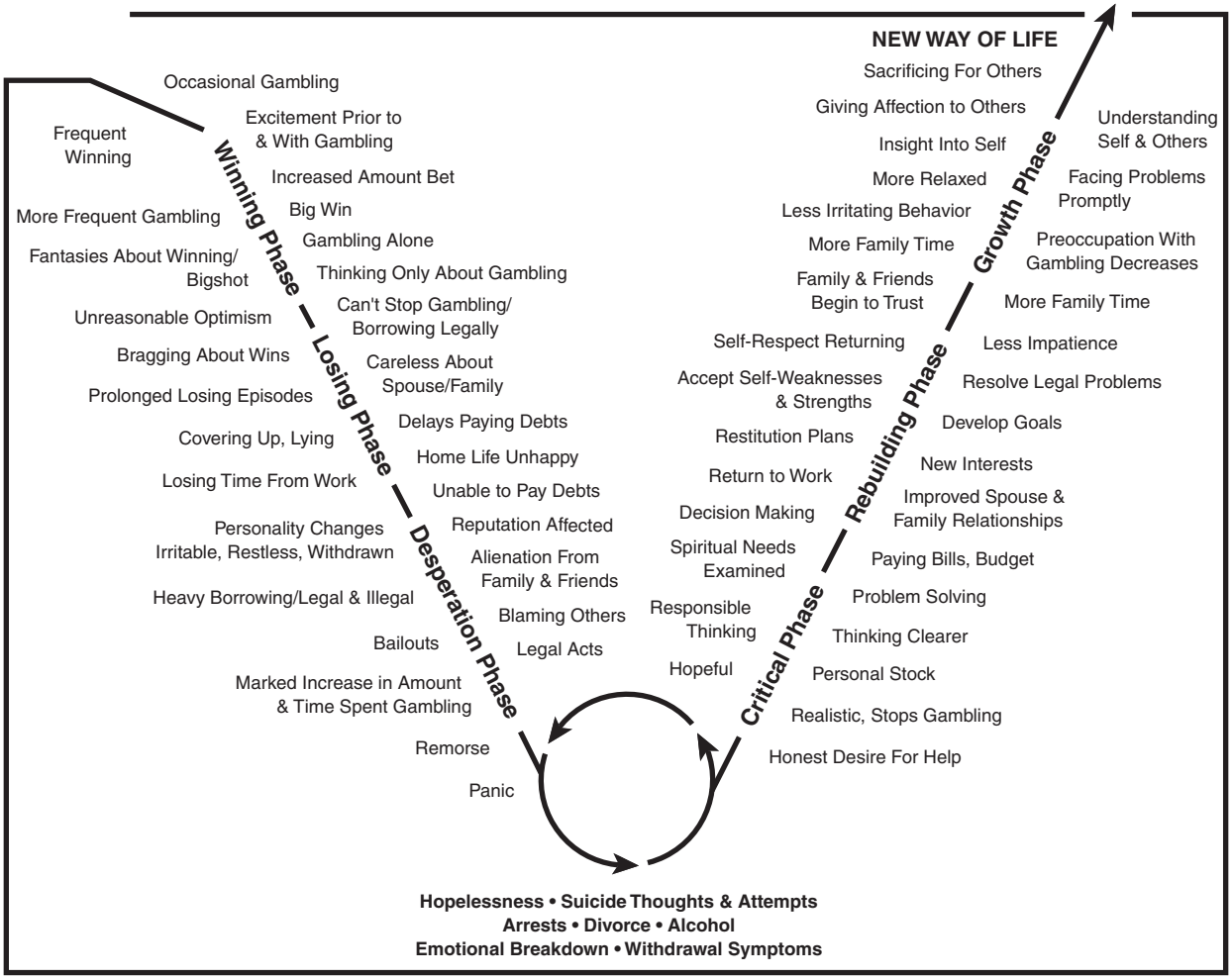

Figure 1
A chart of compulsive gambling and recovery.

bling are likely contributors to higher prevalence rates in certain communities.

Psychological. Recognizable differences between pathological gamblers and non-pathological gamblers have been identified in personality patterns (low frustration tolerance, self-centeredness, mood changes), dissociation and fantasy, as well as irrational and magical thinking. Gender differences have been linked to choice of gambling activities. Men tend to more often be "action" gamblers seeking competition and games of skill (e.g., cards, sports) and women are more likely to be "escape" gamblers seeking solitary and non-competitive activity (e.g., electronic gaming machines).

Spiritual. The 12-Step Recovery programs of Gamblers Anonymous and Gam-Anon, for family members of addicted gamblers (patterned after Alcoholics Anonymous and Al-Anon) attempt to bolster the recovery process by searching for and relying on a higher power to give new meaning to life. Addictions, including pathological gambling, involve substitution of quick fix activities for intimate relations and a spiritual life.

While it has become clearer that the above factors increase the risk of developing a gambling problem, progress toward the development of valid and reliable measures of these factors is evolving slowly but with a quickening pace.

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MARVIN A. STEINBERG

GAMBLING ADDICTION: EPIDEMIOLOGY GAMBLING is a form of risk taking that may be defined as risking (betting or wagering) something of monetary value on the unknown outcome of a future event in order to gain something else of monetary value. Evidence of gambling has been found in early civilizations as well as throughout history. For example, many references to gambling can be found within the Old and New Testaments. However, as with ALCOHOL, wide cross-cultural differences have existed in the degree of acceptability and extent to which gambling is integrated into a culture.

RAPID GROWTH OF LEGALIZED GAMBLING

Historically, gambling in the United States had not been integrated into the larger culture as a major legal recreational pastime until the largest continuous expansion of legalized gambling that began during the last quarter of the twentieth century. Gross gambling revenues dramatically increased in the 1990s. For example, in 1996, $47.6 billion in revenues surpassed the $40.8 billion of combined revenues from movies, recorded music, cruise ships, live entertainment, and spectator sports (Christiansen, 1998). In the 1990s, there

were major increases in the availability of some forms of gambling (casino and lottery) as well as new locations (riverboats and Native American reservations), many of which were immediately accessible (convenience stores). By the end of the twentieth century, gambling in the public mind had moved away from being associated with immorality, personal deviance, and crime and had become a major socially acceptable form of entertainment. At the turn of the twentieth-first century, lottery and casino gambling are the prominent forms of legal gambling in the United States, and there is no indication that this trend is slowing down.

The factors contributing to increased gambling include the perceived need by governments for lottery revenue to avoid raising taxes and to stimulate economic growth in distressed areas. Also contributing are the efforts of gambling entrepreneurs in the private sector and the simultaneous development of new forms of gambling technology, principally electronic gaming devices.

The private gaming industry and state governments trumpet gambling as exciting entertainment that also brings the benefits of more jobs and lower taxes. However, gambling is not solely a societal plus. When gambling is legalized and made more accessible, the number of people who try it increases and a certain percentage of those new gamblers who are vulnerable to addiction develop a problem. Social costs of pathological gambling, such as addiction, bankruptcy, divorce, and crime have been found to be severe in clinical samples of pathological gamblers. Assessment on a large scale of these social costs has only just begun (Lesieur, 1998).

PREVALENCE OF PROBLEM GAMING

The first national study of gambling and problem gambling in 1974 indicated that .77 percent of the sample had at some time in their lifetime been probable pathological gamblers, with another 2.33 percent potential compulsive gamblers (University of Michigan Survey Research Center, 1976). The second federally supported national study was conducted by the National Gambling Impact Study Commission (NGISC) in 1999. It was found that 1.20 percent (2.5 million) of the adult population were probable pathological gamblers in their lifetime and 1.50 percent (3 million) were lifetime problem gamblers (National Research Council, 1999). An additional fifteen million adults were identified as being at risk for developing a gambling problem. As compared to these statistics for the telephone sample where a total of 2.70 percent were lifetime problem and pathological gamblers, this study also surveyed patrons at gambling facilities (regular gamblers) and found that 13.00 percent met criteria for lifetime problem and pathological gambling. The NGISC report estimated that the annual cost for problem and pathological gambling is $5 billion, plus $40 billion in lifetime costs associated with decreased productivity, social service costs, and creditor losses (Gersten et al., 1999).

The prevalence of gambling problems is affected by many factors including the number of legal (and illegal) forms of gambling that are available and accessible. Prevalence rates may also be affected by the increasing availability of forms of electronic gaming. These machines are intrinsically engaging and even mesmerizing for many people. This form of gambling involves an insulated person–machine interaction, which provides the opportunity for more frequent play and reinforcement than other forms of gambling. For the individual susceptible to a gambling addiction, the time in which addiction may occur is foreshortened, especially when such machines are available 24 hours a day. However, any form of gambling may result in addiction for an individual who is vulnerable.

Higher prevalence rates for problem gambling are also likely to result when there is an increased acceptance in society of financial risk taking as gambling. In today's financial world financial resources are gambled away in ways that have not been traditionally considered forms of gambling. For example, excessive and destructive risks are being taken in the business world by pathological gamblers who are not aware that they are acting out a gambling problem. Slightly greater awareness is developing that the stockmarket and other financial markets are also arenas for problem gambling. Despite the fact that most people invest prudently in the financial markets, enormous sums are gambled daily in the markets. In 1997, the United States Securities and Exchange Commission acknowledged for the first time that problem gambling occurs in the financial markets by way of its agreement to distribute a pamphlet on investor problem gambling (Connecticut Council on Prob-

lem Gambling, 1997). However, the brokerage industry has not yet acknowledged problem gambling as a concern.

An additional fact that may influence the prevalence rate of problem gambling is the dramatic increase of accessibility to gambling via the Internet. The number of gambling sites available and number of online gamblers have been increasing rapidly, as indicated by the more than doubling of Internet gambling revenues from $445.4 million in 1997 to $919.1 million in 1998 (Barry, 1998). People at risk for a gambling problem will find it more difficult to avoid gambling and youth will be further tempted with increased accessibility by way of home computers. Even if the federal bill in the year 2000 to prohibit Internet gambling in the United States is enacted, online gambling will still be available on the Internet emanating from many other locations. Regardless of the legal status of Internet gambling, Intranet gambling sites involving *pari-mutuel* wagering will continue to be available to subscribers on home televisions and computers.

PREDISPOSING FACTORS

The National Opinion Research Center (NGISC, 1999) reviewed the available literature on problem gambling and concluded that the following are major predisposing factors to the development of a gambling problem:

- Problem gambling often occurs jointly with substance abuse, mood disorders, and personality disorders.
- Pathological gamblers more often than nonpathological gamblers report having a parent who is a pathological gambler.
- The earlier gambling starts, the more likely pathological gambling will occur.

SUBGROUPS AT RISK

The identification and modification of risk factors have been hampered by the confusion from the mixed messages the public receives. On the one hand, private and government sponsers of gambling on a large scale encourage gambling. On the other hand, consumers receive strong but less frequent messages that gambling to excess or inappropriate gambling can create addiction and related negative life consequences.

Evidence suggests that certain groups are at risk, such as older people, youth, women, and people with low income. Seniors are gambling more frequently and they are one of the major groups being targeted by casinos in their promotional efforts. There is building evidence that people of low income gamble a higher percent of their income than people with higher income. The rate of problem gambling among women appears to have dramatically increased in the 1990s, growing from a small percentage to more than 25 percent of all identified problem gamblers. Statewide prevalence studies have consistently identified teenagers as having a greater number of problem gamblers than adults in the same states (National Research Council, 1999).

These emerging facts raise many more questions that need to be investigated. For example, although seniors are a vulnerable group because of declining physical heath and mental capacity as well as depression due to loss and isolation, it is not known whether seniors as a whole experience a greater rate of problem gambling than adults in general. Perhaps the social contact available in a gambling environment and the alertness and required in concentration on gambling have positive mental health benefits for seniors? Research with subgroups in the population, especially groups at risk, across a wide range of geographical areas is needed.

GROWTH OF COUNCILS ON PROBLEM GAMBLING

To meet the challenges of problem gambling, which have increased with the growth of gambling in the last quarter of the twentieth century, councils on problem gambling have been created in the United States and Canada. As spiritual advisor to Gamblers Anonymous, Monsignor Joseph Dunne, along with recovering compulsive gamblers and family members, founded the National Council on Problem Gambling (NCPG) in 1972. Connecticut became the first state affiliate of the NCPG in 1980 and by 2000 there were thirty-four state affiliate councils. The NCPG was the first professional organization to educate the public about compulsive gambling as a serious public health problem and to advocate for treatment services. Other major priorities of the NCPG and its affiliates include the following: sponsoring helplines, conducting prevention programs, training human services person-

nel, conducting surveys on problem gambling, and collaborating with a variety of relevant organizations, including the public and private gaming industry.

GAMING INDUSTRY'S RESPONSIBLE GAMBLING PROGRAMS

The American Gaming Association (AGA), the national trade association for casinos, in 1996 took a major voluntary step forward in creating the Responsible Gaming Resource Guide, which provided a blueprint for establishing a responsible gaming program. In 1997, AGA also established the National Center for Responsible Gaming that is a significant funder of basic research into problem gambling (American Gaming Association, 1998). In the late 1990s, a few state gaming regulatory bodies began to require responsible gaming programs in order for private sector gambling operators to be licensed. Native American-owned casinos have also developed innovative responsible gambling programs. Although most state lotteries have responsible play programs in the year 2000, government efforts to promote responsible gambling (with a few exceptions) are not as progressive as those of the private sector. This may be due to the inherent difficulty of serving as both the regulator and operator of the lottery or because the lottery is incorrectly viewed as a relatively benign form of gambling. Funding for treatment, prevention and research programs by state governments began gradually in the late 1970s and by the end of the twentieth century approximately half the fifty states funded significant programs.

RECOMMENDATIONS OF THE NGISC

The NGISC's (1999) two-year examination of gambling in the United States has been the most extensive and systematic study of the state of seventy-four recommendations for changes in policies and practices for the public and private and Native American sectors of the gambling industry, state regulators, and the federal government. Some of the major recommendations include:

- A pause in the processing of all new gambling applications to allow for adequate assessment of the gambling already in place
- A rollback of all convenience gambling in communities and a halt to authorization of all new convenience gambling
- A restriction of the minimum legal gambling age to 21
- A ban on betting on collegiate and amateur athletics
- A ban on all aggressive gambling advertisements, and the creation of responsible gambling advertisement guidelines
- Prohibition of Internet gambling not already authorized
- A ban on ATM and credit card machines within or near the immediate gambling area
- Gambling establishments policies to ensure the safety of children and prevent underage gambling
- School programs from the elementary through college level should include warning of the dangers of gambling

NEED FOR A COMPREHENSIVE PLAN

Few epidemiological studies have been undertaken primarily due to an underestimation of gambling's impact on all levels of the community. Most communities lack a comprehensive approach and systematic methodology to determine the overall value of gambling to the community. Given the rush to profit from the popularity of gambling, most state and local governments have not systematically planned (e.g., articulated-short and long-term goals) and conducted a comprehensive study of the likely impact of new or significantly expanded gambling on their communities. Economic projections and gambling regulation have been the primary interests. Consequences can be enormous if initial assessments are not comprehensive, thorough, and accurate. Once gambling is introduced, it is very difficult to roll it back as governments become highly dependent on the revenue. Further, evaluation and monitoring programs have typically not been set up to assess the impact of gambling on communities over time. Needed are the short- and long-term assessment of social costs, the extent to which projected economic benefits have been met and sustained, and the extent to which gambling has changed the communities in other positive and negative ways (NGISC, 1999).

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Marvin A. Steinberg

GAMBLING AS AN ADDICTION Human beings have indulged in games of chance since before recorded history. Archeological sites in both the Old World and the New World yield gambling bones, dice, and counters. The Old and New Testaments mention the casting of lots to determine the distribution of property, presumably as an expression of God's will. In addition, the classical literature of both Eastern and Western cultures includes many accounts of gambling, often with dramatic consequences. Lotteries have been popular in Asia and Europe for centuries. The first European government-sponsored lottery was established by Queen Elizabeth I in sixteenth-century England. The thirteen American colonies and the early American universities—including Harvard, Yale, Princeton, and Columbia—were all supported in part by lotteries.

Gamblers play the slot machines at the Casino Sandia, a gaming facility at the Sandia Pueblo north of Albuquerque, New Mexico, January 10, 1997. (© Miguel Gandert/CORBIS)

Most societies have recognized the popularity of gambling and its potential for generating social good and personal harm. Therefore, governments have sought ways to regulate gambling. Some governments have prohibited all gambling, while others have established laws limiting the availability of gambling to particular locations, establishing a minimum age, specifying types of games allowed, and regulating the gaming industry to prevent fraud and raise revenues. In the United States, government attitudes toward legalizing gambling have changed radically over time. By the mid-twentieth century, some state governments increasingly looked to state lotteries as a fertile source of revenues. In addition, casino and riverboat gambling, sports betting, card rooms, and bingo games were variously legalized, taxed, and regulated. By 1994, some form of gambling was legal in all states but Hawaii and Utah, and several American Indian nations were operating gambling establishments on tribal land. In 1997, an estimated 639 billion dollars was wagered annually in the United States, generating a profit of more than 41 billion dollars—with the vast majority of the total legally bet. Illegal gambling has its own special set of subcultures—with rules, limits, and penalties for its devotees.

With the increases in gambling have come mounting concerns about gambling-related personal and social harm. In 1997, the President and

Congress appointed a National Gambling Impact Study Commission to analyze both the positive and negative impacts of gambling in the United States, and to recommend policy initiatives. The Commission made its report in 1999, estimating a five billion dollar cost to American society. Among its many recommendations were freezing or reducing so-called *convenience gambling* (video gambling machines in retail outlets or taverns), and banning gambling on the Internet. In regard to problem gambling, the report called for more treatment, better health insurance coverage and, state funding for treatment, more and better efforts at prevention, and an increased investment in research.

For most people gambling is a pleasurable, if not very profitable, occasional recreation. For a significant minority, however, gambling has the potential to become a compulsive behavior and a ruinous destructive problem. Compulsive gambling has also been known for centuries. The classic Hindu epic, *The Mahabharata*, tells the story of a wise and just king whose single flaw, the inability to control his gambling, leads him to gamble away his wealth and kingdom in a dice game. Still unable to stop, he gambles his brothers, his wife, and himself into slavery. This critical game of chance sets off a train of events that mark the beginning of division and strife in human society.

Famous people among the ranks of compulsive gamblers include sports figures, entertainers, and artists. Fyodor Dostoyevsky, who wrote his novella, *The Gambler*, to restore his finances, was a self-described compulsive gambler. Sigmund Freud's 1928 essay about Dostoyevsky was one of the first attempts to understand compulsive gambling as a psychopathological process. This conceptualization and its further development guided the treatment of compulsive gamblers with psychoanalytic therapies.

Until 1980, the term *compulsive gambling* was used to describe the syndrome of apparent loss of control in gambling. At that time, the American Psychiatric Association published the third edition of its DIAGNOSTIC AND STATISTICAL MANUAL (DSM-III). For the first time, the DSM-III established standard criteria to diagnose this disorder, which was renamed *pathological gambling*. The term was coined to avoid confusion with other diagnoses in which the word "compulsive" appeared, such as obsessive-compulsive disorder and obsessive-compulsive personality disorder; these disorders were thought to be unrelated to compulsive gambling. Pathological gambling was grouped under the heading Impulse Control Disorders Not Elsewhere Classified, along with such diagnoses as kleptomania (shoplifting) and pyromania (arson). In 1987, the American Psychiatric Association's *Diagnostic and Statistical Manual* was again revised (to be abbreviated DSM-III-R). In this revision, the term *pathological gambling* and its classification as an impulse-control disorder were retained, but the diagnostic criteria were significantly altered in response to new knowledge about the disorder. Likewise, the fourth edition of the *Diagnostic and Statistical Manual* (DSM-IV) has additional changes reflecting additional research.

THE ADDICTION MODEL OF PATHOLOGICAL GAMBLING

The early psychoanalytic literature often referred to compulsive gamblers as ADDICTS, but it was not until the founding of Gamblers Anonymous (GA) in 1957 that the addictive-disease model became a basis for recovery. GA was initiated through the efforts of a recovering alcoholic who was both an ALCOHOLICS ANONYMOUS (AA) member and a compulsive gambler. GA adapted the TWELVE STEPS of AA, the fellowship's traditions, its spiritual base, and the general format of its meetings to aid in the recovery of gambling addicts. Gam-Anon, a twelve-step group for the friends and families of compulsive gamblers, modeled on Al-Anon Family Groups, was established shortly afterward. Local chapters of Gamblers Anonymous are increasingly available in U.S. communities as well as in treatment units, work settings, and prisons.

The growth of the alcoholism- and drug-addiction-treatment system in the 1960s gave rise to a variety of professional program models that incorporated a cooperative working relationship with twelve-step groups such as Alcoholics Anonymous. In 1971, using one of these models, Dr. Robert Custer developed the first inpatient addiction-oriented treatment unit for compulsive gamblers at the Brecksville, Ohio, Veterans Administration Hospital. Custer's approach proved useful and has been adopted with various modifications by other mental-health and addiction-treatment facilities.

COMMON CHARACTERISTICS WITH OTHER ADDICTIVE DISORDERS

The addiction model conceptualizes pathological gambling as a disease characterized by a dependence on what gamblers refer to as "being in action." The term describes their aroused euphoric state—experienced while gambling. Pathological gamblers who are also users of other drugs compare being in action to the "high" derived from COCAINE or other STIMULANTS. The addiction model is also supported by the many similarities between pathological gambling and substance dependence in risk factors, symptoms, the course of the disease, the nature of relapse triggers, treatment goals, and the process of recovery. A core symptom for both types of disorder is a loss of control over the substance use or gambling behavior. There is also an important comorbidity between the various addictive disorders. For example, a 1986 study of 458 adult inpatients admitted for alcohol and other drug (AOD) dependence to South Oaks Hospital in New York found that 9 percent satisfied diagnostic criteria for pathological gambling and an additional 10 percent had some gambling problems. These rates are many times higher than are found among the general public. In a parallel study of 100 younger AOD inpatients (average age 17), 14 percent met criteria for pathological gambling and an additional 14 percent had some gambling problems. In a later study of cocaine-dependent outpatients, Dr. Bruce Rounsaville at Yale University found pathological gambling in 19 percent of the male and 5.5 percent of the female subjects. Failure to recognize and address gambling problems during treatment for alcohol or other drug dependence often leads to relapse to substance use in a gambling situation. Less frequently, the result is a switch of addictions from alcohol or another drug to gambling.

EPIDEMIOLOGY OF PATHOLOGICAL GAMBLING

Epidemiological studies conducted during the 1980s in New York, New Jersey, Maryland, and Quebec yielded similar estimates. Approximately 1.5 percent of adults were found to be probable pathological gamblers and an additional 2.5 percent were found to have some gambling-related problems. In contrast, a lower prevalence was found in Iowa. Unlike the other jurisdictions studied, in which legal gambling was well established, Iowa had just initiated a state lottery at the time of the survey. The Iowa rate climbed over the next few years, and subsequent studies by Dr. Rachel Volberg found that the prevalence of gambling problems in several states correlated with the state's per capita lottery sales and the number of years of exposure of the state's population to legal gambling.

Dr. Howard Shaffer of Harvard and his colleagues conducted a meta-analysis of 120 epidemiological studies of gambling problems in the scientific literature to try to approximate an overall prevalence rate. They found that among adults, about 1.6 percent had a diagnosis of pathological gambling at some time in their lives and an additional 3.9 percent had gambling problems. Criteria for a current diagnosis was met by about 1.1 percent, while 2.8 percent had current gambling problems of a lesser severity.

In general-population studies in the United States, males outnumber females among probable pathological gamblers by a ratio of about two to one. This is in sharp contrast to male to female ratios observed in treatment programs and GA groups, which are closer to eight or nine males to one female. Some general-population studies in the United States have also found an overrepresentation of nonwhite adults (blacks and Hispanics) among probable pathological gamblers; but these groups, like women, are also underrepresented in treatment and GA populations.

Although less is known about the prevalence of pathological gambling among adolescents than among adults, several surveys of high school students revealed that the vast majority gamble to some extent and that many have problems. For example, a New Jersey study of nearly 900 students found that over 90 percent had gambled at some time in their lives and about 35 percent did so at least weekly. Approximately 5.7 percent of these eleventh- and twelfth-grade students—9.5 percent of boys and 2 percent of girls—were classified as probable pathological gamblers. The Shaffer study found consistently higher rates of both gambling problems and pathological gambling in adolescent and college-age populations.

Established risk factors for pathological gambling include being male, having a family history of heavy or problem gambling or of parental alcoholism, and early interest and participation in gam-

bling activities. In addition, some studies show higher rates of problems in people who are non-Caucasian, unmarried, have less than a high school education, have less than average income, or are under the age of thirty.

CLINICAL CHARACTERISTICS

Gambling usually begins in adolescence, although women may begin gambling later in life. Pathological gambling often develops in three phases, originally described by Custer (1985): (1) the winning phase; (2) the losing phase; and (3) the desperation phase. Female pathological gamblers tend to have a later onset of the illness than males, and may never experience a winning phase.

The Winning Phase. Pathological gamblers often start as winners. Also, in a minority of cases, a significant upsurge in gambling activity begins with a "big win"—a sum equal to half a year's income or more. With or without the big win, individuals developing a dependence on gambling often begin with some success. In this context, they develop an intense interest in gambling and derive an increasing proportion of their self-esteem from feeling smart or lucky. The high derived from being in action becomes a major source of pleasure, a solution to life problems, a remedy for boredom, anger, anxiety, depression, and other uncomfortable feeling states. Bets must be gradually increased in size, in frequency, and sometimes in riskiness to produce the desired psychological effects. This phenomenon parallels the development of tolerance in the substance-dependent patient who must continue to increase the alcohol or drug dosage to reach the preferred feeling state. At this stage of the illness, the gambler devotes a great deal of time and effort to handicapping, studying the sports page, selecting a lottery number, or following the stock market, as well as to the gambling itself. As one gambler put it, "When I'm not occupied with gambling I'm preoccupied with it." Even if the gambler is winning more often than losing, time and emotional investment are withdrawn from friends, family, work, and other interests. The gambler's spouse often senses that something is wrong, but may not identify gambling as the problem. Marital counseling is sometimes sought.

An unreasonable attitude of optimism is also common during the early phase of pathological gambling, sustained by concentrating on wins and making excuses for (or even denying) losses. Because of this denial, the gambler often cannot account for money claimed to have been won. Pathological gamblers who begin with a winning phase are often those who state they gamble for excitement or stimulation.

The Losing Phase. All gamblers know that when on a losing streak it is wise to stop wagering, at least temporarily. For the compulsive gambler, however, losses are experienced as a severe injury to self-esteem. This produces an intense drive to continue gambling in an effort to recoup the money that has been lost, called *chasing losses*. Chasing losses is an important characteristic of this disease and an example of the pathological gambler's impaired control of gambling behavior. Chasing losses accelerates the gambler's losing and initiates a downward spiral. As the gambling debts mount, the pathological gambler will use any and all money available—take out loans, sell property, and gamble with money meant for family necessities. When these sources are exhausted, extended family members or friends may be approached for a "bailout," in the form of a loan or gift to relieve immediate financial pressure. In return, the pathological gambler often promises to give up gambling. However, part of the bailout money is usually gambled in the hope of another big win, and the downward spiral resumes. Although there are both wins and losses during the losing phase, the overall result is mounting emotional and financial distress as well as interference with social, vocational, and family functioning. Serious depression and a variety of stress-related somatic disorders are often experienced. Pathological gamblers report insomnia, gastrointestinal symptoms, dizziness, headache, hypertension, palpitations, chest pains, and breathing problems. Medical help may be sought, but again the connection to gambling behavior is seldom recognized. Family problems become more intense and divorce often results. Alcohol and other drug abuse may accompany gambling and/or function as a substitute when gambling is temporarily interrupted.

Pathological gamblers also describe a WITHDRAWAL syndrome when they are prevented from gambling. Symptoms include craving, restlessness, irritability, insomnia, headache, weakness, gastrointestinal symptoms, shakiness, and muscle aches.

Those pathological gamblers who do not experience a winning phase often describe themselves as

gambling for "escape" (from life problems that seem insoluble). However, by the time the disease is well-developed, most pathological gamblers report gambling for both escape and excitement.

The Desperation Phase. The desperation phase often begins when all legitimate sources of funds are exhausted. The gambling now takes on a desperate quality. The gambler's behavior during this phase may be characterized by activities inconsistent with the individual's previous moral standards, such as lying, embezzling, larceny, and forgery. These activities are justified as temporary expedients until the next big win. Pathological gamblers are often imprisoned both for white-collar crime and for illegal gambling activities such as bookmaking. Violent crime is less common. Studies of prison populations have found gambling problems in 15 to 30 percent of inmates.

An irrational belief in the inevitability of a big win sustains hope to some degree during this phase. Family problems become more intense and mood swings are common. Severe anxiety, major depression, and suicidal behavior are increasingly noted during the late stages of the disease. Manic or hypomanic states are also seen in some cases. Most pathological gamblers who enter treatment or Gamblers Anonymous do so in the desperation phase. Surveys of Gamblers Anonymous have reported suicide attempts by 17 to 24 percent of members.

PATHOPHYSIOLOGY

Several studies have examined neurochemical changes in pathological gamblers. One study measured levels of NEUROTRANSMITTERS and their metabolites in the body fluids of male pathological gamblers, comparing these to levels in normal male subjects. The researchers found an elevated level of a NOREPINEPHRINE metabolite in the gamblers' urine and cerebrospinal fluid, presumably caused by an increased production of the neurotransmitter norepinephrine within the brain. Furthermore, a psychological measure of extraversion in the gamblers was correlated with levels of norepinephrine and its metabolites in their body fluids. Other, less direct evidence suggests the involvement of additional neurotransmitters, including DOPAMINE and SEROTONIN. A single study of beta ENDORPHINS in pathological gamblers found lower baseline levels in those who bet on horse races than those who played poker-machines or those who were not gamblers. Although research on the pathophysiology of this disease is still preliminary, commonalities with other addictions through central nervous system mechanisms are being sought.

IDENTIFICATION AND TREATMENT

Since 1987 a valid and reliable paper-and-pencil test, the South Oaks Gambling Screen (SOGS), has been available for screening general or clinical populations for gambling problems. The maximum score on this screening test is 20. A score of 5 or more indicates probable pathological gambling, while a score of 1 to 4 signals some gambling problem. Following screening a formal diagnosis must be established. A thorough assessment of physical, psychiatric, addictive, family, social, financial, and legal problems is also necessary because multiple problems are common. Alcohol and drug dependencies, psychiatric disorders and physical problems are most effectively treated at the same time as the gambling addiction. Several psychoactive medications have been tried as adjuncts to the treatment of pathological gambling. Among them, fluvoxamine, a selective serotonin reuptake inhibitor (SSRI), has shown some promise. However, definitive studies have not yet been reported.

Treatment may be provided in both inpatient and outpatient settings. Psychoeducation, individual and group therapies, psychodrama, relaxation training, family counseling and RELAPSE PREVENTION training are commonly used treatment techniques, usually combined with an introduction to Gamblers Anonymous. Family treatment and long-term follow-up are important as well. Abstinence from all forms of gambling is one of the treatment goals, along with improved physical and psychological well-being.

Addiction model treatment may be organized either in a separate facility or as part of a combined substance-dependence and pathological-gambling program. Studies of patients involved in both models of the addiction program have yielded positive outcomes, with gambling abstinence in 56 to 64 percent of the patients who were followed, and improvement in many other aspects of their lives.

American society has paid little attention to the development and application of methods to prevent gambling problems. Most efforts to date involve regulation of the availability of gambling (e.g., minors are forbidden to buy lottery tickets or play in

casinos) and posting notices of the availability of help, usually in the form of a toll-free helpline number. The government has made almost no effort to educate youth or the general public about risk factors for pathological gambling and its dangers, in spite of the high prevalence of gambling problems among adolescents. Although children of problem gamblers and alcoholics are known to be at higher risk than others, they have not been the target of organized prevention programs. Since the 1980s, makers of trading cards (e.g., baseball or basketball cards) have begun to insert valuable so-called *chase cards* at random into the packets of cards at pre-determined rates (e.g., one special card per 700 cards), to stimulate interest in purchasing the product. Because this is similar to a lottery, there has been concern about its immediate and future effects on the children who buy these packets in hopes of finding the valuable cards.

OTHER MODELS OF PATHOLOGICAL GAMBLING

Pathological gambling has been explained using models other than addictive disease. It has been considered, for example, a symptom of some other psychiatric disorder, a behavior disorder, learned behavior that can be "unlearned", a moral problem, or the result of a faulty gambling strategy. Based on behavioral principles, several types of behavior therapy have been applied to gambling problems. The addiction model has, however, proved a useful framework for research, intervention, treatment and self-help. As future research clarifies the neurophysiological mechanisms that underlie alcohol and other drug addiction, both the neurochemical basis of pathological gambling and a "common pathway" of addiction in the brain may also be discovered.

(SEE ALSO: *Addiction: Concepts and Definitions; Addictive Personality*)

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SHEILA B. BLUME

GAMMA-AMINOBUTYRIC ACID (GABA)

This is an amino acid derived by a single-step decarboxylation from GLUTAMATE. GABA is the most abundant (in micromolar concentrations/mg of protein) inhibitory NEUROTRANSMITTER—and it is found throughout the animal kingdom. Its role as a neurotransmitter was first defined for the inhibitory nerve in lobster muscle, where GABA accounted for the total inhibitory potency of nerve extracts. A central inhibitory neurotransmitter role for GABA

was securely established only when selective ANTAGONISTS, such as bicuculline, discriminated GABA receptors and pathways from glycine, a related inhibitory amino acid neurotransmitter. GABA actions and receptors for GABA have been linked to central nervous system sedatives such as ALCOHOL and BENZODIAZEPINES.

(SEE ALSO: *Research*)

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FLOYD BLOOM

Los Angeles police officers search suspected members of the Rolling 60s gang for weapons and drugs during a sweep in south Los Angeles, March 31, 1985. (© Bettmann/CORBIS)

GANGS AND DRUGS Youth gangs have been part of the U.S. urban landscape for over 200 years. From the earliest mentions of gangs in the social commentaries of post-Revolutionary War America, gangs have been linked to the use and trafficking of illicit intoxicants. In the late eighteenth century, for example, gangs such as the Fly Boys, the Smith's Vly gang, and the Bowery Boys were well known in the streets of New York City (Sante, 1991). As European immigration increased in the early nineteenth century, gangs such as the Kerryonians (from County Kerry in Ireland) and the Forty Thieves formed in the overcrowded slums of the Lower East Side (of New York City). Gangs proliferated quickly in that time, with such colorful names as the Plug Uglies, the Roach Guards, the Hide-Binders (comprised mainly of butchers), the Old Slippers (a group of shoemakers' apprentices) and the Shirt Tails. Many of these gangs were born in the corner groceries that were the business and social center of the neighborhoods. These groceries also hid the groggeries that were important features of neighborhood life, and guarding them provided a steady income for the gangs. Although not involved in theft, robbery, or the unsavory professions of GAMBLING or tavern-keeping, these gangs warred regularly over territory with weapons—including stones and early versions of the blackjack. They occasionally joined forces to defend their neighborhood, and nearly all were united in their opposition to the police.

Throughout the nineteenth century, gangs emerged in the large cities of the Northeast, in Chicago and in other industrial centers of the Midwest. In the early twentieth century, gangs also formed in the Mexican immigrant communities of California and the Southwest. In what still is widely regarded as the classic work on youth gangs, Thrasher (1927) identified over 1,300 street gangs in the economically disadvantaged neighborhoods of industrial Chicago in the 1920s. He interpreted the rise of Chicago's gangs as symptoms of deteriorating neighborhoods and the shifting populations that accompanied industrialization and the changing populations that lived in the interstitial areas between the central city and the industrial regions that ringed it. Wherever neighborhoods in large cities were in transition, gangs emerged, and their involvement in drinking and minor drug use was a regular feature of gang life.

In the 1990s, gangs became present in large and small cities in nearly every state. They reflect the ethnic and racial diversity of American society (Klein, 1992). Gangs are no longer colorful, turf-oriented groups of adolescents from immigrant or poor neighborhoods. Whereas gangs in the past were likely to claim streetcorners as their turf, gangs today may invoke the concept of turf to stake claims to shopping malls, skating rinks, school corridors, or even cliques of women. Gangs use graffiti and "tagging" to mark turf and communicate news and messages to other gangs and gang members

(Huff, 1989). The participation and roles of young women in gangs has also changed. Through the 1960s, women were involved in gangs either as auxiliaries or branches of males gangs, or they were weapons carriers and decoys for male gang members. Today, female gangs have emerged that are independent of male gangs. Fights are common between the new female gangs. There also is some evidence of sexually integrated gangs, where females fight alongside males (Taylor, 1993).

Traditionally, stealing and other petty economic crimes have long been the backbone of gang economic life. For example, Saint Francis of Assisi commented that nothing gave him greater pleasure than stealing in the company of his friends. English common law in the 13th century accorded especially harsh punishments to the roving bands of youths who moved across the countryside stealing from farmers and merchants. The House of Refuge, the first U.S. residential institution for boys, opened in New York City in 1824, largely in response to the unsupervised groups of youths who roamed the city stealing and drinking. For some contemporary gangs, however, entrepreneurial goals, especially involving drug selling, have replaced the cultural goals of ethnic solidarity and neighborhood defense that historically motivated gang participation and activities. A few gangs have functional ties to adult organized crime groups. Other gangs have become involved in drug selling and have developed a corporate structure that has replaced the vertical organization that in the past regulated gang life.

This article examines recent data on the drug and alcohol involvement of street gangs. Recent changes in the social structure of cities has led to a new generation of gangs and gang cities. We look to these changes in cities and neighborhoods to explain the new patterns of substance use and drug distribution among gangs. Changes in the conception of work, the institutionalization of drug selling, and cultural shifts in gangs and ganging, have influenced gang involvement in drugs and alcohol. This article discusses the relationship between political and economic factors that shape the social structure of communities, the neighborhood effects that result from those forces, and the mediating effects of neighborhood processes on the formation of gangs and their use of substances.

DRUG AND ALCOHOL USE AMONG YOUTH GANGS

ALCOHOL and MARIJUANA use have always been, and continue to be, the most widely used substances among both gang and non-gang youths (Fagan, 1989, 1990; Sheley, Smith, & Wright, 1992). Drinking and other drugs (primarily marijuana) consistently are mentioned as a common part of gang life throughout gang literature. For instance, Short and Strodtbeck's (1965) study of Chicago gangs showed that drinking was the second most common activity of gang members of all races, exceeded only by hanging out on the streetcorner. Although COCAINE may be trafficked by some gang members, it is not often used in either its powder or smokeable forms (Fagan, 1990).

Ethnographic studies of gang life (Hagedorn, 1988; Campbell, 1990; Stumphauzer, Veloz & Aiken, 1981; Vigil, 1988; Padilla, 1992; Moore, 1978, 1992a, 1992b; Taylor, 1993) also show the commonplace occurrence of drinking and its place in a broad pattern of substance use. Dolan and Finney (1984) and Campbell (1990) illustrated the commonplace role of drug use in gang life among both males and females. Stumphauzer et al. (1981) noted that use patterns varied within and among Los Angeles gangs, but changed for individuals over time. MacLeod (1987) noted high rates of drinking among white gang members but only occasional beer use among the Brothers, a predominantly black (but somewhat integrated) gang. Sanchez-Jankowski (1991) found that all members of all gangs drank regularly, using gang proceeds for collective purchases. Although they used drugs in varying patterns, alcohol was mentioned consistently. But Sanchez-Jankowski also mentioned that the Irish gangs least often used illicit drugs, since access was controlled by nonwhites with whom they did not want to engage in business.

Vigil (1985, 1988) described a variety of meanings and roles of substances among Chicano gang members in East Los Angeles, from social "lubricant" during times of collective relaxation to facilitator for observance of ritual behaviors such as *locura* acts of AGGRESSION or VIOLENCE. In these contexts, drug use provided a means of social status and acceptance, as well as mutual reinforcement, and was a natural social process of gang life. Vigil (1988) notes that these patterns are confined to substances that enhance gang social proccesses—

alcohol, marijuana, PHENCYCLIDINE (PCP), and CRACK-cocaine. There is a sanction against HEROIN use among Chicano gangs. Heroin involvement is seen as a betrayal of the gang and the barrio; one cannot be loyal to his addiction and the addict ("tecato") culture while maintaining loyalty to the gang. Vigil noted that gang members prepared for imminent fights with other gangs by drinking and smoking PCP-laced cigarettes. During social gatherings, the gang members used the same combinations to "kick back" and feel more relaxed among one another. Evidently, gang members had substantial knowledge about the effects of alcohol (and its reactivity to PCP), and they had developed processes to adjust their reactions to the mood and behaviors they wanted.

Feldman el al. (1985) observed three distinct "styles" among Latino gangs in San Francisco that in part were determined by the role and meaning of substances in gang social processes. The "fighting" style included males in gangs who were antagonistic toward other gangs. They aggressively responded to any perceived move into their turf by other gangs or any outsider. Drinking and drug use were evident among these gangs, but this was only situationally related to their violence through territoriality. Violence occurred in many contexts unrelated to drug use or selling and was an important part of the social process of gang affiliation. The "entrepreneurial" style consisted of youths who were concerned with attaining social status by means of money and the things money can buy. They very often were active in small-scale illegal sales of marijuana, pill amphetamines, and PCP. While fighting and violence were part of this style, it was again situationally motivated by concerns over money or drugs. The last style was evident in gangs whose activities were social and recreational, with little or no evidence of fighting or violence but high rates of drinking and marijuana use.

Padilla's (1992) study of a Puerto Rican gang in Chicago described how alcohol and marijuana often accompanied the rituals of induction and expulsion of gang members. These ceremonies often were tearful and emotional, with strong references to ethnic solidarity. Padilla described how emotions intensified as the ceremony progressed, and drinking was a continuous process during the events.

Drinking or drug use also is disallowed in some youth gangs, regardless of the gang's involvement in drug selling. Chin (1990) found that intoxication was rejected entirely by Chineses gangs in New York City. Although they used violence to protect their business territories from encroachment by other gangs, and to coerce their victims to participate in the gang's ventures, "angry" violence was rare; violent transactions were limited to instrumental attacks on other gangs.

Taylor (1990a) and Mieczkowski (1986) described organizations of adolescent drug sellers in Detroit who prohibited drug use among their members but tolerated drinking. Leaders in these groups were wary of threats to efficiency and security if street-level sellers were high and to the potential for co-optation of its business goals if one of its members became involved with consumption of their goods. The gangs were organized around income and saw drug use (but not alcohol) as detracting from the selling skills and productivity of their members. Expulsion from the gang resulted from breaking this rule, but other violent reprisals also were possible. However, gangs in both studies accepted recreational use of substances by members, primarily alcohol, marijuana, and cocaine in social situations not involved with dealing.

In the Mieczkowski study, the sellers particularly found danger in being high on any drug while on the job. Gang superiors enforced the prohibition against heroin use while working by denying runners their consignment and, accordingly, shutting off their source of income. Violence was occasionally used by superiors (crew bosses) to enforce discipline. Gang members looked down on their heroin-using customers, despite having tried it at some point in their lives, which in part explains the general ideology of disapproval of heroin use.

Buford (1980) depicted crowd violence among English football (soccer) "supporters" as an inevitable consequence of the game's setting and the dynamics of crowds of youths. Expectancies of both intoxication and violence preceded the arrival of the "lads" at drinking locations surrounding the stadiums. The expectancies were played out in crowd behavior through rituals that were repeated before each match. Alcohol consumption before and during episodes of unrestrained crowd violence was an integral part of the group dynamic, but Buford does not attribute to alcohol an excuse function, nor is alcohol a necessary ingredient for the relaxation of social norms. In fact, he pointedly notes that the heaviest drinkers were incapacitated by inebriation and were ineffective rioters, while

the crowd leaders were relatively light drinkers. In this context, alcohol was central but hardly necessary to the attainment of the expected behavior—the setting itself provided the context and cues for violence.

GANGS AND DRUG SELLING

In the 1980s, the confluence of social problems involving gangs, violence, and drug trafficking changed both popular and political perceptions of gangs. Beginning with the crack-cocaine crisis, youth gangs have been confounded with new forms of drug distribution organizations that involve young men and women in "underclass" neighborhoods. This terminology has been interchangeably used to describe these groups as gangs. Indeed, the growth in cocaine use in the 1980s did coincide with more visible gang involvement in drug selling and drug-related violence (Huff, 1992). Although drug use and selling have been central features of gang life for decades, gangs were often blamed for many of the new drug problems (Newsweek, 1986; U.S. Department of Justice, 1989; Conley, 1992; Los Angeles County District Attorney, 1992). Once seen as streetcorner groups protecting "turf" and neighborhood, gangs were portrayed in the 1980s by the popular press and criminal-justice officials as nascent organized-crime groups focused on the distribution of drugs, with elaborate intercity networks well-financed by drug income.

Several trends contributed to this changing characterization of gangs. First, gangs became highly visible. Although gangs traditionally have been active in larger cities, gangs emerged in the 1980s in smaller cities—with populations as low as 25,000 (U.S. Department of Justice, 1989). Among the 100 largest U.S. cities, gangs have emerged in 42 cities since 1980 (Klein, 1992). Their recent emergence may belie their stability, however, since gangs are well known to often be temporary groupings with short half-lives (Spergel, 1989). The validity of police reports of gang activity itself remains a difficult measurement problem.

Second, gang violence has become more visible if not more prevalent. Gang-related homicides in Los Angeles grew sharply throughout the 1980s, exceeding 500 annually after 1989 (Los Angeles County District Attorney, 1992). In 1991, record homicide rates were set in both Los Angeles and Chicago, two cities with extensive youth-gang networks (FBI, 1992). Yet the classification of homicides as "gang-related" is quite sensitive to definitions; and this trend, too, may reflect anomalies in definition and measurement of gangs and gang incidents. Maxson and Klein (1989) showed that the Los Angeles Police Department rates, based on the gang affiliations of victims or perpetrators, could be halved by applying the motive-based definition used by the Chicago Police Department.

Third, gang involvement in drug trafficking reportedly grew in the 1980s (U.S. Department of Justice, 1989; Spergel, 1990; but see Moore, 1992a). Spergel (1990) found that 75 percent of gang members on probation in San Diego County were convicted of drug offenses at one time or another. Among the 1,200 youths in Chicago, Los Angeles, and San Diego interviewed by Fagan (1990), 32 percent of the gang members reported they were involved in drug selling, compared to fewer than 8 percent of the nongang youths. Based on 45 interviews with California prison inmates Skolnick et al. (1989) claimed that linkages existed between prison gangs and street gangs around drug distribution. But Klein et al. (1991) showed that adolescent participation in rock-cocaine selling in Los Angeles grew equally for gang and nongang youths.

Drug selling also contributed to changes in the organization and meaning of gangs. Taylor (1990b) and Mieczkowski (1986) illustrated the transformation of Detroit gangs from streetcorner groups protecting territory to highly efficient drug-selling organizations. Padilla (1992) describes how Puerto Rican youths in a Chicago gang refocused the gang to drug dealing as the primary source of income. Hagedorn (1991) showed that twenty-two of thirty-seven African-American gang members in Milwaukee went on to become involved in adult drug organizations.

Fourth, reports of gang migration contributed to the perception of gangs as highly disciplined entrepreneurs intent on establishing intercity drug networks to expand their profits. Gang members are alleged to have set up "franchises" or branch offices in remote cities for selling drugs. Huff (1992) reports arrests of Los Angeles gang members in large and small Ohio cities and Detroit gang members in Cleveland (1989); members of Los Angeles gangs have been arrested on drug charges in cities as far east as Columbus (Ohio) (U.S. Department of Justice, 1989). This diffusion of gang activity

from the major gang centers of Los Angeles and Chicago is often cited as a bellwether of the evolution of gangs to a new and more dangerous form.

The temporal proximity of these trends led to their confounding in the popular and sociological literatures, with hasty assumptions that they were causally linked. Few phenomena have been stereotyped as easily as gangs, violence, and drug use, especially in conjunction. These perceptions were amplified in the popular culture through movies and hip-hop music depicting gang life as a stew of violence, drug money, police repression, and the exploitation of women (Taylor, 1992). These perceptions were fueled by gang-related violence in the theaters at the opening of recent films depicting gang life (*Colors*, *Boyz 'n the 'Hood*), as well as reports of violence at rap concerts and in local clubs specializing in "house" and "hip-hop" music (Huff, 1992). These cultural vehicles falsely signaled a transformation of youth gangs from streetcorner groups to more sophisticated crime groups reaping great profits from drug distribution and specializing in lethal violence. In the context of the crack crisis of the mid-1980s and the violence that accompanied it, these portrayals also created a perception that there was an increase in gangs—with more youths in gangs and more violent gangs in urban centers throughout the United States.

The 1980s' changes in drug-use trends, together with earlier changes in labor markets, income dynamics, and demographics in urban centers, suggest that the drug-gang nexus is part of a larger, more complex process of urban change tied to the economic and social transformation of cities. This was a significant era because of the sharp reduction in wholesale cocaine prices, the emergence of crack, and the expansion of street-level drug markets for cocaine and crack distribution (Fagan, 1992a). Reports from law-enforcement agencies suggest that by 1980 gangs formed in smaller cities not traditionally known as gang centers (Klein, 1992), and having little to do with drugs (Fagan & Klein, 1992). This was also an era marked by the emergence or persistent poverty in urban centers and the growth of an "urban underclass" (Wilson, 1987, 1991; Jargowsky & Bane, 1990; Ricketts & Sawhill, 1988; Jencks, 1991). In fact, the emergence of gangs in the 1980s was motivated by broad changes in economic and social conditions during the 1970s, changes that reflected deindustrialization and growing social and economic isolation in large U.S. cities (Jackson, 1991; Hagedorn, 1988; Sullivan, 1989; Fagan & Klein, 1992).

Despite the historically uneven relationship between gangs and drug use or selling (Klein, Maxson, & Cunningham, 1991; Spergel, 1989; Fagan, 1989), recent studies contend that the lucrative and decentralized crack markets in inner cities have created a new generation of youth gangs (Skolnick et al. 1989; Taylor, 1990b). Young drug sellers in these gangs have been portrayed as ruthless entrepreneurs, highly disciplined and coldly efficient in their business activities, and often using violence selectively and instrumentally in the service of profits. This vision of urban gangs suggests a sharp change from the gangs of past decades, and much of the change is attributed to the dynamics of the inexpensive, smokeable cocaine market.

The empirical data suggest otherwise (Fagan, 1989, 1990; Klein et al. 1991; Vigil, 1988; Padilla, 1992; Moore, 1992b; Hagedorn, 1988). Drug selling has always been a part of gang life, with diverse meanings tied to specific contexts and variable participation by gangs and gang members (Fagan, 1990). For example, Fagan (1989) found diverse patterns of drug selling within and across three cities with extensive, integenerational gang traditions, while Klein et al. (1992) reported variability within and across Los Angeles gangs in crack selling.

GANGS, DRUGS, AND NEIGHBORHOOD CHANGE

What are the changes that occurred in cities and communities to explain variation and change in gang participation in drug selling? Two factors have in particular contributed to changes in gangs and the substitution of instrumental and monetary goals for the cultural or territorial affinities that unified gangs in earlier decades. First, cocaine markets changed dramatically in the 1980s, with sharp price reductions. Before cocaine became widely available, drug distribution was centralized, with a small street-level network of heroin users responsible for retail sales (Curtis, 1992; Johnson et al., 1985). The heroin markets from the 1970s were smaller than the mid-1980s crack market, both in total volume of sales and the average purchase amount and quantity. Street-level drug selling in New York City, for example, was a family-centered heroin and marijuana business until the 1980s,

when new organizations developed to control the distribution of cocaine (Curtis, 1992; Johnson et al., 1990). The psychoactive effects of HEROIN (a depressant) and its methods of administration (by injection) limited its sales volume and number of users.

Cocaine was different in every way—a stimulant rather than a depressant, ingested in a variety of ways (nasally, smoked, or injected), and with a shorter half-life for the "high." The price declined as cocaine became widely available, and the discontinuity in distribution systems across successive drug eras created new opportunities for drug selling, and may even have encouraged participation in it. The sudden change in cocaine marketing, from a restricted and controlled market in the 1970s to a fully deregulated market for crack, spawned intense competition for territory and market share (Fagan, 1992a; Williams, 1989). Law-enforcement officials in New York City characterized the crack industry as "capitalism gone mad" (New York Times, 1989).

In inner-city neighborhoods that since the 1970s had grown more socially isolated, and where legal economic activity was declining quickly, drug selling became a common form of labor market participation. Young men began to talk about drug selling and crime as "going to work" and the money earned as "getting paid" (Padilla, 1992; Sullivan, 1989). In the closed milieu of these neighborhoods, the tales of extraordinary incomes had great salience and were widely accepted, even if the likelihood of such riches was exaggerated (Bourgois, 1989; Reuter, MacCoun, & Murphy, 1990; Fagan, 1992a, 1992b). The focus of socialization and expectations shifted from disorganized groups of adult males to (what was perceived as) highly organized and increasingly wealthy young drug sellers. Many other sellers kept one foot in both licit and illicit work, lending ambiguity to definitions of work and income (Reuter et al., 1990; Fagan, 1992a, 1992b).

Second, profound changes in the social and economic makeup of cities (Tienda, 1989; Wacquant & Wilson, 1989) combined to disrupt social controls that in the past mediated gang behavior (Curry & Spergel, 1988). In this context, gangs became less concerned with cultural or territorial affinities and instead became focused on instrumental and monetary goals (Taylor, 1990; Padilla, 1992; but see Moore, 1992a). The interaction of these two trends provided ample opportunities for gangs to enter into the expanding cocaine economy of the 1980s.

As drug selling expanded into declining local labor markets, it became institutionalized within the local economies of the neighborhoods. Whether in storefronts, from behind the counters in *bodegas* (groceries), on streetcorners, in crack or "freak" houses, or through several types of "fronts," drug selling was a common and visible feature of the neighborhoods (Hamid, 1992). Young men and, increasingly, women had several employment options within drug markets—support roles (lookout, steerer), manufacturing (cut, package), or direct street sales (Johnson et al., 1990). Legendary tales, often with little truth, circulated about how a few dollars' worth of cocaine could be turned into several thousand dollars within a short time. Such quick riches had incalculable appeal for people in chronic or desperate poverty.

THE IMPACT OF DRUGS ON GANGS AND GANG CULTURE

The transition from streetcorner group to ethnic enterprise profoundly shaped the social organization of youth gangs. Money became the driving force and organizing principle for these groups. Greed was elevated to a set of beliefs, expressed consistently among gangs and gang members in the neighborhoods with extensive drug markets (Padilla, 1992). The use of the language of work ("getting paid," "going to work") to describe drug selling signals an ideological shift in the social definition of work and the confounding of illegal and legal means of making money. For the young men using this language, there was no particular meaning assigned to drug selling: they pursued commodities that offered instrumental value as signs of wealth (Sullivan, 1989; Padilla, 1992). Any high-demand contraband consumable commodity would likely have inspired the same behavior (as for example weapons, guns).

Not surprisingly, "materialism" is evident in the motivations expressed by young people participating in drug selling—the attainment of wealth as a manifestation of individual power and achievement. Within the isolated, concentrated poverty areas in inner cities, the absence of mediating social definitions allowed the pursuit of material wealth to become transformed into the very substance of so-

cietal bonds and conventional values. Americans always have looked up to the Horatio Algers, whose "self-made" business success defied social odds. As these models were elevated to societal icons, the attainment of wealth seemed to supersede the importance of law or the collective societal good (Wall Street Journal, 1989).

The interaction of drug selling, violence, and material goals often are combined in an emerging set of sociocultural processes within gangs. Padilla (1992) describes how older gang members reoriented the gangs to become business organizations to fuel increases that were disproportionately taken by the older members. In effect, the older members became local employers themselves. Since the older members were no longer the keepers of the culture and regulators of gang organization, they used traditional appeals to ethnic and neighborhood loyalties to recruit and motivate younger gang members. However, they added money incentives to the mix to strengthen their controls over young gang members.

The emphasis on money, individual gain, and quick wealth was so strong that gang members in Detroit (Taylor, 1990a) and Padilla's Chicago neighborhood themselves regarded low-level drug sellers, even their own "homeboys," as "working stiffs" who were being exploited by other gang members. In the past, such denigration of gang work was heretical: entry level jobs in the service of the gang typically would be seen as serving the gang's collective interest. Padilla describes how the new pattern of exploitation of lower-level workers (street sellers) in the gang was obscured by appeals to gang ideology (honor, ethnic solidarity, and neighborhood loyalty) combined with the lure of income to control them. Taylor (1990a) also talks about the use of money as social control within Detroit drug-selling gangs—if a worker steps out of line, he simply is cut off from the business, a punishment far more salient than threats to physical safety. Moore (1992b) describes similar age-related exploitation within chicano gangs in Los Angeles but with little involvement in drug selling.

The exaggerated, almost ideological emphasis on money and material wealth interacts in a very complex fashion with ethnicity and local context. It marks a dramatic shift from the gangs of 1970 to 1985. It is difficult to disentangle the order of events. Did drugs bring in more money, and did money take on greater importance (raised the stakes) because of the economic transformation of the cities? Or did the loss of economic structures make drugs more salient? Did the increased stakes/money bring in guns, which in turn increased the lethality of gang violence? Or did the guns come as a manifestation of power for those who are rejected from any other source of economic or personal power?

In Chicago (Padilla, 1992) and Detroit (Taylor, 1990a, 1990b, 1992), gangs superficially are ethnic enterprises, but more substantively serve as economic units with management structures oriented toward the maintenance of profitability and efficiency. For the African-American gangs in Detroit, there was little concern with the neighborhood or the traditional meaning of gang life. Although forms of internal control varied, money was manipulated along with appeals to ethnic solidarity to maintain loyalty and discipline within groups that otherwise had evolved from gangs or streetcorner groups to become economic organizations. Among the "Diamonds" in Chicago, appeals to Puerto Rican solidarity were used by older gang members to maintain order and motivation within the gang, while these older members kept the lion's share of the gang's profits from drug sales.

GANG MIGRATION

The appearance of Crips, Bloods, Vice Lords, Black Gangster Disciples, Latin Kings, and other well-known gang names in new gang cities across the country has created concerns that gangs are expanding and migrating. Migration is a term that actually includes several distinct patterns: franchising, opening "branch offices," or acquiring and operating local subsidiaries. Gang migration was virtually unknown until the 1980s, when law enforcement and media reports claimed that gang members were setting up illegal businesses in other cities to expand their drug-selling territories.

There are few instances of gangs operating directly in other cities. Migration seems to be concentrated along interstate highway routes, such as I-75 ('Caine Lane, named for its volume of cocaine traffic) connecting Detroit with Ohio cities, or the I-5 route from Los Angeles through California's Central and San Joaquin valleys (Huff, 1992). Others (Waldorf, 1992) found no evidence of gang migration among San Francisco gangs, either in-migration from Los Angeles gangs or reports of

gang members doing gang "business" in other cities.

More often, what appears as migration reflects natural social dynamics of residential relocation, court placements, mimicry, and other forms of gang diffusion. Gang migration also has been confused with the enterprising behavior of individual gang members. There have been sporadic incidents of deliberate migration, isolated among specific gangs in specific cities. But most often, local gangs are composed of local youths who may have adopted the names, graffiti, and other symbols of established gangs from the larger cities.

There are few documented instances of gang migration. Hagedorn (1988) reported that Milwaukee gangs adopted the name of the Vice Lords, a Chicago gang, but had little contact with them. Some Crip or Blood members relocating from Los Angeles may have organized small crews to sell drugs, but law-enforcement officials interpreted this as evidence that Crip chapters had opened in their cities. Chicago gang graffiti appeared in Mississippi as young males were sent away to live with relatives to escape gang violence; but this event was viewed as signs of Chicago gang expansion into the South (Lemann, 1991).

Critics suggest federal initiatives and funds to control gangs have created incentives (and funds) for zealous law-enforcement agencies to identify streetcorner groups or drug gangs as interstate gang conspiracies. Indeed, there have been isolated instances of what Carl Taylor (1990b) calls gang "imperialism," where gangs have established business locations in other cities. Most often, this includes drug selling—and nearly always among entrepreneurial or corporate gangs. Their motives appear to be simply market expansion and increasing profits. Chicago gangs have influenced the gang scene in nearby Evanston. Chinese street gangs operate both regionally throughout the New York metropolitan area and in cities in the Northeast including Philadelphia, Albany, and Hartford (Chin, 1990). The Chinese gangs are not involved in drug trafficking, but their multiple enterprises include extortion and the smuggling of illegal aliens.

SUMMARY

Few phenomena have been stereotyped as easily as gangs, violence, and drug use, especially in conjunction. Drug use has always been a part of gang life, as has peddling of small quantities of whatever street drugs were popular at the time. Many gangs also adopted codes prohibiting drug use, fearing that loyalty to one's drug habit conflicts with loyalty to the gang or efficiency in drug selling. The cocaine and crack crises of the 1980s created opportunities for gang and nongang youths alike to participate in drug selling and increase their incomes. There is little evidence that gang members have become involved in drug selling more so than nongang adolescents. Malcolm Klein and his colleagues, based on police arrest reports following the appearance of crack in Los Angeles in 1985, found no evidence that gang members were arrested more often than nongang members for crack sales, or that drug-related homicides were more likely to involve gang members than nongang members.

Among gangs, involvement in the drug trade varies by locale and ethnicity. Chicano gangs in Los Angeles do not sell cocaine but sell small quantities of other drugs. The crack and cocaine trades are dominated by African-American youths, both gang members and nongang youths. Crack sales began in Chicago more than five years after Los Angeles gangs began selling drugs. As in Los Angeles, both gang and nongang youths are involved. Crack sales in New York flourished beginning in 1986, but there was no discernible street gang structure that participated in drug selling. Instead, loosely-affiliated selling crews provided an organizational structure for drug sales. Chinese gangs have remained outside the cocaine and crack trades. However, some members (but not the gangs themselves) have been involved in transporting or guarding heroin shipments from Asia.

Not all gang members sell drugs, even within gangs where drug selling is common. Drug-selling cliques within gangs are responsible for gang drug sales. These cliques are organized around gang members who have contacts with drug wholesalers or importers. Among the "Diamonds," Padilla (1992) describes how drug selling is a high-status role reserved for gang members who have succeeded at the more basic economic tasks of stealing and robbery. Despite public images of gang members using drug profits for conspicuous consumption of luxury items, drug incomes in fact are quite modest for gang members who sell drugs. Drug incomes are shared within the gang, but the bulk of the profits remain with the clique or gang member

who brought the drugs into the gang. The profits from drug selling, combined with the decline in economic "exits" from gang life, provide some incentive for older gang members to remain in the gang.

(SEE ALSO: *Adolescents and Drug Use; Crime and Drugs; Ethnicity and Drugs; Poverty and Drug Use*)

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GANJA Ganja is a Hindi word (derived from Sanskritic) for the HEMP plant, *Cannabis sativa* (marijuana); the term *ganja* entered English in the late seventeenth century. Ganja is a selected and potent preparation of MARIJUANA used for smoking.

The hemp plant was introduced into the British West Indies by indentured laborers from India who arrived in Jamaica in 1845. Considered to be a "holy" plant, ganja is often used in religious ceremonies in both countries. The Indian Hemp Drug Commission traced the origin of ganja use to India.

Although usually smoked, *Cannabis* may also be mixed with foods or drinks; it is considered a remedy for many ailments in herbal medicine. A medical-anthropological study of ganja users in Jamaica was conducted in 1972; the results revealed little evidence of a deleterious effect among users, as compared with nonusers. These conclusions were criticized, however, by investigators who claim that the tests of maturation and mental capacity that were used were not sensitive enough to detect decrements in higher level mental skills or motivation.

(SEE ALSO: *Bhang; Plants, Drugs from*)

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GATEWAY DRUGS *See* Adolescents and Drugs

GATEWAY FOUNDATION *See* Treatment Programs/Centers/Organizations: An Historical Perspective

GENDER AND COMPLICATIONS OF SUBSTANCE ABUSE Does gender have an influence on whether a drug has complications? There is limited research available to answer this question, for many studies include men only. In general, women drink less often and in smaller amounts than men do, and they suffer fewer ALCOHOL-related problems and less dependence (WITHDRAWAL) symptoms. Women use illicit drugs less often than men do, although women have a higher consumption of prescription tranquilizers, sleeping pills, and over-the-counter drugs. Thus, the differences seen between the genders in complications largely reflect the differences in the respective patterns and prevalence of their alcohol and drug use.

The effects of the drugs are relatively similar between men and women. For example, in a heavy drinking and heavy SMOKING sample population, there is little difference in the mortality rates between men and women. Alcohol- and drug-using women are more likely to have partners who are alcohol and drug users. Such women are often victims of violence. Illicit-drug-using women frequently support their drug habits by prostitution, putting themselves at risk for sexually transmitted diseases (STDs) including HUMAN IMMUNODEFICIENCY VIRUS (HIV) and hepatitis B, even if they are not needle users. Accidents and trauma related to substance abuse are more common in men. The skid-row lifestyle is more common in men. Men report DRINKING AND DRIVING more often than women.

ALCOHOL

Women appear to be more susceptible than men are to alcohol-related LIVER damage. For women, cirrhosis may develop with consumption of 20 grams of alcohol (1–2 drinks) per day—as compared to 80 grams (6 drinks) per day for men. Women alcoholics have death rates 50 to 100 percent higher than their male counterparts. Women develop hypertension, obesity, anemia, malnutrition, and gastrointestinal hemorrhage at lower alcohol consumption levels and with a shorter time course of drinking. Women become intoxicated after drinking smaller quantities of alcohol than do men. For an equivalent dose of alcohol corrected for body weight, women absorb alcohol faster and reach a higher peak BLOOD ALCOHOL CONCENTRATION compared to men. These differences can be explained, in part, by the lower total body water of women compared to men. With a higher percentage of fat and lower water content, there is less volume in which to dilute the alcohol, and its concentration is therefore increased. Women also produce less stomach alcohol dehydrogenase—the enzyme re-

sponsible for breaking down alcohol. This leads to higher blood alcohol levels, since less is metabolized as it passes through the wall of the stomach and, therefore, as compared to men, more alcohol gets into the bloodstream. There may also be some hormonal or immune effects that account for the increased damage in women.

TOBACCO

Women are at risk for all the same health complications of smoking as are men. The differences seen in the 1990s largely reflect the lower prevalence of women smokers in past generations. For example, as smoking rates have increased in women, lung cancer rates have also increased.

REPRODUCTION

A woman's drinking pattern may be influenced by the mood changes associated with the phases of the menstrual cycle, and her blood alcohol level actually measures higher during the premenstrual period for any given amount of alcohol. This may make it difficult for a woman to predict the effects of her drinking. Oral contraceptives interact with cigarette smoking in contributing to coronary heart disease in women. Cigarette smoking is also correlated with an earlier onset of menopause. In her role as childbearer, a woman's substance use may have harmful effects on the FETUS and newborn. These effects may be related to her lifestyle, such as poor nutrition and poor prenatal care, or to the toxic effects of the drugs themselves resulting in fetal growth retardation, at-birth neonatal abstinence syndrome (withdrawal), and neurobehavioral abnormalities in the child.

Alcohol, tobacco, and illicit drugs like COCAINE and HEROIN are all associated with decreased fertility, increased rate of spontaneous abortion (miscarriage), and decreased birthweight in the newborn. The severely dependent woman may stop menstruating altogether. Menses resume, however, when abstinence or stabilization on methadone maintenance is achieved.

(SEE ALSO: *Fetal Alcohol Syndrome; Pregnancy and Drug Dependence; Women and Substance Abuse*)

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GENE A gene is a unit of heredity that confers some trait or function on the organism. Most genes are thought to be essential to development and normal functioning. Genes are often a primary determinant of interpersonal differences; for example, they determine whether you have blue or brown eyes. A disrupted or mutated gene can cause serious, even fatal problems. Genes are composed of DNA and found in the chromosomes in the nucleus of a cell. At present, we are on the verge of identifying all the human genes due to the efforts of the genome projects.

MICHAEL J. KUHAR

GENE REGULATION: DRUGS To understand the regulation of genes, the following sequence of events must be appreciated: A gene, made of DNA, is "transcribed" to produce a messenger RNA, or mRNA, that is "translated" to produce a protein product. Gene regulation refers to the regulation of production of mRNA from the gene.

Although every organism contains a collection of genes necessary for its survival and reproduction, not every gene is turned on or is producing mRNA at any given moment. At any moment or stage of development such as in the adult, only a subpopulation of genes is expressed (i.e., producing mRNA). During the development and growth of an organism, certain gene products guide and program growth. Because only a subgroup of the genome is expressed at any stage of the life cycle, gene regulation must occur. Gene regulation also occurs during a life cycle as in the adult brain.

A gene contains an important part called the "promoter" that controls the rate at which mRNA is produced. A human gene, for example, can be expressed or turned on when the promoter is activated. For the purposes of our discussion here, the promoter of a gene is the site of gene regulation. Perhaps a reasonable analogy is that a promoter is like a light switch; the switch must be "activated" or turned to "on" before light can be produced, and not every light has to be turned on. There are many different kinds of promoters, each of which is turned on or off by its own "transcription factor", which is a protein that binds to the promoter region of a gene and alters the rate of transcription (production of mRNA).

The brain has evolved such that receptors for drugs and neurotransmitters, through complex biochemistry, can affect the state of the promoters for genes by activating transcription factors. The active transcription factor then regulates gene expression by binding to the promoter region of the gene.

A simplified summary of the events involved in gene regulation is as follows: A drug is taken and it goes to the brain where it interacts with its receptor; the activated receptor, through biochemical pathways, can activate transcription factors; the transcription factors then bind to the promoter of the gene; binding of the transcription factor to the promoter changes the rate (faster or slower) at which the gene produces mRNA, which ultimately changes the level of the protein product in the cell. The most important point for consideration here is that drugs of abuse can change the biochemical composition of the brain by this mechanism or pathway. Many scientists believe that change in brain composition by this mechanism are one of the bases for drug addiction. It is often said that the drug dependent brain is a changed brain, and this is what that statement means. Drugs change the balance of proteins in the brain and that influences how the brain functions.

Understanding how drugs change brain protein composition by altering gene regulation is an important area of research, because this is a key to understanding what makes a brain (and, of course, a person) addicted. Once that is understood, then we can begin to repair the addicted brain by intervening in various ways or by reversing such changes in brain protein composition. If gene regulation can be controlled or influenced, then the protein composition of the brain can be influenced, and the way the brain functions can be correspondingly influenced. Science does not have the knowledge or skill to do this now, but it is one of many hopes for the future.

MICHAEL J. KUHAR

GENETICS *See* Causes of Substance Abuse: Genetic Factors; Vulnerability as Cause of Substance Abuse: Genetics

GENOME PROJECT The project with the goal of sequencing the human genome, which is the collection of all human genes. In the 1990s, a scientific commitment was made to identify all the genes in human chromosomes, and some other organelles such as mitochondrion. This involved international cooperation and a very significant effort by many laboratories. Genes are made of DNA, which is composed of sequences of four different nucleotides, with perhaps as many as three billion nucleotides total in the genome. The genome project's goal is to determine all the nucleotides and their sequence, and then make this information available through the Internet. A gene is a functional segment of this DNA sequence that produces a product, an mRNA, which in turn guides the formation of a protein. Estimates of the number of genes in humans vary widely, with the averages falling in the range of 70,000 to perhaps 120,000. It is believed that identifying the genome sequence is the first step necessary for producing dramatic advances in biology and medicine. In addition to the human genome project, there are similar on-going projects attempting to sequence the genomes of other organisms as well.

MICHAEL J. KUHAR

GINSENG Ginseng is the most revered and well-known plant of Chinese herbal medicine; it is sold over the counter in Asian apothecaries and groceries worldwide. This plant of the family Araliaceae grows on both sides of the Pacific, with *Panax schinseng* the Asian form and *Panax quinquefolius* the North American form. It is a perennial herb with five-foliate leaves, and its fleshy aromatic root is valued as a tonic and a medicine.

The root has been used by Native Americans, Siberians, Chinese, and other Asians for millennia. Usually it is taken as a tea—once a day as a general preventative tonic, more frequently for therapeutic purposes. Since the North American form is considered the most potent, it is now grown in ASIA along with the local variety. American ginseng is also exported to Asia, then sometimes reimported into the United States as a Chinese or Korean herbal. Both the wild and cultivated forms are used. Roots older than five years are needed for good effect, and the older and larger the root (seven to fifteen years is prized), the more the ginseng costs. Dried roots

Figure 1
Ginseng

are heated and sliced thinly to make tea, but pieces may be kept in the mouth, sucked, and eaten. The many ginseng products now sold (sodas, candies, etc.) have no real tonic or therapeutic value.

Ginseng has a bittersweet aromatic flavor, contains ALKALOIDS, and is said to be good for mental arousal and general well-being. It has not been established in Western medicine and pharmacology, although it contains properties that might be isolated and used pharmacologically.

(SEE ALSO: *Plants, Drugs from*)

MICHAEL J. KUHAR

GLUE/GLUE SNIFFING *See* Inhalants

GLUTAMATE Glutamate (GLU) is a dicarboxylic aliphatic amino acid. Chemically symbolized as $COOH\text{-}CH_2\text{-}CH_2[NH_2]\text{-}COOH$, it is abundant (micromolar concentrations/mg protein) in NEURONS (nerve cells) as well as in almost all other cells of the body. Its role as the major excitatory NEUROTRANSMITTER in the brain was recognized reluctantly; its universal ability to excite all neurons was considered too nonspecific for a neurotransmitter, so it awaited the development of drugs that antagonized GLU and the specific neuropathways from which it was released.

Its source for this special role in NEUROTRANSMISSION is unknown, but the synaptic vesicles of glutamatergic neurons have a selective ion-ex-

change mechanism to compartmentalize GLU from other metabolic pathways. Excessive GLU-receptor activation can lead to neuronal death.

(SEE ALSO: *Research*)

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FLOYD BLOOM

GLUTETHIMIDE Glutethimide was introduced into clinical medicine in 1954. It was prescribed to treat insomnia and sold as Doriden. It was first acclaimed as a safer "nonbarbiturate" hypnotic—implying that it was free of the problems of abuse, addiction, and withdrawal that were, by then, recognized drawbacks of the older barbiturate SEDATIVE-HYPNOTICS. Within ten years, however, it was recognized that, in most respects, its actions are like those of the BARBITURATES and it shares the same disadvantages.

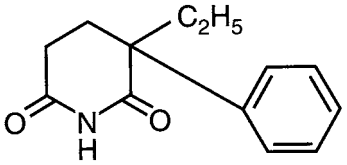

Figure 1
Glutethimide

Glutethimide is structurally related to the barbiturate drugs and, like the short-acting barbiturates, it depresses or slows the central nervous system. Side effects from its proper use are relatively minor, but a rash is often seen. Like barbiturates, it can produce intoxication and euphoria; TOLERANCE and DEPENDENCE can result with daily use. Glutethimide is metabolized somewhat differently than barbiturates, and OVERDOSE is often far more difficult to treat than barbiturate overdose; fatalities are not uncommon. As a consequence of this and its ABUSE POTENTIAL, glutethimide is included in Schedule III of the CONTROLLED SUBSTANCES ACT. Since the introduction of the BENZODIAZEPINES to treat short-term insomnia, the use of glutethimide has decreased considerably.

(SEE ALSO: *Barbiturates; Complications; Sedatives*)

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GOLDEN TRIANGLE AS DRUG SOURCE The world's largest illicit OPIUM-growing area is the Golden Triangle—a region in Southeast Asia of some 150,000 square miles (388,500 sq km). The Golden Triangle extends from the Chin hills in the west of Myanmar (formerly Burma), north into China's Yunnan province, east into Laos and Thailand's northern provinces, and south into the Kayah state of Myanmar. It encompasses all the Shan state in Myanmar and supplied some 35 percent of the HEROIN used in the United States between the 1960s and early 1990s. Between 1990 and 1999, however, changes in heroin trafficking greatly reduced the importation of heroin from Southeast Asia. In 1990, Myanmar, Thailand, and Laos supplied about 56 percent of the heroin consumed in the United States. By 1999, Latin America supplied most of the heroin to the United States, accounting for 82 percent of the heroin seized in the U.S. The Southeast Asian opium crop, which was on the rise in the early 1990s, suffered a sharp decline due to adverse weather in the later 1990s.

The United States Government has supplied millions of dollars to Myanmar and Thailand in an effort to reduce OPIUM-POPPY (*Papaver somniferum*) cultivation and interdict heroin destined for the United States. As they have done for years, disenfranchised tribal people cultivate opium as a medicinal and cultural product, as a cash crop to buy food and supplies and improve living conditions, and as a means to procure weapons. Political events in Southeast Asia are complex and are changing constantly. The U.S. government has managed a limited success in helping to reduce opium cultivation in Laos and Thailand; it is anxious over the increased production in Myanmar and the increasing flow of heroin exiting that country

via China to Hong Kong, through Rangoon toward Malaysia and Singapore, and through India and Bangladesh.

THE OPIUM SUPPLY

The Golden Triangle had favorable weather in the early 1990s, which resulted in record opium crops. By far the largest producer is Myanmar, which until 1988 had attempted to reduce illicit opium production, strike at illicit refineries, and interdict shipments of illicit drugs. In 1988, however, the military government shifted its police and military away from drug-control efforts, to suppress domestic political opponents. This policy did not shift during the 1990s, despite constant efforts by the United States to have Myanmar take more effective antidrug actions. Myanmar produces over 50 percent of the world's opium.

Laos, isolated and largely ignored by the West since 1975 when the Communist Pathet Lao seized power, cultivated opium in its nine northern provinces—about 20 percent of Myanmar's production. Partly because of the 1990 collapse of the Soviet Union, Laos's principal trading partner and ally, the Laotian government has entered into a number of cooperative agreements with Western nations. Opium production decreased by 16 percent from 1998 to 1999, due mostly to severe weather. However, Laos still accounted for 11 percent of the production in the region.

Thailand is more important as a TRANSIT COUNTRY for Myanmar's opium and heroin. Thailand's already marginal production dropped 38 per cent in 1999, accounting for less than one percent of Southeast Asia's potential production. A traditional producer of opium since the mid-1800s and a net importer of heroin, Thailand's opium is grown in the northern highlands by nomadic hill tribes who are not tied to Thailand culturally, religiously, or politically. Opium cultivation in Thailand remains illegal, so the government has sponsored both eradication and crop-substitution efforts in the north.

China has become a major narcotics transit point because of its open border with Myanmar, its location adjacent to the Golden Triangle, and its excellent transportation and communication links with the trade ports of Hong Kong and Macao. Much of the heroin processed from opium by the Kokang Chinese in the Golden Triangle transits through Yunnan, Guangxi, and Guangdong provinces by road to Hong Kong for overseas distribution.

CULTIVATION CONDITIONS

A number of factors have contributed to the thriving opium economy of the Golden Triangle—and the complex politics surrounding and sustaining it. First, the topographical and climatic conditions are ideal for opium cultivation. The demographic conditions also provide a division of labor conducive to an economic system rooted in drug cultivation, processing, and trafficking. The area under cultivation is largely mountainous, ranging from about 5,000 feet (1,500 m) to more than 9,850 feet (3,000 m), with four major river systems supporting the transportation networks and any ongoing economic-development efforts. The remote harsh terrain has fostered great efforts to topple the central governments and to capitalize on the economic opportunities offered by the opium trade.

Second, the ethnography of the region is complex. The region is inhabited by a multitude of ethnic groups, possessing a diversity that defies simple classification. Burman, Shan, Kachin, Thai, and Yunnanese are broad categories that contain widely varied ethnic subgroups. At least twenty-five mutually unintelligible dialects are spoken among the Kachin people. Moreover, there are numerous other groups who do not belong to the larger ethnic division—such as Ahka, Hmong (Miao), Lisu, Lahu, Karenni, and Wa, to name a few. Most of these groups are nomadic—not geographically localized; therefore, little basis exists for territorial political organization. Yet, national boundaries have paid little heed to this fact and have often cut apart ethnic groups, fueling insurgency as the dominant form of politics in the region.

Cultivating opium in the Golden Triangle has been a way of life since the mid-1800s and has represented an important source of income for impoverished, nomadic hill tribes.

(SEE ALSO: *Crop Control Policies; Foreign Policy and Drugs; International Drug Supply Systems; Source Countries for Illicit Drugs; Transit Countries for Illicit Drugs*)

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GROUP THERAPY *See* Treatment Types: Group versus Individual

H

HABIT *See* Addiction: Concepts and Definitions

HABITUATION *See* Addiction: Concepts and Definitions

HAGUE OPIUM CONFERENCE OF 1911 *See* Britain, Drug Use in; Opioids and Opioid Control

HAIGHT-ASHBURY FREE CLINIC *See* Treatment Programs/Centers/Organizations: An Historical Perspective

HAIR ANALYSIS AS A TEST FOR DRUG USE Because every drug taken becomes a permanent part of the user's hair, laboratory analysis of hair can reveal the presence of a variety of drugs, including HEROIN, COCAINE, AMPHETAMINES, PHENCYCLIDINE, MARIJUANA, NICOTINE, and BARBITURATES. Hair analysis is widely accepted by courts, parole boards, police departments, and employers around the country for detecting long-term drug use. It's also increasingly used to determine maternal/fetal drug exposure and to validate self-reports of drug use.

Unlike urinalysis, which can only detect drugs ingested within the past three to four days, hair analysis can reveal the ingestion of drugs during the past ninety days (or longer). Since hair grows at a relatively constant rate of ½ inch (1 cm) per month, segmental analysis of hair strands could localize the time of drug exposure to within as little as one particular week. Although various hair treatments—such as tinting and perming—may remove some of the evidence, detectable traces will indelibly remain in the hair.

DRUGS IN HAIR

Hair is nonliving tissue composed primarily of a sulfur-rich protein called keratin. Hair growth occurs at a rate of 0.3 to 0.4 millimeters (0.011 to 0.012 inches) per day from the follicle (a saclike organ in the skin) in cycles of active growth followed by a resting phase. For an adult, approximately 85 percent of scalp hair is in the growing stage at any time. Two sets of glands are associated with the follicle: The sebaceous glands, which excrete sebum (a waxy substance), and the apocrine glands, which excrete an oil that coats the hair. Hair color is determined by genetic programming for varying amounts of melanin, a pigment that is synthesized in hair cells called melanocytes.

The exact mechanism by which drugs enter hair is unknown. They may be deposited from the capillaries, which supply blood to the follicles, or they may be excreted in the sebum, oil, or sweat that coat the hair shafts. Drugs can also be deposited on

Psychemedics Corporation president Raymond Kubacki shows his company's new drug testing pack at a press conference in New York City, July 12, 1995. The kit allows parents to clip a lock of their children's hair and mail it in to test for drug use. (Reuters/Mark Cardwell/Archive Photos)

the hair by environmental exposure (such as marijuana smoke or cocaine powder in the air).

When hair is analyzed for drug use, a sample is taken from either the head or the body. It's washed to remove dirt and any external drug deposits (the wash medium is also tested), then stripped of melanin. The actual analysis is performed by RADIOIMMUNOASSAY that detects not only traces of drugs but their *metabolites*, chemicals that appear only when the body has metabolized (processed) the drug. All positive samples are confirmed by gas chromatography/mass spectrometry (GC/MS). This second test has a cutoff level to eliminate specimens containing drug levels that could come from environmental exposure (inhaling second-hand marijuana smoke or eating food that contains poppy seeds).

SIGNIFICANCE OF HAIR TESTING

Once a drug is embedded in hair it appears to be stable indefinitely, although concentration diminishes somewhat over time. (Cocaine metabolite has, for example, been detected in hair from a pre-Columbian mummy more than 500 years old.) This is an obvious advantage over other methods of DRUG TESTING, such as urinalysis, which can detect drugs ingested only within the past few days. Depending on length, hair analysis can determine drug use from months to years in the past. Hair is also easily collected and stored. If more testing is required, another sample may be easily obtained.

One disadvantage of hair analysis is that it won't reveal drug use during the three to five days before testing, since hair does not grow quickly enough to show this. Hair analysis is also more expensive than urinalysis, and the results take longer to be determined. The two tests can always be used in combination, however, to give a more complete picture of the individual's past and present drug use.

IS IT FAIR?

Some groups have raised concerns that hair testing may be biased against minority subjects because coarser, darker hair tends to trap more environmental drug residue than lighter, thinner hair. Hair testing labs say that their processes, which remove melanin from samples, removes any chance of distinction or discrimination by race or ethnic group. The Society of Forensic Toxicologists disagrees, arguing that even removing the pigment from hair does not eliminate the risk of bias in analysis.

Definitive proof of drug use, however, is based not on environmental exposure to drugs, but on the metabolites incorporated into the hair shaft. These indicators can only appear when the subject's body has metabolized the drug. The results of hair analysis are widely used and accepted by courts, law enforcement bureaus, and government agencies, including Federal Reserve banks and more than 80 state programs and medical research projects.

(SEE ALSO: *Industry and Workplace, Drug Use in; Military, Drug and Alcohol Abuse in the U.S.*)

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HALF-LIFE *See* Dose-Response Relationship

HALFWAY HOUSES Although the term is of recent origin as used in connection with alcohol or drug treatment, the basic idea of the *halfway house* is almost two hundred years old. It designates a residential facility that provides a drug-free environment for individuals recovering from drug or alcohol problems but not yet able to live independently without jeopardizing their progress. By definition, halfway houses are not located in hospitals or PRISONS, but they vary in the extent to which they are integrated with local community life, and in size, sponsorship, sources of financial support, regulatory status (licensed or unlicensed by a state agency), treatment philosophy, and the degree of legal coercion to which residents are subject. Some specialize in alcohol abusers or drug abusers, while some serve both; some focus on specific population groups like offenders, ADOLESCENTS, or WOMEN, while others are inclusive. Some will accept only those with at least a few days of abstinence; others provide DETOXIFICATION services. Some are loosely structured and rely for staff on recovering people; others provide formal treatment and employ a professional staff.

In sum, the term covers a lot of ground and has no stable meaning. Indeed, its meaning in any given state depends on that state's licensing provisions, and whether these make any distinctions among halfway houses, recovery homes, and other similar forms of residential treatment. At a minimum, however, the term implies a group of people with alcohol and/or drug problems living together in a formal, therapeutic arrangement and abiding by the rule of abstinence. In 1987, there were more than 1,300 such programs in North America, many of them members of the National Association of Halfway Houses.

Although there is increasing interest in establishing residential forms that tolerate off-site consumption that does not disrupt facility life, these would not be considered halfway houses in the common use of the term. Further, because the halfway house is a sponsored, therapeutic program, however informally operated, it is everywhere subject to special zoning ordinances that regulate the location of therapeutic agencies. Thus, the halfway house is distinct from what is called "alcohol and drug-free (or sober) housing." The latter is designed to be part of a locality's ordinary housing stock and to be exempt from such regulation.

The Federal Anti-Drug Abuse Act of 1988 (Public Law 100-690) included a provision to encourage the development of ALCOHOL- AND DRUG-FREE HOUSING. Each state that receives federal block grant funds for drug and alcohol programs must establish a 100,000 dollar revolving fund to make start-up loans for such facilities. Although this money can be used to develop halfway houses, as we have defined them, the revolving fund has in practice been used to stimulate less formal approaches to housing recovering people.

(SEE ALSO: *Homelessness, Alcohol, and Other Drugs, History of; Treatment: History of*)

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HALLUCINATION The word *hallucinate* is derived from the Greek *halyein*, meaning "to wander in mind." Hallucinations are perceptions that occur in the absence of a corresponding external sensory stimulus. They are experienced by the person who has them as immediate, involuntary, vivid, and real. They may involve any sensory system, and hence there are several types of hallucinations: auditory, visual, tactile (e.g., sensations on the skin), olfactory (smell), and gustatory (tastes). Visual hallucinations range from simple (e.g., flashes of light) to elaborate visions. Auditory hallucinations can be noises, a voice, or several voices carrying on a conversation. In command hallucinations, the voices often order the person to do things that at times involve acts of violence.

Hallucinations have been a hallmark of mental illness throughout history. They are an important clinical feature of several psychiatric conditions in which psychosis can occur, such as SCHIZOPHRENIA, manic-depressive illness, major DEPRESSION, and dissociative states. WITHDRAWAL from ALCOHOL can cause visual as well as other sensory hallucinations. In alcoholic hallucinosis, a person dependent on alcohol develops mainly auditory hallucinations that can persist after the person has stopped drinking. Hallucinations may be induced by illicit drugs, such as COCAINE, AMPHETAMINES, and LSD. These hallucinations are usually visual, but they can also be auditory or tactile, as in the sensation of insects crawling up the skin (an example of a haptic hallucination). Occasionally, after repeated ingestion of drugs, some people experience "flashbacks"—that is, spontaneous visual hallucinations during a drug-free state, often months or years later.

The cause of hallucinations is not known, but it is likely to be multifactorial through a combination of physiological, biological, and psychological variables. Numerous hypotheses have been proposed. According to a perceptual release theory, hallucinations develop from the combined presence of intense states of psychological arousal and decreased sensory input from the environment (e.g., sensory deprivation) or a reduced ability to attend to the sensory input (e.g., in delirium). This leads to the emergence of earlier images and sensations that are intepreted as originating in the environment. Other researchers suggest that abnormalities in brain cell excitability or in the information processing system of the central nervous system cause hallucinations. Biochemical theories implicate brain NEUROTRANSMITTERS such as DOPAMINE. Drugs that block brain dopamine activity (ANTIPSYCHOTICS) alleviate hallucinations, whereas drugs that stimulate dopamine release induce hallucinations.

Hallucinations can occur in people who are not mentally ill. In acute bereavement, some people report seeing or hearing the deceased. Sensory, SLEEP, food, and water deprivation can produce hallucinations, as can the transition from sleep to wakefulness and vice versa (called hypnopompic and hypnogogic hallucinations, respectively). These hallucinations can occur as side effects of prescribed medications, such as drugs that treat cardiac conditions, or in various medical disorders (e.g., migraines, Parkinson's Disease, infections). They have been described in persons with hearing loss and blindness; in these instances, it has been hypothesized that they may be due to chronic sensory deprivation.

The treatment of hallucinations is part of the treatment of the entire psychotic syndrome. Antipsychotic medications (e.g., haloperidol, chlorpromazine) are effective in reducing and often eliminating hallucinations. When the hallucinations are part of a medical disorder, it is necessary to correct the underlying condition, or remove the causative agent, in addition to prescribing antipsychotic medication.

(SEE ALSO: *Complications: Mental Disorders; Delirium Tremens; Hallucinogenic Plants; Hallucinogens*)

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HALLUCINOGENIC PLANTS Literally hundreds of hallucinogenic substances are found in

many species of plants. For example, a variety of mushrooms contain indole-type HALLUCINOGENS, the most publicized being the Mexican or "magic" mushroom, *Psilocybe mexicana*, which contains both the hallucinogenic compounds PSILOCYBIN and psilocin, as do some of the other *Psilocybe* and *Conocybe* species. The PEYOTE cactus (*Lophophra williamsii* or *Anhalonium lewinii*), which is found in the southwestern United States and northern Mexico, contains MESCALINE. The seeds of the MORNING GLORY, *Ipomoea*, contain hallucinogenic LYSERGIC ACID derivatives, particularly lysergic acid amide. Many of these plants and plant by-products were and are used during religious ceremonies by Native Americans and other ethnic groups.

Some plant substances may contain prodrugs, that is to say, compounds that are chemically altered in the body to produce PSYCHOACTIVE substances. For example, NUTMEG contains elemicin and myristicin, whose structures have some similarities to the hallucinogen mescaline as well as the psychostimulant AMPHETAMINE. It has been hypothesized that elemicin and myristicin might be metabolized in the body to form amphetamine- and/or mescaline-like compounds, but this has not been proven. The fact that hallucinogenic substances are found in nature does not mean that they are safer or purer than compounds that have been synthesized in the laboratory. Some common edible mushrooms that can be purchased in any supermarket may be sprinkled with LSD or other hallucinogens to be misrepresented as magic mushrooms. In addition, serious problems—even death—may occur when species of hallucinogenic plants are misidentified and people mistakenly ingest highly toxic plants, such as poisonous mushrooms.

(SEE ALSO: *Ayahuasca; Ibogaine; Jimsonweed; Plants, Drugs from*)

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R. N. PECHNICK

HALLUCINOGENS The term *hallucinogen* literally means producer of HALLUCINATIONS. A variety of drugs and medicines as well as various disease states can lead to the development of hallucinations. They can occur during a high fever, after acute brain injuries, or as part of a DELIRIUM, accompanied by confusion in judgment, intellect, memory, emotion, and level of consciousness. The patient is said to be "out of it"—not in touch with reality. In fact, many infections affecting the brain, conditions that disrupt the availability of nutrients essential for brain function, or direct brain injury can cause transient or prolonged delirium. Disease states not directly involving the brain also can alter brain function. For example, the overproduction of thyroid or adrenal hormones in endocrine disease can cause psychotic mental symptoms. In addition, poisoning or other toxic reactions can produce hallucinations.

Some drugs used to treat certain illnesses, although not prescribed for their behavioral effects, may be PSYCHOACTIVE and cause auditory and/or visual hallucinations in some but not all patients. High doses of the adrenal hormone, cortisone, which is prescribed to reduce inflammation in arthritis or allergies, can produce elation or depression and mood-related hallucinations. Similarly, the administration of thyroid hormones for the treatment of thyroid grand deficiencies can cause restlessness, nervousness, excitability and irritability, and psychotic mental symptoms. Drugs derived from the belladonna plant, such as atropine and SCOPOLAMINE, have many uses in clinical medicine but in high doses can cause memory lapses and illusions. Delirium also may result from the sudden withdrawal after the chronic administration of certain drugs, especially ethanol (ALCOHOL) and SEDATIVE drugs of the BARBITURATE class. The vivid hallucinations of DELIRIUM TREMENS (DTs) during the WITHDRAWAL from alcohol have been vividly portrayed in the cinema and television.

Many drugs that affect behavior can alter the level of consciousness or the perception of the environment. PHENCYCLIDINE (known as PCP or "angel

dust") can produce a state of altered consciousness in which sensations from the body and relationship to the environment are misinterpreted. The subject may experience numbness in the limbs and feel as though they are removed from their bodies. These distorted perceptions of the real world can lead to confusion, delusions, and hallucinations—and violent behavior can occur with the slightest provocation. There is controversy as to whether these varied reactions are psychotomimetic (imitating mental illness with psychoses), but not about the extent to which, depending on the dose, subjects are out of it. High and/or frequent doses of stimulants such as AMPHETAMINE, METHAMPHETAMINE ("speed" or "ice"), or COCAINE can cause paranoid thought or delusions. Moreover, high doses of MARIJUANA or HASHISH can lead to dreamy illusions or hallucinations. Thus, many drugs under certain conditions can cause hallucinations as part of the production of a complex behavioral syndrome, which may include a general alteration of the level of consciousness and the disruption of the ability of the brain to process information and appreciate the real world.

The term *hallucinogens* has come to mean a group of compounds that reliably, temporarily, and universally alter consciousness without delirium, sedation, excessive stimulation, or any intellectual or memory impairment as prominent effects. Indeed these altered mental effects are the main effects of such drugs. There are a number of synonyms for drugs that produce hallucinations that occur with clear consciousness, but the term *psychedelic* has come into wide use. In the 1960s the term was coined by Humphrey Osmond, a British psychiatrist who came to North America to continue studies of the psychiatric effects of MESCALINE and LSD, and was enthusiastic about their use in enhancing insight in psychotherapy. The term *psychedelic* was invented from greek roots to mean "mind manifesting," from *psyche* (mind, soul) + *deloun* (to show). This refers to the convincing clarity with which a subjective experience is compellingly revealed to the subject who has taken a hallucinogen. What is seen, thought, and felt is vivid—contrasting sharply with the normally ordered perceptions of the world in which we move about and perform our practical tasks. Key to the hallucinogenic experience is that drab everyday reality, while clearly perceived in this drug state, has simply lost its importance in favor of vivid subjective sensations and perceptions and interpretations of them that absorb attention. A door is recognized but not simply for its utility; rather the grain of the wood and its fine detail becomes fascinating, and the grain of the wood seems to move and flow. Thus, during the hallucinogenic experience, it is not the utility of what is seen but rather some aspect of shapes and colors and passing thoughts or memories that take on a life of their own, commanding attentive interest. The color of an object is more important than the object. The subjective impact is that thoughts and sights have some uncanny, undeniable, but inexplicit meaning. The sense of great truth is present, but not an urge to test the truth of these images. Rather, one is a spectator of a "TV show in the head." These events are not only clearly "seen" but remembered without confusion. This has been called "consciousness expanding," implying control over a vast span of experiencing. That is wrong, since judgment is *not* enhanced. Rather, the effect is of enhancing the sense of importance of normally unimportant subjective experiences of sensations and perceptions.

Since with hallucinogens everything—even the most familiar scenes—seems novel and is seen in a new way, the experience is in startling contrast with our normal view of the world. Such effects invite many uses. The intrinsic effects of hallucinogenic drugs not only shift perceptions, making the old new, but evoke a loosening of emotions and thoughts. Hence there were efforts to use hallucinogenic drugs therapeutically—to stimulate and enhance new ways of examining problems. But in spite of the alluring promise, no lasting improvement in learning or problem solving has been found after numerous studies. Similarly, the effects produced by hallucinogens seem so significant and strikingly different from everyday life that they can readily be used to enhance mystical thought and belief. Some Native American groups thus use the hallucinogen PEYOTE in religious ceremonies. The intent is to dispose the celebrants to higher thoughts (to be "in the mind of God"); they are told not to attend to the odd perceptions and rather to relax and contemplate higher thoughts. Because with hallucinogens one is not interested in tracking detail, there is greater suggestibility and dependence on structure, on a leader, on a prior belief, or on the flow of music to guide, interpret, or "carry" one through the experiences.

Whether these drugs produce actual hallucinations or, more commonly, illusions (which the subject usually *feels* are very real but *knows* are not) has sometimes been debated, but not the fact that these perceptions occur. Seeing geometric abstract designs is not unusual. A characteristic effect is the experience of sound triggering color and of the mixing rather than the clear separation of different sensory modalities—called *synesthesia*. For example, sounds may be "seen," or colors "heard." What has just been seen—say, a wall clock—sometimes persists as one focuses on a face. Rather than suppressing a previous perception as we normally do, it may linger. Perceptual boundaries are thus loosened.

The commonly abused hallucinogenic substances can be classified according to their chemical structure. All these hallucinogens are organic compounds, and some are found in nature. Hallucinogenic drugs can be placed in two major groups. The first is known as the indole-type hallucinogens. This family of hallucinogens has in common some structural similarities to the NEUROTRANSMITTER SEROTONIN, suggesting that their mechanism of action could involve the disruption of or some alteration in neurotransmission in NEURONS (nerve cells) that use serotonin as the chemical messenger. The indole-type hallucinogens include lysergic acid derivatives such as LYSERGIC ACID DIETHYLAMIDE (LSD) and other compounds that have structural similarities, such as DIMETHYLTRYPTAMINE (DMT), PSILOCYBIN, and psilocin (see Figure 1).

The second major group of hallucinogens is the substituted phenethylamines (see Figure 2). These are MESCALINE, 2,5-dimethoxy-4-methylamphetamine (DOM or STP), 3,4-methylenedioxyamphetamine, (MDA), and 3,4-methylenedioxymethamphetamine (MDMA or ecstasy). These hallucinogens are structurally related to the phenethylamine-type neurotransmitters, NOREPINEPHRINE, epinephrine, and DOPAMINE. As with the indole-type hallucinogens, the structural similarities of the phenethylamine-type hallucinogens to these natural neurotransmitters may indicate that at least

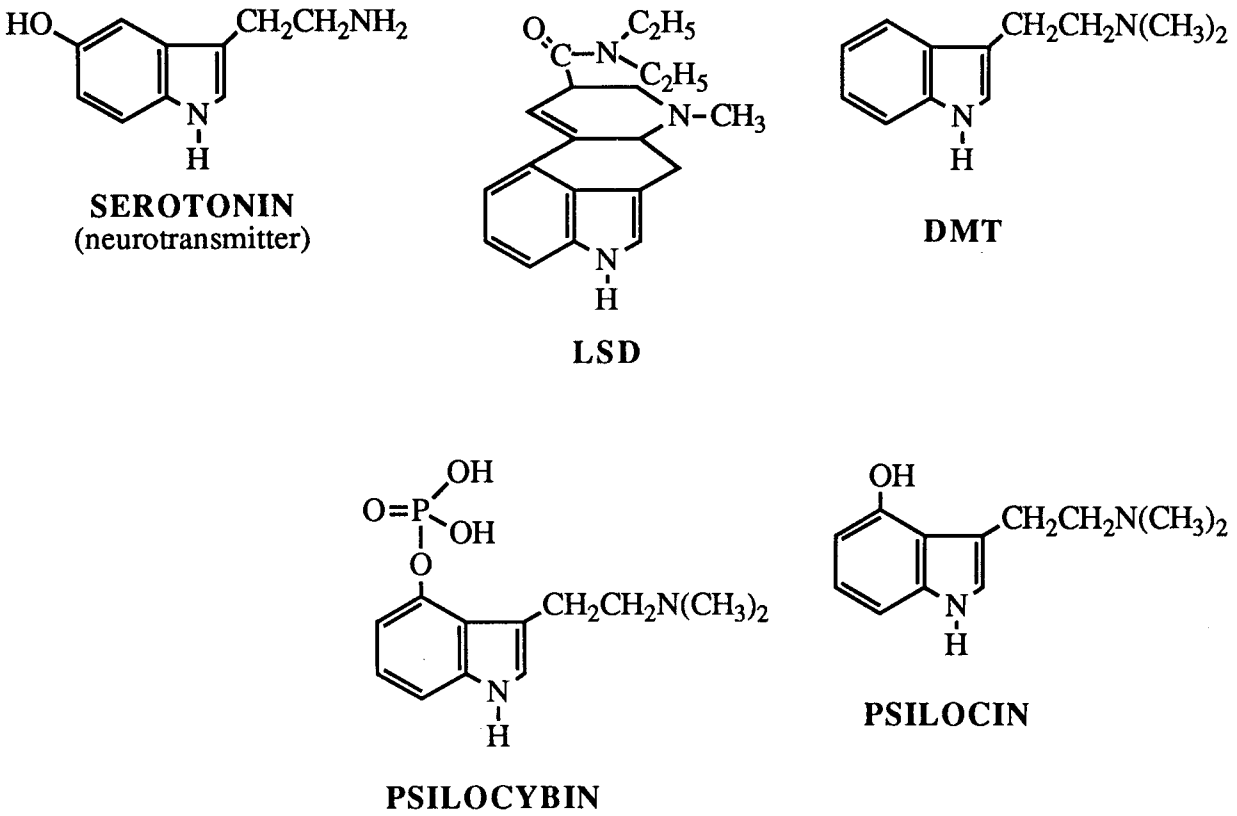

Figure 1
Indole-type Hallucinogens

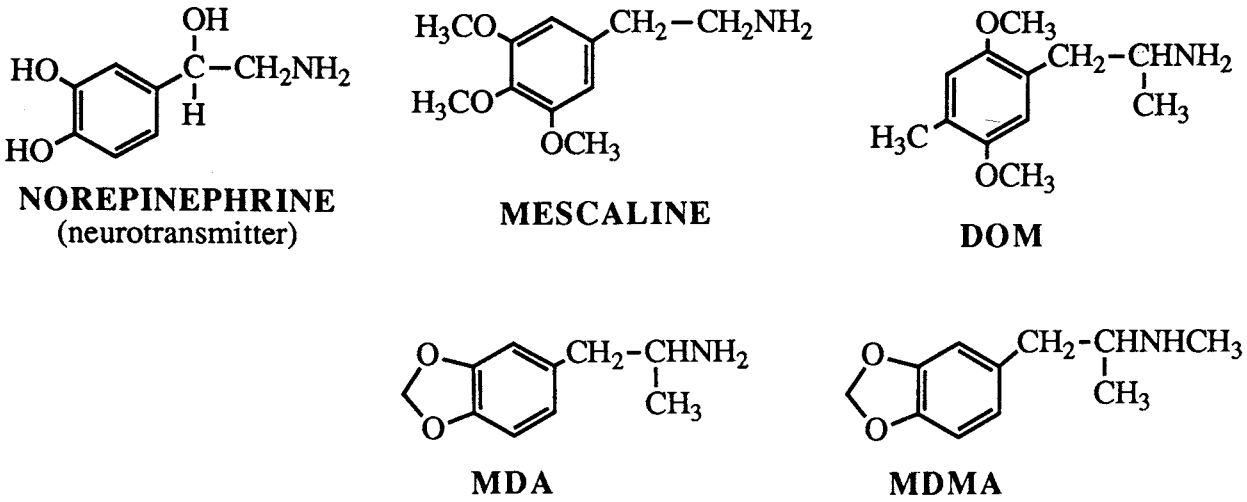

Figure 2
Substituted Phenethylamines

some of their effects involve interactions with systems that use these neurotransmitters. DOM, MDA, and MDMA are synthesized compounds that have structural similarities to the psychostimulant AMPHETAMINE. Thus, they also have some stimulant properties aside from their hallucinogenic activity. They have inaccurately been called psychotomimetic amphetamines, and they are sometimes referred to as stimulant-hallucinogens. It should be pointed out that there are literally hundreds of analogs of the above compounds that have been synthesized and sometimes are found on the street, the so-called DESIGNER DRUGS.

The overall psychological effects of the hallucinogens are quite similar—but the rate of onset, duration of action, and absolute intensity of the effects can differ. As the various hallucinogens differ widely in potency and in the duration of their effects, some of the apparent qualitative differences between hallucinogens may be due, at least in part, to the amount of drug ingested. Aside from their behavioral effects, the hallucinogens also possess significant autonomic activity, meaning that they can affect the sympathetic and parasympathetic nervous systems. The autonomic effects can include marked pupillary dilation and exaggerated reflexes. There may be increases in blood pressure, heart rate, and body temperature. Some of the hallucinogens may initially cause nausea. These autonomic effects of the hallucinogens are variable and may be due, at least in part, to the anxiety state of the user. Acute adverse reactions include panic attacks and self-destructive behavior.

(SEE ALSO: *Ayahuasca; Complications: Mental Disorders; Hallucinogenic Plants; Ibogaine; Plants, Drugs from*)

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HARM REDUCTION *See* Needle and Syringe Exchanges and HIV/AIDS; Netherlands, Drug Use in the; Policy Alternatives

HARRISON NARCOTICS ACT OF 1914

The first international drug-control initiative, the 1909 SHANGHAI OPIUM COMMISSION, brought the international community together in efforts to curb the illicit traffic and consumption of OPIUM, a NARCOTIC drug. The Shanghai Commission encouraged participants to enact national legislation that would address the problem of narcotics in their own countries. Representatives of several countries met at the Hague at conferences in 1911 and 1913.

During this period, the U.S. Congress became aware of public opinion favoring PROHIBITION of all "moral evils," especially alcohol and drugs. New York Representative Francis B. Harrison, encouraged by both the Shanghai Commission's directive to enact national legislation to curb narcotics and the reformists in the Progressive movement in the United States who wanted to eradicate drug use completely, introduced two measures—one to prohibit the importation and nonmedical use of opium and one to regulate the production of opium in the United States. Congress enacted the Harrison Act in December 1914 with minimal debate because public opinion considered its passage necessary to combat the "evils" of drugs.

PROVISIONS OF THE HARRISON ACT

Congress regulated drugs by imposing licensing requirements on manufacturers, distributors, sellers, importers, producers, compounders, and dispensers. The Harrison Act required these parties to register with the director of Internal Revenue, within the Treasury Department, and to pay a gradually increasing occupational tax. Congress wanted to monitor the flow of opium and COCA leaves so that government authorities would have records of any transaction involving these drugs. They would be allowed only for limited medical and scientific purposes. Those individuals found in violation of the act faced a maximum penalty of five years in jail, a 2,000 dollar fine, or both.

TREASURY DEPARTMENT REGULATIONS

Congress intended the Harrison Act to generate revenue by imposing taxes on parties involved in the trade, sale, and distribution of drugs. As a result, Congress entrusted enforcement responsibility to the Treasury Department, in particular the Internal Revenue Service and subsequently the Narcotics Unit of the Bureau of Prohibition. The Treasury Department attempted to limit narcotics to medical and scientific use and prevent their illegal diversion by physicians and druggists. The Harrison Act required pharmacists to review prescriptions to determine whether the quantity was unusually large—that is, a suspicious or coerced prescription.

Sales and transfers of narcotics could only be made pursuant to official order forms obtained from the director of Internal Revenue. District offices of the Internal Revenue Service maintained these records for two years. The act permitted a few notable exceptions to form filings. For example, qualified practitioners (physicians, dentists, and veterinarians) could prescribe or dispense narcotics to patients without completing the order forms but were required to maintain records of all the substances distributed. Druggists could also fill lawful prescriptions without completing order forms.

The Treasury Department interpreted the Harrison Act to prohibit drug addicts from obtaining narcotics. Addicts were prohibited from registering and could receive narcotics only through a licensed physician, dentist, or veterinarian. The Treasury Department regulations also prohibited physicians from maintaining a patient-addict on narcotics, a practice frequently used to help addicts avoid severe WITHDRAWAL pain while they were gradually weaned from narcotic DEPENDENCE. The Treasury Department interpreted possession of narcotics as prima facie evidence of a Harrison Act violation, and the burden of proof shifted to the suspect, who had to document that the narcotics were obtained legally.

The Treasury Department enforced the Harrison Act primarily through warnings. At times, however, the department charged physicians and druggists with conspiracy when authorities arrested an individual who possessed narcotics without a prescription made in good faith, and a connection could be made that the physician or the druggist provided the narcotics.

THE HARRISON ACT AND U.S. DRUG POLICY

Many critics of the Harrison Act argue that the legislation created more problems than it solved. In particular, they charge that the measure failed to eradicate the narcotics problem, primarily because it failed to prohibit the sale and distribution of MARIJUANA. In addition, detractors argue that the act did not resolve the issue of whether drug addicts should be treated as criminals or as patients requiring medical treatment. They also contend that the courts hampered the Treasury Department's enforcement authority. Specifically, courts prohibited the Treasury Department from seizing narcotics, interpreting the Harrison Act to serve as a revenue, rather than as a penal, measure. After passage of the Harrison Act, illicit use of narcotics increased initially as a result of these omissions or ambiguities.

Despite these criticisms, the Harrison Act is significant because it led to a national focus on the dangers of narcotics and drug abuse. Most important, the Harrison Act served as the impetus for further legislation, such as the 1970 Controlled Substances Act, all of which attempt to combat the illegal sale, distribution, and consumption of narcotics and other abusable substances in the United States, while ensuring their availability for medical purposes.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy; Britain, Drug Use in; Legal Regulation of Drugs and Alcohol; Opioids and Opioid Control: History; Psychotropic Contention; Rolleston Report; Treatment: History of*)

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HASHISH Hashish is the Arabic word for a particular form of CANNABIS SATIVA; it came into English at the end of the sixteenth century. Hashish is the resin derived principally from the flowers, bracts, and young leaves of the female hemp plant. The resin contains cannabinoids—the one of major interest being TETRAHYDROCANNABINOL (THC). The THC content will vary depending upon the composition of the hashish, but often it is about 4 percent or more. Usually the resinous portion is sticky enough to allow the material to be compressed into a wafer or brick. Some preparations contain only the resin and are known as hashish oil. Similar preparations of the resinous material and flowering tops of the plant have been given a variety of names in different regions—*charas* in India, *esvar* in Turkey, *anascha* in areas of the former USSR, *kif* in Morocco and parts of the Middle East.

One of the ways in which hashish is prepared is to boil *Cannabis* leaves in water to which butter has been added. THC, being extremely fat-soluble, binds with the butter, which can then be used for making various confections, cookies, and sweets; these are eaten to obtain the effects of the drug. Although hashish is often taken by mouth, it can also be smoked, just as MARIJUANA is.

Hashish was introduced to the West in the middle of the nineteenth century by a French psychiatrist, Moreau de Tours, who experimented with the drug as a means of understanding the phenomenon of mental illnesses. He not only experimented on himself but on a coterie of friends of considerable literary talent. These included Theophile Gautier, Alexander Dumas, and Charles Baudelaire. This group named themselves "Le Club des Haschschins" or "The Club of Hashish-Eaters." The lurid descriptions of the drug effects by these talented writers no doubt helped popularize the drug. Most of their accounts dwelt on beautiful HALLUCINATIONS and a sense of omnipotence.

TABLE 1
Net Hashish Production, in Tons

Source Country		U.S. Measure	Metric Measure
Lebanon	1990	110	100
	1991	600	545
Pakistan	1990	220	200
	1991	220	200
Afghanistan	1990	330	300
	1991	330	300
Morocco	1990	94	85
	1991	94	85
TOTAL	1990	754	685
	1991	1,244	1,130

SOURCE: *International Narcotics Control Strategy Report 1992.*

Doses must have been large, since the effects described are more characteristic of HALLUCINOGENIC DRUGS than effects experienced by present-day users (smokers) of marijuana.

Hashish was introduced into England at about the same time, by an Irish physician, O'Shaughnessy, who had spent some time in India, where he had become familiar with it. The material was soon hailed as a wonder drug, being used for all sorts of complaints: PAIN, muscle spasms, convulsions, migraine headaches, and inflamed tonsils. As most of the preparations were weak and the doses used were small, any beneficial effects might be attributable to a placebo effect.

A preparation, Tilden's Extract of Cannabis Indica, became a popular remedy in the United States in the 1850s. An amateur pharmacologist, Fitz Hugh Ludlow, used this preparation for self-experiments in which he was able to explore its hallucinogenic properties. He may have become somewhat dependent on hashish but finally gave it up. His descriptions of the effects of the drug were similar to what had previously been experienced by Asian users: euphoria and uncontrollable laughter; altered perceptions of space, time, vision, and hearing; synesthesias and depersonalization.

Hashish is currently the most potent of all *Cannabis* preparations: A lot of drug effect is packed into a small parcel. Regulation of the dose is difficult because of its variable potency, and labels for street drugs are notoriously unreliable, however. What may be sold as hashish may often be closer to ordinary marijuana in potency.

(SEE ALSO: *Amotivational Syndrome; Creativity and Drugs; Epidemics of Drug Use; Marihuana Commission; Plants, Drugs from*)

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HAZELDEN CLINICS *See* Treatment Programs/Centers/Organizations: An Historical Perspective

HEART DAMAGE *See* Alcohol: Complications; Cocaine; Tobacco: Medical Complications

HEMP In the narrow sense, hemp refers to a fiber derived from certain strains of CANNABIS SATIVA, a bushy herb that originated in ASIA. In the broader sense, it also denotes the other use of the plant, as a source of MARIJUANA. Although *Cannabis sativa* is generally considered to be a single species, two genetic strains show considerable differences. One is used for fiber production and has been so used for centuries to make rope, floor coverings, and cloth. Hemp plants have been grown for this purpose as commercial crops in Asia and even in colonial America; during World War II, they

Figure 1
Hemp Plant

were grown in the midwestern United States when the Asian supply was unavailable.

The other strain of the hemp plant produces a poor fiber but has a relatively high drug content; it is used for its PSYCHOACTIVE effect. Near the end of the nineteenth century, the Indian Hemp Drug Commission (1895) produced one of the first major assessments of *Cannabis* as a drug, finding it not a major health hazard. Consequently, it remains in legal use in India for both medicinal and social purposes, where it is called BHANG.

(SEE ALSO: *Plants, Drugs from*)

LEO E. HOLLISTER

HEROIN MORPHINE was first identified as the pain-relieving active ingredient in OPIUM in 1806. But morphine was not free of the habit-forming and toxic effects of opium. By the late nineteenth century, the idea of modifying molecules to change their pharmacological actions was well established. It seemed quite reasonable to use this approach to develop new chemical entities that might be free of the problems seen with morphine. In Germany, in 1898, H. Dresser introduced such a new drug—3,6-diacetylmorphine—into medical use; it was named there by the Bayer Company, which produced and marketed it, named it heroin (presumably from *heroisch*, meaning "heroical"), because it was more potent than morphine.

Although heroin is structurally very similar to morphine, it was hoped that it would relieve PAIN without the tendency to produce ADDICTION. Turn-of-the-century medical writings and advertisements, both in Europe and the United States, claimed that heroin was effective for treating pain and cough. Many suggested that it was less toxic than morphine and was nonaddictive. A few even suggested that heroin could be a nonaddicting cure for the morphine habit. Clearly, this was not the case, and within a year or two of its introduction, most of the medical community knew so. By the 1920s, heroin had become the most widely abused of the OPIATES.

PHARMACOLOGY

Heroin is a white powder that is readily soluble in water. The introduction of just two esters onto the morphine molecule changes the physical properties of the substance such that there is a significant increase in solubility, permitting solutions with increased drug concentrations. A more subtle advantage of heroin is its greater potency compared to morphine. The volume of drug injected may be particularly important when high doses are used. Thus, 1 gram of heroin will produce the effects of 2 to 3 grams of morphine; by converting morphine to heroin, producers increase both the potency and the value of the drug.

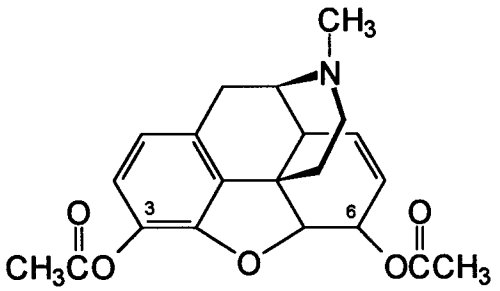

Figure 1
Heroin

Following injection, heroin is very potent, with the ability to cross the blood-brain barrier and enter the brain. This barrier results from a unique arrangement of cells around blood vessels within the brain, which limits the free movement of compounds. Many factors contribute to the barrier—in general, the less polar a drug, the more rapidly it enters the brain. Heroin, however, has a very short half-life in the blood (amount of time that half the drug remains). It is rapidly degraded by esterases, the enzymes that break ester bonds. The acetyl group at the 3-position of the molecule is far more sensitive to these enzymes than the acetyl group at the 6-position. Indeed, the 3-acetyl group is attacked almost immediately after injection and, within several minutes, virtually all the heroin is converted to a metabolite, 6-acetylmorphine. The remaining acetyl group at the 6-position is also lost, but at a slower rate. Loss of both acetyl groups generates morphine. It is believed that a combination of 6-acetylmorphine and morphine is responsible for the actions of heroin.

MEDICINAL USE

The pharmacology of heroin is virtually identical to that of morphine. This probably reflects its rapid conversion to 6-acetylmorphine and morphine. De-

Mexican brown heroin and Southeast Asian heroin. Although pure heroin is a white powder, most heroin that is sold on the street varies in color from white to dark brown, with an average purity of 35 percent. (Drug Enforcement Administration)

tailed studies comparing the actions of heroin and morphine in cancer patients with severe pain have shown very little difference between the two agents, other than simple potency. Heroin may have a slightly more rapid onset of action than morphine and it is certainly two to three times as potent (presumably due to its greater facility in crossing the blood-brain barrier). This difference in potency is lost with oral administration. The pain relief (analgesia) from both agents is comparable when the doses are adjusted appropriately. At equally effective ANALGESIC doses, even the euphoria seen with heroin is virtually identical to that of morphine. From the clinical point of view, there is little difference between one drug and the other. Both are effective analgesics and can be used beneficially in the treatment of severe pain. Heroin is more soluble, which makes it somewhat easier to give large doses by injection, with smaller volumes needed. Many of the similar semisynthetic agents, such as HYDROMORPHONE, however, are many times more potent than heroin and offer even greater advantages.

One widespread use of heroin in the United Kingdom was in the early formulations of Brompton's Cocktail, a mixture of drugs designed to relieve severe pain in terminal cancer patients. The heroin employed in the original formula is now typically replaced with morphine without any loss in effectiveness. For many years, some groups have maintained that heroin is more effective in the relief of cancer pain than morphine is. Careful clinical studies show that this is not true, but the most important issue is using an appropriate dose. Thus, heroin offers no major advantage over morphine from the medical perspective.

STREET HEROIN

Since heroin has no approved medical indications in the United States, it is only available and used illicitly. The marked variability of its purity and the use of a wide variety of other substances and drugs to "cut" street heroin poses a major problem. This inability to know what is included in each drug sale makes the street drug more than doubly dangerous. Typically, heroin is administered intravenously, which provides a rapid "rush," a euphoria, which is thought to be the important component of heroin's addictive properties. It can be injected under the skin (subcutaneously, SC) or deep into the muscle (intramuscularly, IM). Multiple intravenous injections leave marks, called tracks, in a much-used injection site, which often indicate that a person is abusing drugs; but heroin can also be heated and the vapors inhaled through a straw (called "chasing the dragon"). It can also be smoked in a cigarette. While the heat tends to destroy some of the drug, if the preparation is pure enough, a sufficient amount can be inhaled to produce the typical opiate effect.

Heroin use is associated with TOLERANCE AND DEPENDENCE. Chronic use of the drug leads to a decreased sensitivity toward its euphoric and analgesic actions, as well as to dependence. Like morphine, the duration of action of heroin is approximately 4 to 6 hours. Thus, addicts must take the drug several times a day to prevent the appearance of WITHDRAWAL signs. Many believe that the need to continue taking the drug to avoid withdrawal enhances its addictive potential.

Patients taking opiates medicinally can be taken off them gradually, without problems. Lowering the opiate dose by 20 to 25 percent daily for two or

three days will prevent severe withdrawal discomfort and still permit rapid taper off the drug. Abrupt withdrawal of all of the drug is very different—and leads to a well-defined abstinence syndrome that is very similar for both heroin and morphine. It includes eye tearing, yawning, and sweating after about eight to twelve hours past the last dose. As time goes on, people develop restlessness, dilated pupils, irritability, diarrhea, abdominal cramps, and periodic waves of gooseflesh. The term *cold turkey* is now used to describe abrupt withdrawal with the associated gooseflesh. The heroin withdrawal syndrome peaks between two and three days after stopping the drug, and symptoms usually disappear within seven to ten days, although some low-level symptoms may persist for many weeks. Babies of mothers dependent on opiates are born dependent, and special care must be taken to help them withdraw during their first weeks. Medically, although miserable, heroin withdrawal is seldom life threatening—unlike withdrawal from alcohol, which can sometimes be fatal.

OVERDOSE

Overdosing is a common problem among heroin addicts. The reason is not always clear, but wide variation in the purity of the street drug can make it difficult for the addict to judge a dose. Some impurities used to cut the drug may be toxic themselves. With OVERDOSE, a person becomes stuporous and difficult to arouse. Pupils are typically small and the skin may be cold and clammy. Seizures may occur, particularly in children or babies. Breathing becomes slow, and cyanosis—seen as a darkening of the lips to a bluish color—may develop, indicating inadequate levels of oxygen in the blood. With respiratory depression, blood pressure may then fall. These last two signs are serious, since most people who die from overdose, die from respiratory failure. Complicating the problem is the fact that many addicts may have taken other drugs, used alcohol, and so on. Some of them may have been taken on purpose, and some may have been a part of the street drug.

NALOXONE can readily reverse some opiate problems, since it is a potent opiate ANTAGONIST. This drug binds to opiate RECEPTORS and can reverse morphine and heroin actions. The appropriate dose may be a problem, however, since naloxone can also precipitate a severe abstinence syndrome in a dependent person.

(SEE ALSO: *Addiction: Concepts and Definitions; International Drug Supply Systems; Methadone Maintenance Programs; Opioids: Complications and Withdrawal; Treatment: History of*)

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GAVRIL W. PASTERNAK

HEROIN EPIDEMICS *See* Epidemics of Drug Abuse

HEROIN: THE BRITISH SYSTEM What is sometimes referred to as the British "system" of drug control is not really a system; rather, it is a set of principles and programs that represent one form of societal response to HEROIN use and OPIATE DEPENDENCE. The principles encompass the idea that government ought to offer public-health and medical programs that will help contain Britain's heroin problem, in addition to its response in the form of law enforcement. In BRITAIN, the concept of punishing heroin-dependent individuals for dependence as such is as alien as punishing people for becoming infected with syphilis or needing insulin for diabetes.

A key element in this system is allowing medical practitioners to provide maintenance doses of OPIATES or opioid drugs (sometimes including heroin as well as METHADONE and other opioids) when a diagnosis of heroin dependence can be substantiated. The initial programmatic efforts allowed for the prescribing of such drugs by general medical practitioners; but more recently, responsibility for treatment of opioid-dependent persons has shifted to government-run specialized Drug Dependency Units.

BACKGROUND

Drug control in Britain was established between 1910 and 1930, with a solid grounding in public health and medical practice. This British approach to drug problems as public-health problems seemed especially attractive as an alternative to U.S. drug prohibition policies, even when the heroin problem in the United States was relatively small, back before 1960. Thus, beginning in the late 1940s, some Americans started to advocate the use of the British system in the United States—that is, a nonpunitive, public-health approach to the treatment of drug dependence, especially dependence on heroin.

In 1960, the drug problem was essentially a nonissue in the political life of Britain, although the structures for control in the two countries remained very different. In the United States, a prohibitionist policy continued in place whereby criminal penalties were imposed for heroin possession and use—and sometimes for being addicted to heroin. Physicians rarely treated opiate addicts and could not legally provide a known addict with opiates on a maintenance basis. As a result, from early in the twentieth century, virtually all heroin addicts purchased supplies from illegal heroin sellers. With the exception of a brief time during which maintenance programs were available, relatively few addicts sought drug treatment from doctors, and treatment for heroin dependence often was available only at two federal narcotic hospitals and select public and private facilities. In NEW YORK and CALIFORNIA, in particular, large numbers of heroin abusers were arrested and imprisoned for heroin sales, for possession, or for other crimes sometimes committed to gain funds to purchase illegal heroin (e.g., robbery, burglary).

In contrast, by 1960, Britain had had many years of experience with a "medical" or "public-health" policy for controlling heroin and opiates (originating with the ROLLESTON REPORT of 1926). Fewer than 100 heroin addicts and fewer than 500 abusers of all drugs were known in Britain in 1960. Persons identified by a doctor as being addicted to heroin or other dangerous drugs could be (and usually were) treated by a private practitioner. The physician was required to notify the Home Office of the names of the addicts but was at liberty to prescribe heroin or opiates for them in any amounts for long time periods. Their treatment became funded by the National Health Service after World War II, like any other medical service. No other treatment (at a clinic, hospital, or nonmedical facility) was available. Penalties for the illegal sale of heroin or opiates carried sanctions of less than a year and were rarely imposed. Few British prisoners were heroin addicts.

British drug policy has been and continues to be set primarily by Home Office staff in collaboration with leading physicians and addiction specialists. British law-enforcement and criminal-justice practitioners were largely excluded from policymaking—whereas their counterparts in the United States have a primary role in formulating American drug policy. Following the Rolleston precedent, several special committees issued reports establishing the basic directions of British drug policy. The first Brain Committee (1958) reaffirmed the Rolleston recommendation to provide heroin and allow maintenance doses of opiates; it opposed U.S.-sponsored proposals to prohibit heroin manufacture in Britain.

CHANGING MEDICAL POLICIES ON DRUG CONTROL

The situation changed in the early 1960s, however, and, based on recommendations of the second Brain Committee (1964), clinics for controlling and containing the heroin problem were implemented under the Dangerous Drug Act Regulations in 1968. Responsibility for the treatment of addicts generally was shifted from general practitioners (GPs) to Drug Dependency Units (DDUs). When a heroin abuser seeks treatment from a GP, however, the doctor can refuse treatment, refer the patient to a DDU, or provide declining methadone doses over six months (called long-term detoxification in the United States) or provide regular methadone maintenance (although this is rarely done by a GP).

The DDUs or drug clinics provide a range of services funded by the National Health Service. In 1989, thirty-five DDUs operated in Britain and were directed by consulting psychiatrists who specialized in addiction treatment and prescribing. In smaller towns without clinics, one or two GPs can be licensed by the Home Office to provide treatment for addicts in the area. New applicants are interviewed and their urine tested to verify opiate use. The clinic physician develops a treatment plan with the patient, arranges weekly conferences, and

mails the prescription directly to a local pharmacy; it will be filled for the client on a daily basis. The Home Office also convenes meetings with several DDU directors to discuss common policies and practices, and to recommend approval or removal of licenses, when necessary, for physicians to prescribe dangerous drugs.

When the DDUs opened, most clinics made decisions to shift patients receiving prescriptions for injectable heroin onto injectable methadone. The pharmacist dispensed needles, syringes, and ampoules of methadone.

Over the period 1975 to 1983, many clinic directors shifted most patients from injectable to oral methadone maintenance. In the early 1980s, as illegal supplies of heroin became common in British cities, many clinics shifted away from oral methadone maintenance. Instead, the treatment policy at several clinics was to provide gradual withdrawal (detoxification in the United States); rarely were patients provided with long-term maintenance doses. As AIDS was tied to shared needles and syringes by injecting addicts, prevention became an important subgoal of drug treatment; however, new emphasis was then placed on oral methadone maintenance. In the early 1990s, the DDUs had heroin-abusing clients, many of whom received gradual reduction (detoxification) and others who received maintenance on methadone. Relatively few received prescriptions for injectable methadone or heroin, even though DDU doctors could legally and appropriately provide such services.

A continuing controversy within Britain in the 1990s has been whether the clinic system could stem or contain the heroin problem, and whether the clinic's shift away from prescribing heroin and injectable drugs contributed to the growth of black-market heroin. In discussion groups, some experts argued that many black-market heroin users would seek treatment if the clinics returned to prescribing injectable heroin or methadone. Such a policy also might reduce addict crime and prevent transmission of the AIDS virus. This, however, would change the profile of patients: Clinic directors would have to deal with addicts who have no intention of stopping heroin use.

The British have amended the Dangerous Drug Act several times since 1960, thereby making the illegal sale of heroin, cocaine, and marijuana criminal offenses. Although the vast majority of drug arrestees are only "cautioned," even after repeated instances of offense, many illegal sellers and heroin abusers arrested for robbery, burglary, and theft can be and are imprisoned. Thus, an increasingly larger proportion of British prisoners are heroin addicts. Between 1979 and 1984, seizures of illegal drugs went up tenfold, incarcerated drug offenders went up fourfold, and the consumption of heroin increased by 350 percent—but heroin prices decreased by 20 percent.

Rise of Nonmedical Drug Treatment. The increase in black-market heroin, substantial increases in heroin abusers who avoid the DDUs, apparent increase in penal sanctioning, and a host of complex issues have led to dissatisfaction with the original British System, with its medical model of drug treatment. Influenced by U.S. therapeutic communities and outpatient local programs that promote a drug-free environment, British social service agencies have begun developing similar programs thereby "reaching out" to clients and providing alternative services in a context that is different from the practice settings dominated by the consulting psychiatrists at DDUs.

Other emerging British programs are increasingly built around a philosophy of "harm reduction." This emphasizes informing people of safer ways to take drugs for those who will continue to do so, helping addicts recognize drug-related problems (e.g., infections or diseases), and making sterile injection equipment and/or drug treatment available with minimal restrictions. The program's staff also suggests alternative ways of altering consciousness or seeking pleasure.

AIDS Prevention. Since the years 1984 to 1985, the British have been international leaders in devising innovative programs to reduce the spread of the AIDS virus. Because of the legal provision of opiates by physicians and DDUs, the sale of syringes was never prohibited nor seriously constrained. Addicts using black-market heroin could always purchase sterile needles cheaply as well as receive instructions on safe injection practices although in some areas pharmacists might refuse to sell them to addicts.

Gerry Stimson, a sociologist who had conducted studies of heroin addicts from 1960 through the 1970s, became a leading government consultant in the 1980s in formulating British AIDS prevention policies. Together with other experts, he recommended establishing syringe exchanges to promote safe disposal of used needles (possibly infected with

the AIDS virus) and to reach injecting drug users who avoid the clinics. His subsequent research established the facts that untreated addicts could be attracted to these exchanges but that retention rates were low. Possibly as a result of these efforts, the AIDS infection rate in Britain is much lower than that in many cities of the United States.

Heroin Abuse. After 1960, several major increases in heroin use and abuse occurred in Britain. In the early 1960s, a few British physicians began prescribing large amounts of legal heroin to private patients, some of whom resold it to other people. The number of known heroin abusers grew to 2,240 in 1968 and then increased slowly during the 1970s. In the early 1980s, however, a major increase in illegal importation of heroin to Britain was followed by an epidemic of heroin use in that country—thus, 12,500 heroin abusers were reported to the Home Office in 1984. In the mid-1990s, many heroin abusers avoid clinics and doctors and are not reported to the Home Office. Therefore, the actual number of regular heroin abusers in Britain now is estimated to be between 50,000 to 100,000.

CONCLUSION

Since the 1960s, the British system of drug control has evolved and changed in many important ways. Although the heroin problem expanded dramatically in the 1980s, the major policy decisions of the Rolleston Report have continued to govern the British approach. The British government continues to collaborate closely with medical and public-health experts. Treatment practices have been refined by experience and practical considerations, but not because of imposition by government fiat. Prohibition of heroin did not occur and punishment of drug abusers remains a secondary consideration in British policymaking (but is still a dominant consideration in the United States). Since 1960, the British heroin problem has grown and become complex. Drug-policy and treatment response have become diverse and, therefore, there is less of a clear "system."

In comparison with the situation in the United States, British policymakers and the general public favor public-health considerations over other moral concerns. Some British newspapers do promote "dope fiend" images and demand punitive responses—and the American "drug free" and "just say no" philosophies are often articulated. Nonetheless, British drug policy and funding are primarily directed by medical and public-health specialists. This means that heroin addicts and drug abusers are not as heavily stigmatized as they are in the United States.

The British public accepts the idea of providing heroin and methadone as medicine, has few moral qualms about addicts, and little fear of needles. Lacking the harsh and punitive moral consensus against drugs that prevails in the United States, the British government has considerable latitude to experiment with differing policies, to shift treatment practices to accord with practical experience, and to keep modifying its policy responses to the ever-changing drug scene. Whether the British system could work in the United States, which is much larger and more populous than Great Britain, remains an open question.

(SEE ALSO: *British System of Drug-Addiction Treatment; Needle and Syringe Exchanges and HIV/AIDS; Policy Alternatives*)

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BRUCE D. JOHNSON

HEROIN TREATMENTS *See* Methadone Maintenance; Treatment

HEROIN WITHDRAWAL *See* Opioid Complications and Withdrawal

HEXANE *See* Inhalants

HIGH *See* Slang and Jargon

HIGH SCHOOL SENIOR SURVEY The use of illegal drugs by large numbers of young people in the United States became an issue of considerable concern during the late 1960s and early 1970s. At that time, there were few accurate data available to assess the extent of use on a national basis. In 1975, Lloyd Johnston and Jerald Bachman of the University of Michigan initiated *Monitoring the Future: An Ongoing Study of the Lifestyles and Values of Youth*, which was intended to address this lack of information.

One of the major purposes of the study was (and is) to develop an accurate picture of the nature and extent of drug use among young people. An accurate assessment of the amount and extent of illicit drug use in this group is a prerequisite for rational policy making. Reliable and valid data on prevalence are necessary to determine an appropriate allocation of resources and to prevent or correct misconceptions. Reliable and valid data on trends allow for early detection of emerging problems and make it possible to assess the impact of external events, including historical events and deliberate policy changes.

In addition, the study was designed to monitor factors that might help explain the observed changes in drug use—that is, it was intended to serve both an epidemiological function (to learn how many young people use drugs) and an etiological function (to study why young people use drugs). The factors measured included attitudes toward drugs, peer norms and behaviors in regard to drugs, beliefs about the dangers of drugs, perceived availability of drugs, religious attitudes, and various life-style factors. The monitoring of these factors has, among other things, provided the country with valuable information. A particular contribution has been to help address a central policy-making question in the nation's war on drugs: The relative importance of supply eduction versus demand reduction in bringing about some of the observed declines in drug use.

STUDY DESIGN

The core feature of the design is an annual survey of each new high school senior class, beginning with the class of 1975. Each year approximately 16,000 seniors are surveyed in approximately 135 public and private high schools that have been scientifically elected to provide an accurate, representative cross section of high school seniors throughout the coterminous United States. Data are collected following standardized procedures via closed-ended questionnaires administered in classrooms by University of Michigan representatives and their assistants.

In 1991, the project was expanded to include nationally representative samples of students from the eighth and tenth grades as well as from the twelfth grade. Approximately 18,000 eighth graders and 16,000 tenth graders are surveyed annually, using procedures similar to those used in the twelfth grade surveys.

One limitation of the design is that it does not include in the target population the young men and women who drop out of high school before graduation, and who make up between 15 and 20 percent of each age group nationally, according to U.S. Census statistics. The omission of high school dropouts does introduce biases in the estimation of certain characteristics of the entire age group, but, because the dropouts are a relatively small proportion of the entire group, the bias due to their omission is small. Because relatively few adolescents drop out before the end of tenth grade, the bias is particularly small for the eighth and tenth graders. It should also be noted that because any bias resulting from exclusion of the dropouts usually remains constant from year to year, their exclusion should introduce little or no bias in estimates of change or trends.

An issue that is relevant to the study of sensitive behaviors, such as drug use, is the extent to which respondents will answer honestly. Considerable inferential evidence suggests that the procedures used in this study produce largely valid data. This evidence includes the following points: Large proportions of respondents report using illegal substances; various drugs exhibit trends in different ways over time; there are very few missing data in response to questions on drug use, even though respondents are instructed not to answer questions they would prefer not to answer; the high correlations with other behaviors such as grades, delinquency, religious attitudes, and truancy indicate a high degree of construct validity; a high degree of consistency can be noted over time in individuals' reports (that is, the responses

are reliable); and other factors that are discussed in detail in other publications (see Johnston, O'Malley, & Bachman, 2000; O'Malley, Bachman, & Johnston, 1983).

MAJOR FINDINGS

One dramatic finding that emerged from the Monitoring the Future surveys was the decrease between about 1980 and 1992 in young Americans involved in the use of illicit drugs.

Illicit Drugs. Annual use of any illicit drug (that is, any use in the past twelve months) peaked among high school seniors in 1979, when more than half (54%) of all high school seniors reported having used such a drug. This peak occurred following a rise in the late 1970s—from 45 percent in 1975, when the first reliable national data were collected. By 1992, the proportion had fallen to 27 percent, half of the peak rate.

The statistics for lifetime prevalence are also dramatic. In the peak year of 1981, 66 percent of the graduating class reported having used an illicit drug at some point in their lifetime. By 1992, that percentage was down by about one third, to 41 percent.

Unfortunately, a second dramatic finding that has emerged from the Monitoring the Future surveys is an increase in the numbers of young Americans involved in the use of illicit drugs during the 1990s. After reaching a low of 27 percent in 1992, annual use among seniors was back up to 42 percent in 1999. Lifetime use was back to 55 percent.

Increases were particularly sharp among the eighth and tenth graders. No data are available before 1991, so longer term trends are not so clear. However, it is clear that there were significant increases in the 1990s. Among eighth graders in 1991, 11 percent had used an illicit drug in the past twelve months; that figure increased to 21 percent by 1999 (and actually peaked in 1996 at 24%). Similarly, among tenth graders, annual use increased from 21 percent in 1991 to 36 percent (and peaked at 39% in 1997).

Among the various illicit drugs, marijuana is the most prevalent. The use of marijuana, as indicated by its annual prevalence, peaked among high school seniors in 1979, when a majority (51%) reported that they had used it in the past twelve months, and it steadily declined after that, reaching a low of 22 percent in 1992. The annual prevalence, thus cut by more than half, declined from one in two seniors in the class of 1979 to less than one in four seniors in the class of 1992. However, by 1999 the figure was back to 38 percent, so that well over one in three seniors had used marijuana in the past twelve months.

A particularly striking trend in marijuana use occurred between 1975 and 1978, when the proportion of seniors who reported using marijuana on a daily or near-daily basis in the past thirty days increased from 6 percent to an unprecedented 10.7 percent. This figure subsequently came down by more than 80 percent and stood at 2 percent in 1992; by 1999 it was back to 6 percent, exactly where it was in 1975.

Among eighth graders, annual marijuana use increased from 6.2 percent in 1991 to 17 percent in 1999 (peaking at 18% in 1996). Among tenth graders, annual marijuana use almost doubled between 1991 and 1999, from 17 percent to 32 percent (peaking at 35% in 1997).

Never as common as marijuana, cocaine became the drug on which the most attention was focused during the mid-1980s, when the national concern about the drug epidemic was at its highest level. The concern with cocaine was well founded because its use, unlike that of marijuana, had not begun to decline in the very early 1980s. As with marijuana, the daily use of cocaine had increased substantially between 1975 and 1979: Annual prevalence doubled from 5.6 percent to 12.0 percent. Several years followed during which there was little change, with annual prevalence reaching a peak of 13 percent in both 1985 and 1986. A period of decline then ensued during which annual use declined to 3.1 percent in 1992; this was the lowest value recorded since reliable data had begun to be collected in 1975. Like marijuana, however, use increased in the 1990s, and by 1999 annual cocaine among seniors had doubled, reaching 6.2 percent.

These data refer to the use of any form of cocaine, including crack cocaine. Crack cocaine first appeared in the early 1980s and became a significant factor among the illicit drugs in the mid-1980s. It was first assessed on a national basis in 1986, and its annual prevalence among high school seniors at that time was recorded at a disturbingly high 4.1 percent. That first reading turned out to be a peak level, and the use of crack cocaine declined thereafter, reaching 1.5 percent

TABLE 1
Trends in Annual Prevalence of Use of Various Drugs among Eighth, Tenth, and Twelfth Graders

	1975	*1976*	*1977*	*1978*	*1979*	*1980*	*1981*	*1982*	*(Percent who used in 1983*	*1984*	*1985*
Any Illicit Drug[a]											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	45.0	48.1	51.1	53.8	54.2	53.1	52.1	49.4	47.4	45.8	46.3
Any Illicit Drug Other Than Marijuana											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	26.2	25.4	26.0	27.1	28.2	30.4	34.0	30.1	28.4	28.0	27.4
Marijuana/ Hashish											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	40.0	44.5	47.6	50.2	50.8	48.8	46.1	44.3	42.3	40.0	40.6
Inhalants											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	–	3.0	3.7	4.1	5.4	4.6	4.1	4.5	4.3	5.1	5.7
LSD											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	7.2	6.4	5.5	6.3	6.6	6.5	6.5	6.1	5.4	4.7	4.4
MDMA (Ecstasy)											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	–	–	–	–	–	–	–	–	–	–	–
Cocaine											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	5.6	6.0	7.2	9.0	12.0	12.3	12.4	11.5	11.4	11.6	13.1
Crack Cocaine											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	–	–	–	–	–	–	–	–	–	–	–

NOTE: See Johnston, O'Malley, & Bachman (2000) for more specific details about measures.
[a]Use of "any illicit drugs" includes any use of marijuana, hallucinogens, cocaine, or heroin, or any non-medical use of other opiates, amphetamines, barbiturates, or tranquilizers.

in 1992. Its lifetime prevalence reached a peak of 5.4 percent among the high school class of 1987 but declined to 2.6 percent by 1992. Use of crack cocaine increased during the 1990s, reaching a lifetime prevalence of 4.6 percent in 1999, and an annual prevalence of 2.7 percent. These figures are still below the peak levels reached in the mid 1980s.

Similar trends were observed among eighth and tenth graders in the 1990s, though at lower absolute levels.

Although not necessarily illicit drugs, inhalants are sometimes used illicitly for the purpose of getting high. This particular behavior is generally more often seen among younger students rather than among high school seniors. In 1999, for exam-

last twelve months)													
1986	*1987*	*1988*	*1989*	*1990*	*1991*	*1992*	*1993*	*1994*	*1995*	*1996*	*1997*	*1998*	*1999*
–	–	–	–	–	11.3	12.9	15.1	18.5	21.4	23.6	22.1	21.0	20.5
–	–	–	–	–	21.4	20.4	24.7	30.0	33.3	37.5	38.5	35.0	35.9
44.3	41.7	38.5	35.4	32.5	29.4	27.1	31.0	35.8	39.0	40.2	42.4	41.4	42.1
–	–	–	–	–	8.4	9.3	10.4	11.3	12.6	13.1	11.8	11.0	10.5
–	–	–	–	–	12.2	12.3	13.9	15.2	17.5	18.4	18.2	16.6	16.7
25.9	24.1	21.1	20.0	17.9	16.2	14.9	17.1	18.0	19.4	19.8	20.7	20.2	20.7
–	–	–	–	–	6.2	7.2	9.2	13.0	15.8	18.3	17.7	16.9	16.5
–	–	–	–	–	16.5	15.2	19.2	25.2	28.7	33.6	34.8	31.1	32.1
38.8	36.3	33.1	29.6	27.0	23.9	21.9	26.0	30.7	34.7	35.8	38.5	37.5	37.8
–	–	–	–	–	9.0	9.5	11.0	11.7	12.8	12.2	11.8	11.1	10.3
–	–	–	–	–	7.1	7.5	8.4	9.1	9.6	9.5	8.7	8.0	7.2
6.1	6.9	6.5	5.9	6.9	6.6	6.2	7.0	7.7	8.0	7.6	6.7	6.2	5.6
–	–	–	–	–	1.7	2.1	2.3	2.4	3.2	3.5	3.2	2.8	2.4
–	–	–	–	–	3.7	4.0	4.2	5.2	6.5	6.9	6.7	5.9	6.0
4.5	5.2	4.8	4.9	5.4	5.2	5.6	6.8	6.9	8.4	8.8	8.4	7.6	8.1
–	–	–	–	–	–	–	–	–	–	2.3	2.3	1.8	1.7
–	–	–	–	–	–	–	–	–	–	4.6	3.9	3.3	4.4
–	–	–	–	–	–	–	–	–	–	4.6	4.0	3.6	5.6
–	–	–	–	–	1.1	1.5	1.7	2.1	2.6	3.0	2.8	3.1	2.7
–	–	–	–	–	2.2	1.9	2.1	2.8	3.5	4.2	4.7	4.7	4.9
12.7	10.3	7.9	6.5	5.3	3.5	3.1	3.3	3.6	4.0	4.9	5.5	5.7	6.2
–	–	–	–	–	0.7	0.9	1.0	1.3	1.6	1.8	1.7	2.1	1.8
–	–	–	–	–	0.9	0.9	1.1	1.4	1.8	2.1	2.2	2.5	2.4
4.1	3.9	3.1	3.1	1.9	1.5	1.5	1.5	1.9	2.1	2.1	2.4	2.5	2.7

ple, 5.6 percent of twelfth graders reported using inhalants to get high at least once in the past twelve months, compared to 7.2 percent of tenth graders, and 10.3 percent of eighth graders.

The longer term trend in the use of inhalants was slightly upward from its lowest level of 3.0 percent in 1976 (when it was first assessed), to a peak level of 8.0 percent in 1995 (before declining to 5.6% in 1999). Thus, the use of this class of substance, unlike the use of illicit drugs in general, did not show the general decline from 1980 to 1992. Among eighth and tenth graders, annual use levels are not very different between 1991 and 1999: for eighth graders the respective values were 9 percent and 10.3 percent, and for tenth graders they were 7.1 percent and 7.2 percent.

TABLE 1 (Continued)
Trends in Annual Prevalence of Use of Various Drugs among Eighth, Tenth, and Twelfth Graders

	1975	*1976*	*1977*	*1978*	*1979*	*1980*	*1981*	*1982*	*(Percent who used in 1983*	*1984*	*1985*
Heroin											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	1.0	0.8	0.8	0.8	0.5	0.5	0.5	0.6	0.6	0.5	0.6
Other Narcotics											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	5.7	5.7	6.4	6.0	6.2	6.3	5.9	5.3	5.1	5.2	5.9
Amphetamines[b]											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	16.2	15.8	16.3	17.1	18.3	20.8	26.0	20.3	17.9	17.7	15.8
Barbiturates											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	10.7	9.6	9.3	8.1	7.5	6.8	6.6	5.5	5.2	4.9	4.6
Tranquilizers											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	10.6	10.3	10.8	9.9	9.6	8.7	8.0	7.0	6.9	6.1	6.1
Alcohol[c] Any use											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	84.8	85.7	87.0	87.7	88.1	87.9	87.0	86.8	87.3	86.0	85.6

NOTE: See Johnston, O'Malley, & Bachman (2000) for more specific details about measures.
[b]In 1982, the question about amphetamine use was revised; the prevalence rate declined as a result.
[c]In 1993, the question about alcohol use was revised; the prevalence rate declined as a result.

Hallucinogens are the other major class of illicit (or illicitly used) substances that did not evidence declines in the late 1980s and the early 1990s. LSD (lysergic acid diethylamide) in particular is a very significant exception; its use hardly changed among high school seniors, remaining at an annual prevalence of about 5 percent from 1987 to 1991 after a period of some decline. Like marijuana however, there was an increase in the 1990s, reaching 8.8 percent in 1996, the highest value ever recorded. (The lowest recorded value was 4.4 percent in 1985). By 1999, use had declined only slightly, to 8.1 percent.

Very similar patterns of change were evident among eighth and tenth graders in the 1990s, albeit at lower levels.

Substances that generally showed declines during the period from the 1970s to the early 1990s include heroin, opiates other than heroin, amphetamines, barbiturates, and tranquilizers. All of these substances also showed an increase during the mid-1990s.

Thus, five classes of illicitly used drugs had a particularly important impact on appreciable proportions of young Americans: Marijuana, cocaine, amphetamines, LSD, and inhalants. In 1999, they showed annual prevalence rates among high school seniors of 38 percent, 6 percent, 10 percent, 8 percent, and 6 percent, respectively. Among eighth graders, the respective figures were 17 percent, 3 percent, 7 percent, 2 percent, and 10 percent.

In the late 1990s, some "club drugs" appeared on the drug scene. One in particular, MDMA, or

last twelve months) *1986*	*1987*	*1988*	*1989*	*1990*	*1991*	*1992*	*1993*	*1994*	*1995*	*1996*	*1997*	*1998*	*1999*
–	–	–	–	–	0.7	0.7	0.7	1.2	1.4	1.6	1.3	1.3	1.4
–	–	–	–	–	0.5	0.6	0.7	0.9	1.1	1.2	1.4	1.4	1.4
0.5	0.5	0.5	0.6	0.5	0.4	0.6	0.5	0.6	1.1	1.0	1.2	1.0	1.1
–	–	–	–	–	–	–	–	–	–	–	–	–	–
–	–	–	–	–	–	–	–	–	–	–	–	–	–
5.2	5.3	4.6	4.4	4.5	3.5	3.3	3.6	3.8	4.7	5.4	6.2	6.3	6.7
–	–	–	–	–	6.2	6.5	7.2	7.9	8.7	9.1	8.1	7.2	6.9
–	–	–	–	–	8.2	8.2	9.6	10.2	11.9	12.4	12.1	10.7	10.4
13.4	12.2	10.9	10.8	9.1	8.2	7.1	8.4	9.4	9.3	9.5	10.2	10.1	10.2
–	–	–	–	–	–	–	–	–	–	–	–	–	–
–	–	–	–	–	–	–	–	–	–	–	–	–	–
4.2	3.6	3.2	3.3	3.4	3.4	2.8	3.4	4.1	4.7	4.9	5.1	5.5	5.8
–	–	–	–	–	1.8	2.0	2.1	2.4	2.7	3.3	2.9	2.6	2.5
–	–	–	–	–	3.2	3.5	3.3	3.3	4.0	4.6	4.9	5.1	5.4
5.8	5.5	4.8	3.8	3.5	3.6	2.8	3.5	3.7	4.4	4.6	4.7	5.5	5.8
–	–	–	–	–	54.0	53.7	48.5	46.8	45.3	46.5	45.5	43.7	43.5
–	–	–	–	–	72.3	70.2	66.4	63.9	63.5	65.0	65.2	62.7	63.7
84.5	85.7	85.3	82.7	80.6	77.7	76.8	74.4	73.0	73.7	72.5	74.8	74.3	73.8

"ecstasy," has shown substantial increases, reaching 5.6 percent annual prevalence among seniors in 1999. The corresponding figures for eighth and tenth graders are 1.7 percent and 4.4 percent.

Alcohol and Tobacco. The history of the use of the major licit drugs—alcohol and tobacco—is rather different than that of the use of most illicit drugs. One significant difference was the extent of the use of alcohol and tobacco. The daily use of cigarettes was far greater than the daily use of any other substance. In 1999, more than one in five (23%) high school seniors had smoked one or more cigarettes per day in the past thirty days. Even among eighth graders, one in twelve was a daily cigarette smoker (8%).

About one in thirty (3.4%) seniors had drunk alcohol daily or almost daily. All other drugs were used on a daily basis by 0.3 percent or less of seniors. Although the daily use of alcohol was relatively infrequent among high school seniors, episodic or periodic drinking was more frequent. In 1999, nearly one third (31%) of seniors reported they had had five or more drinks in a row at least once during the past two weeks. (Drinking five or more drinks "in a row" is likely enough to render the average teenager intoxicated.) This behavior showed some declines in the late 1980s and early 1990s. From 1975 through 1988, the figure for such drinking had been between 35 percent and 41 percent, or consistently more than one in three high school seniors. Between 1988 and 1991, it declined to 30 percent, which represented an encouraging downward trend, although the absolute level remained impressively high; the trend in the 1990s

TABLE 2
Trends in Prevalence of Daily Use of Marijuana, Alcohol, and Cigarettes among Eighth, Tenth, and Twelfth

	(Percent who used daily										
	1975	*1976*	*1977*	*1978*	*1979*	*1980*	*1981*	*1982*	*1983*	*1984*	*1985*
Marijuana/Hashish											
Any daily use											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	6.0	8.2	9.1	10.7	10.3	9.1	7.0	6.3	5.5	5.0	4.9
Alcohol[a]											
Any daily use											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	5.7	5.6	6.1	5.7	6.9	6.0	6.0	5.7	5.5	4.8	5.0
5+ drinks in a row in last 2 weeks											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	36.8	37.1	39.4	40.3	41.2	41.2	41.4	40.5	40.8	38.7	36.7
Cigarettes											
Any daily use											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	26.9	28.8	28.8	27.5	25.4	21.3	20.3	21.1	21.2	18.7	19.5
1/2 pack+/day											
8th Grade	–	–	–	–	–	–	–	–	–	–	–
10th Grade	–	–	–	–	–	–	–	–	–	–	–
12th Grade	17.9	19.2	19.4	18.8	16.5	14.3	13.5	14.2	13.8	12.3	12.5

NOTE: See Johnston, O'Malley, & Bachman (2000) for more specific details about measures.
[a]In 1993, the question about alcohol use was revised slightly.

was not so encouraging, with the level in 1999 slightly higher, at 31 percent.

The trends in the 1990s for eighth and tenth graders are also not encouraging: 1999 levels of heavy drinking are slightly higher than they were in 1991. For example, 23 percent of 1991 tenth graders reported having had five or more drinks in a row in the past two weeks, compared to 26 percent of 1999 tenth graders.

Among seniors, daily use of cigarettes peaked in 1977, when 29 percent of high school seniors smoked daily. By 1992, this had declined to 17 percent, but most of the decline had occurred by 1981, when the figure stood at 20 percent. Between 1992 and 1999, the figure increased substantially, to 23 percent. A measure of heavier smoking, the percent of high school seniors who smoked a half pack or more of cigarettes per day, showed a similar trend; it peaked in 1977 at 19 percent, declined to 14 percent by 1981, was down to 10 percent in 1992, but was back to 13 percent in 1999. Thus, although the 1980s showed some declines in cigarette smoking among young Americans, these declines were far more modest than one might have expected. Given the large increases in antismoking legislation, restrictions as to where smoking is allowed, and the general spread of antismoking attitudes, the declines were surprisingly small, and have eroded some in the 1990s.

The upward trend in cigarette use during the 1990s was strikingly present among eighth and tenth graders. Monthly use increased among both grades by about 50 percent from 1991 to 1996 (from 14 percent to 21 percent among eighth graders, and from 21 percent to 30 percent among tenth graders), before moderating slightly after that.

Graders

in last thirty days)

1986	*1987*	*1988*	*1989*	*1990*	*1991*	*1992*	*1993*	*1994*	*1995*	*1996*	*1997*	*1998*	*1999*
–	–	–	–	–	0.2	0.2	0.4	0.7	0.8	1.5	1.1	1.1	1.4
–	–	–	–	–	0.8	0.8	1.0	2.2	2.8	3.5	3.7	3.6	3.8
4.0	3.3	2.7	2.9	2.2	2.0	1.9	2.4	3.6	4.6	4.9	5.8	5.6	6.0
–	–	–	–	–	0.5	0.6	0.9	1.0	0.7	1.0	0.8	0.9	1.0
–	–	–	–	–	1.3	1.2	1.7	1.7	1.7	1.6	1.7	1.9	1.9
4.8	4.8	4.2	4.2	3.7	3.6	3.4	3.0	2.9	3.5	3.7	3.9	3.9	3.4
–	–	–	–	–	12.9	13.4	13.5	14.5	14.5	15.6	14.5	13.7	15.2
–	–	–	–	–	22.9	21.1	23.0	23.6	24.0	24.8	25.1	24.3	25.6
36.8	37.5	34.7	33.0	32.2	29.8	27.9	27.5	28.2	29.8	30.2	31.3	31.5	30.8
–	–	–	–	–	7.2	7.0	8.3	8.8	9.3	10.4	9.0	8.8	8.1
–	–	–	–	–	12.6	12.3	14.2	14.6	16.3	18.3	18.0	15.8	15.9
18.7	18.7	18.1	18.9	19.1	18.5	17.2	19.0	19.4	21.6	22.2	24.6	22.4	23.1
–	–	–	–	–	3.1	2.9	3.5	3.6	3.4	4.3	3.5	3.6	3.3
–	–	–	–	–	6.5	6.0	7.0	7.6	8.3	9.4	8.6	7.9	7.6
11.4	11.4	10.6	11.2	11.3	10.7	10.0	10.9	11.2	12.4	13.0	14.3	12.6	13.2

DEMOGRAPHIC DIFFERENCES

Drug use among several demographic groups is monitored in the surveys, including by gender, four-year college plans, parental education (an indicator of socioeconomic status), geographical region, population density, and racial or ethnic identification.

Gender. By senior year, male adolescents are more likely than female adolescents to use most illicit drugs, and the differences tend to be largest at the higher frequency levels. In 1999, for example, 8 percent of male high school seniors reported that they were using marijuana daily, versus 4 percent of female seniors. For many specific substances, there is little gender difference in use among eighth and tenth graders. Indeed, female eighth graders have slightly higher rates of annual use than males for inhalants, amphetamines, and tranquilizers.

There are large gender differences in the prevalence of occasions of heavy drinking among high school seniors (38 percent for male adolescents versus 24 percent for female adolescents in 1999); thus, as with heavy use of illicit drugs, heavy use of alcohol is more likely among male adolescents than it is among female adolescents. This gender difference is somewhat smaller than the one obtained in 1975, when the figures were 49 percent and 26 percent, respectively. The narrowing of the difference is primarily attributable to the greater decrease in heavy drinking among male adolescents than among female adolescents. The current differences are similar, though smaller, among the younger students. Among 1999 eighth graders, 16 percent of boys reported heaving drinking compared

to 14 percent of girls; the corresponding figures for tenth graders were 30 percent and 22 percent.

In general, there is not much difference between male and female students in cigarette use. As with most drugs, the greater difference is seen among older, heavy smokers, but even so the difference is rather small: In 1999, 15 percent of male seniors reported smoking at the rate of a half pack or more per day, versus 12 percent of female seniors.

College-Bound versus Non-College-Bound. Non-college-bound students are more likely than college-bound students to use any of the licit or illicit drugs. More frequent use of the drug tends to show greater differences. For example, 6 percent of non-college-bound eighth graders report smoking marijuana daily, compared to 1 percent of the college-bound; corresponding figures for tenth and twelfth graders are 10 percent versus 3 percent, and 9 percent versus 5 percent, respectively. Striking differences show up between college-bound and non-college-bound students in cigarette smoking rates. For example, smoking a half pack or more a day is more than six times more prevalent among the non-college-bound 1999 eighth graders than among the college-bound (13% versus 2%). Among seniors, half a pack or more smoking is more than twice as prevalent among the college-bound, 23 percent versus 10 percent. (The greater ratio in the younger students is likely due to the presence of the eventual dropouts in the eighth and tenth grades, because dropouts tend to have higher rates of smoking than nondropouts.) Non-college-bound students are also more likely than their college-bound counterparts to report having had five or more drinks in a row in the past two weeks (39 percent versus 24 percent among tenth graders, for example).

Parental Education. Among high school seniors there is (perhaps surprisingly) rather little association between parental education and use of illicit drugs. There is somewhat more of an association among the lower grades, particularly among eighth graders, with the lowest level or lower two levels having somewhat higher use rates than the others.

Geographical Region. Overall, use of illicit drugs does not vary dramatically by region. As of 1999, the annual use of any illicit drug was (slightly) lowest in the South among tenth and twelfth graders, but in the Northeast among eighth graders.

Both the South and the West tend to exhibit slightly lower rates of alcohol use than the Northeast and the North Central states. For example, in 1999 the prevalence of heavy-drinking occasions (that is, five or more drinks in a row on at least one occasion in the past two weeks) among the seniors was 34 percent and 32 percent in the Northeast and North Central states, respectively, compared with 30 percent and 29 percent in the South and the West. Cigarette smoking tends to be lowest in the West; for example, among 1999 seniors, smoking daily was 23 percent in the Northeast, 26 percent in the North Central, 24 percent in the South, and 17 percent in the West.

Population Density. As of 1999, the differences in high school seniors' use of illicit drugs by population density are quite small. This lack of large differences reflects the fact that illicit-drug use has spread widely throughout the nation. One substance that has shown some significant difference by population density over time is the use of cocaine. The substantial increase in cocaine use in the late 1970s, and the continuing high levels of use until the mid-1980s, was primarily an urban phenomenon. The annual prevalence rates for cocaine were nearly twice as high among high school seniors in the large standard metropolitan statistical areas as they were for seniors in the more sparsely populated areas. Cigarette use varies somewhat by population density. Among eighth graders, daily use in 1999 was at 13 percent in non-metropolitan areas, compared to 5 percent in the largest metropolitan areas, and 7 percent in other metropolitan areas.

Racial or Ethnic Identification. It is difficult to make definitive statements about even the larger minority groups such as African Americans and Hispanics, because of the relatively small numbers who participate in the surveys; it is virtually impossible to make definitive statements about other minority groups. Even Hispanics, who constitute a large segment of the population in many areas, often cannot be accurately represented because there are many important subgroups among the several Hispanic groups (e.g., Mexican, Puerto Rican, Cuban, and Latin American, among others). Nevertheless, certain findings appear to be reliable.

Among high school seniors, African-American students report less use of virtually all substances than do white or Hispanic students. Generally, African-American students in eighth and tenth grades

also report less use of most substances, although marijuana is an exception in the eighth grade, where white students report less use.

By senior year, Hispanic students report higher rates of cocaine and crack cocaine than white or African American students. These differences are stronger among eighth and tenth graders. And, particularly among eighth graders, Hispanic students tend to show the highest rates of use for some substances, including marijuana, tranquilizers, and cigarettes. In other words, in eighth grade, before most dropping out of school occurs, Hispanic students are relatively high in use of substances, while white students tend to have higher rates by twelfth grade. Very likely, the higher rates of dropping out of school observed among Hispanic adolescents (U.S. Dept. of Education, 1992) account for the shift in differences.

Some of these differences could be due to differential reporting biases, but J. M. Wallace and J. G. Bachman (1993) argue that this is unlikely to be an important part of the explanation.

SUMMARY

Between 1975 and 1992, appreciable declines were found in the use of a number of illicit drugs among high school seniors, but not in all drugs. LSD and inhalants were the notable exceptions. Moreover, some relatively slight declines were seen in alcohol use and even smaller declines in cigarette use. This picture of general improvement abruptly changed, with substantial increases seen from 1992 to 1997. The increases were evident not only among seniors, but also among eighth and tenth graders as well, with proportional changes being greater among the younger students. The situation moderated slightly, or changed rather little between 1997 and 1999, at which time drug use remained at high levels among American youth. Some items of interest are:

- As of 1999, about 55 percent of young Americans had tried an illicit drug by the time they had neared the end of their last year of high school; this proportion included about 29 percent who had tried some illicit drug other than marijuana. About 28 percent of young Americans had tried an illicit drug before they finished eighth grade, including 16 percent who had tried some illicit drug other than marijuana.
- Marijuana had been tried by 50 percent of seniors, 41 percent of tenth graders, and 22 percent of eighth graders.
- One in ten (10%) twelfth graders had tried cocaine, and about one in every twenty-two (4.6%) had tried crack cocaine.
- A significant number of high school seniors in 1999 smoked marijuana daily (6%).
- Almost a third (31%) of high school seniors in 1999 had had five or more drinks in a row at least once in the prior two weeks.
- More than a third (35%) of seniors had smoked cigarettes in the month prior to the survey, and 23 percent smoked daily. More than a sixth (18%) of eighth graders had smoked cigarettes in the month prior to the survey, and 8 percent already smoked daily.

In addition to providing basic epidemiologic information on prevalences, trends, and demographic differences, the Monitoring the Future study also contribute information on the reasons for the trends and differences. The study's demonstration that attitudes and beliefs affect drug-use trends (especially in the case of marijuana and cocaine) is particularly important (Bachman, Johnston, & O'Malley, 1998; Johnston, O'Malley, & Bachman, 2000). By virtue of its cohort-sequential design, the study has been able to distinguish among the several possible types of competing changes associated with trends in use-specifically, age, period, and cohort (or birth group) effects (O'Malley, Bachman, & Johnston, 1988). In addition, the study has been able to provide important data with which researchers could evaluate the effects of changes in the laws dealing with marijuana (Johnston, O'Malley, & Bachman, 1981) and alcohol (O'Malley & Wagenaar, 1991). All of these contributions have been vital in the continuing debates about policy regarding the use of licit and illicit drugs.

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HISPANICS AND DRUG USE, IN THE UNITED STATES Hispanics in the United States are a large, growing, diverse group. More precisely, 1990 U.S. Census figures put the total at 22 million—of these, 63 percent are Mexican in origin, 11 percent Puerto Rican in origin, and 5 percent Cuban in origin. These three groups are the largest, yet another 14 percent of Hispanics are from the various Central and South American countries; still another 8 percent are classified as "other Hispanic" by the U.S. Bureau of the Census. In this essay the terms *Hispanic* and *Latino* are used interchangeably. *Hispanic* is commonly used in official statistics, and *Latino* is more widely used within the population itself.

The rapid growth of the Latino population within the United States also is noteworthy. It grew by 53 percent between 1980 and 1990. A high birth rate and continuous new immigration fuels this growth.

On average, Hispanics are younger than other minorities and other American population groups. When youthfulness is combined with POVERTY or discriminatory practices, the result sometimes is a disproportionate degree of conflict with law enforcement, especially in connection with drug abuse and drug dealing. The media coverage of these conflicts may lead many into a prejudicial belief about Latinos and drug use.

Although there are many notable exceptions, most Hispanics live in cities in the United States and, lacking other options, they are steadily crowding into the poorest areas of New York, Los Angeles, Chicago, and other large cities. In 1990, 25 percent of Latinos in the United States lived in poverty compared with 31 percent of black families and 13 percent of all other Americans. Poor education, difficulty with the English language, and urban concentration can compound this impoverishment—as it has for the other immigrant minorities in the United States—thereby contributing to the complexity of modern urban problems that they must face daily.

All segments of this highly diverse group are changing rapidly. Documented and undocumented new immigration combined adds about 500,000 arrivals each year, and this flow is increasing. Many of the newcomers crowd into old barrios, and this reduces the quality of life for older residents. Great pressure is therefore exerted on local educational services, health resources, job sources, and job-training services—a pressure that is compounded by problems of acculturation. Many Mexican-American communities predate the Mexican-American War of the 1840s, but other Latino communities have become established in significant numbers only since World War II. Puerto Ricans, for example, settled mostly in the large cities of the Rust Belt in the late 1940s and early 1950s, forming a particularly large concentration in New York City. Like

Mexican Americans (Chicanos), they have been sharply affected by recent shifts in the American economy that relegate poorly educated workers to poorly paid service jobs. Central and South Americans are found in diverse locations, with concentrations in New York, Houston, and Los Angeles, tending to work at the bottom of the labor market. Cubans, who are concentrated primarily in Miami, have been helped both by a vigorous enclave economy (with Cubans owning many of the enterprises and hiring fellow Cubans) and by Miami's emergence as a center for Latin American trade.

HISPANICS AND ILLICIT DRUGS

Latinos often are typecast as drug users (see Helmer, 1975). Such stereotypes persist partly because there is little research information. National statistics about Hispanics mask important variations within the population, not only in ethnicity but in class and culture. Drug problems of the community are treated principally as criminal phenomena, and indeed, in many states a disproportionate number of Latinos are imprisoned for drug-related offenses. The context for drug use is little studied.

What then is really known about drug use by Hispanics? Specifically, 1991 figures from the annual survey of the National Institute on Drug Abuse (NIDA) show that Hispanics are generally less likely to use drugs in their lifetime than either blacks or the white-majority population. However, Hispanics are most likely to have used COCAINE, and next most likely (after blacks) to have used CRACK cocaine. National surveys do not report on HEROIN, an illicit drug that has posed major problems for Latinos, particularly in New York and the Southwest. Heroin use has been studied in several southwestern communities, in particular in the context of peer group and FAMILY in Los Angeles barrios.

The aggregate figures also conceal significant subgroup differences. Puerto Ricans are especially likely to use cocaine, for example, and Cubans are notably less likely to use any drug. (However, clinical data indicate that Cuban drug use is actually higher than survey data show.)

The aggregate figures conceal geographic differences as well. Studies of persons arrested for crimes, for example, show that more than two-thirds of Hispanic arrestees in Chicago, New York, Philadelphia, and San Diego were using drugs but that proportions were far lower in most other cities (U.S. Department of Justice, 1991). Finally, drug-use patterns may change rapidly, even in a high-risk population: for example, 68 percent of San Antonio's Hispanic arrestees were using some drug in 1988, but by 1991 only 47 percent were, according to U.S. Department of Justice figures (1990). Glick (1990) has analyzed the shifting drug-use patterns in Chicago's Puerto Rican community.

Differences in drug use by males and females are sharper for Hispanics than for other ethnic or cultural groups. Mexican American and Puerto Rican boys and girls are socialized very differently to alcohol and drug use—that is, there is more parental and community disapproval for girls and more permissiveness for boys. Yet research on drug use among Hispanic women is scarce. Among the available research, of particular interest is the finding that sedatives and prescription drugs are used differently by women than they are by men (Gonzalez & Page, 1991). There is also research showing that most female heroin addicts usually begin to use heroin with a male friend, spouse, or common-law partner, thus suggesting that the use depends on a relationship. Hispanic women appear to be greatly influenced by traditional ideas about the role of women, even under the pressures of urbanization, acculturation, and poverty (Moore, 1990).

As to adolescents, the most susceptible group, there is little information about how adolescent Hispanic groups differ from other adolescent groups in drug use. National surveys of high school seniors discover only small differences, but the surveys omit dropouts, who are often the adolescents most at risk, and Hispanic adolescents have very high dropout rates. Most studies confirm that the same risk factors that are important for other youth are important for Hispanics: above all, a disruptive family environment; availability of drugs; peer influences; and patterns of unconventional behavior (such as low school achievement, rebelliousness, early sexual activity). These influences (plus the degree of acculturation and individual judgments of the adolescent) seem to be related, in a general way, with beginning drug use and a steady use of drugs (Booth, Castro, & Anglin, 1990). One notable fact is that gender differences are less significant for adolescent Hispanics than they are for adult Hispanics (Gilbert, 1985).

A special factor that affects Latinos is the overriding importance in the culture of the family. This influence has both positive and negative effects. The extended family among Puerto Ricans in New York may limit drug use by protecting and controlling youngsters in both single- and two-parent households (Fitzpatrick, 1990). In Cuban families, by contrast, illicit drug use may occur when the family structure is severely disrupted, often by the trauma of refugee migration, and researchers argue that the very cohesiveness of the Cuban family may be associated with parental overprotectiveness and adolescent rebellion, sometimes accompanied by drug use as a symptom (Rio et al., 1990).

Recent research suggests that Hispanic clients achieve only mixed success in treatment, but that finding needs qualification, because of the limitations of available treatment programs. Because of poverty and residence in blighted areas, a disproportionate number of Latino heroin users, for example, are enrolled in programs that simply administer blocking drugs (e.g., methadone), with virtually no other treatment. Urban drug treatment programs generally face chronic shortages of money and personnel. When drug abusers do get access to broader treatment, failure can often be blamed upon the absence of culturally sensitive therapies (Rio et al., 1990). Fitzpatrick (1990) has suggested that Puerto Ricans in New York City show an "extraordinary" ability to cope with a community saturated with drugs and that efforts should be made to build on this ability.

HISPANICS AND ALCOHOL

Among Hispanic and many other groups, ALCOHOL use has been easier to study than the use of illicit drugs; many of its patterns are similar to and may shed light on drug use. As they do with drugs, Hispanics use less alcohol over their lifetimes than do "Anglos" (i.e., non-Hispanic white U.S. inhabitants in general, not just those of English ancestry), and their usage is only very slightly more than that of blacks. Again as with drugs, there are sharp gender differences in alcohol use, which are especially noteworthy among immigrants. Among Mexican Americans, the gap between male and female drinking narrows but never disappears in succeeding generations, and much recent research focuses on this acculturation effect, so critical in a large new immigrant population (Canino, 1994). Among younger women, the narrowing gap seems to reflect both acculturation and upward social mobility. Even within one city, Mexican-American drinking habits vary greatly by class (Trotter, 1985). But Gilbert found that Mexican Americans in California also speak of family, financial, and job problems as factors in abusive drinking; they tend to recognize alcoholism not as a medical problem but as a failure of will (Gilbert, 1985). Certainly there is no one set of beliefs, behaviors, and norms associated with Latinos and drinking. Lifestyle diversity within Latino subgroups suggests the need for a corresponding diversity of treatment approaches. The failure of such standard treatments as ALCOHOLICS ANONYMOUS among Hispanics in certain areas should be noted.

Finally, as noted before in regard to drugs, there are important differences in drinking behavior between subgroups of Hispanics. Mainland-dwelling Puerto Ricans' use of both alcohol and drugs is comparatively high wherever studied (Gordon, 1985). Pentecostal church groups have had notable success in influencing the drinking behavior of some Puerto Ricans, although some clinicians have expressed the view that Puerto Ricans are reluctant to use treatment services. Cuban drinking patterns are generally moderate: Cultural values of self-control forbid discernible drunkenness for both men and women. With increasing acculturation, there is gradually increasing alcohol usage but reduced reliance on minor TRANQUILIZERS by Cuban women. All the (scanty) information available on the subject stresses the importance of individual ethnic experience.

(SEE ALSO: *Ethnic Issues and Cultural Relevance in Treatment: Ethnicity and Drugs; Families and Drug Use; High School Senior Survey; Inhalants: Extent of Use and Complications*)

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HISTORY OF TREATMENT *See* Treatment, History of, in the United States

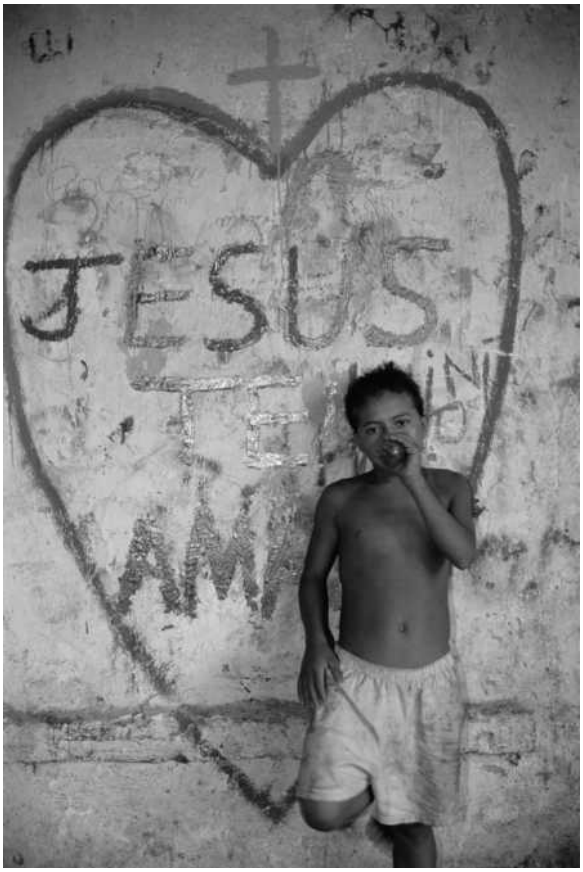
HIV *See* Assessment of Substance Abuse: HIV Risk Behaviors Survey; Injecting Drug Users and HIV; Needle and Syringe Exchanges and HIV/AIDS; Substance Abuse and AIDS

HOMELESSNESS, ALCOHOL, AND OTHER DRUGS, HISTORY OF The word *homeless* has a long and complex use. In its most literal meaning of houseless, it has been employed since the mid-1800s to describe those who have slept outdoors or in various makeshifts, or who resided in temporary accommodations like the police-station lodgings of earlier generations or the emergency shelters of the present day. Another early meaning of the word draws upon the absence of a sense of belonging to a place and with the people who live there. This usage was handed down from the largely rural and small-town society of the nineteenth century, in which the coincidence of family and place provided the basis for community and social order, nurturing traditions of mutual aid and the control of troublesome behavior. To be homeless was to be "unattached," outside this web of support and control; it was to be without critical resources and, equally important, beyond constructive restraint. Many of the young men and women who moved from farm to city, or those who emigrated during the nineteenth and early twentieth centuries, were unattached in this respect. Organizations like the YWCA, and YMCA, and various ethnic mutual-aid societies were invented both to help and superintend them by creating surrogate social ties.

HISTORY

By the 1840s, it was common for Americans to link homelessness with habitual drunkenness. In the popular view, habitual drunkards, usually men, drank up their wages and impoverished their families; they lost their jobs and their houses, and drove off their wives and children by cruel treatment. They became outcasts and drifters and their wives entered poorhouses while their children became inmates of orphanages. By the 1890s, the same logic served to explain the downward, isolated spiral of opiate and cocaine "friends" (as they were called) and the unhappy circumstances of their families.

Until the early years of the Great Depression (which began in 1929), habitual drunkenness, in



A homeless young boy inhales glue in front of graffiti that reads "Jesus loves me." (© Bill Gentile/CORBIS)

particular, often was cited as a principal cause of homelessness. Even so, after the financial collapse of 1893 and an ensuing five-year depression of unprecedented severity, most thoughtful observers did not understand heavy drinking or habitual drug use to cause homelessness in any *direct* manner. Although scholarly studies during the first decades of the 1900s were crude by today's technical standards, their explanations of homelessness were not simple-minded. In fact, they foreshadow today's explanations.

Perhaps most important, pre-Depression students of homelessness noted that the ranks of the dispossessed grew and diminished in close relation to economic conditions. They understood that the profound depressions that haunted the economy long before 1929 caused large numbers of people to lose their grip on security. They noted as well that certain occupations were especially affected by seasonal fluctuations in the demand for labor and by technological change—by the 1920s, agricultural workers, cigar makers, printers, and others had high rates of "structural" unemployment. That is, their jobs had been lost permanently to changes in methods of production and distribution.

These scholars also understood the importance of decisions that employers made about hiring and firing. Workers without families to support and those regarded as the least productive were let go first when the economy soured. Usually, these were single young women assumed able to return to their natal families, married women presumed to be working for "pin money" (people who are today known as secondary wage earners), older men, and in particular, single men known to drink heavily. Minority racial and ethnic status also marked people for layoff. Conversely, in times of high demand for labor, employers relaxed their standards for hiring and job performance. In boom times all but the most seriously disabled, and the most erratic and disruptive heavy drinkers and drug users, could find some kind of work. The ranks of the homeless thus thinned considerably.

Pre-Depression observers also emphasized the impact of working conditions, disability, and the absence of income supports on the creation of homelessness. In an era of dangerous work and widespread chronic disease (especially tuberculosis), large numbers of men, in particular, became substantially disabled, often at a young age. In an era before significant public disability benefits or much in the way of welfare or effective medical treatment, they rapidly became abjectly poor, reduced to begging, soup kitchens, and bedding down in mission shelters or the cheapest, most verminous lodginghouses ("flophouses," as they came to be called).

Some of these men were heavy drinkers, and some were habitual drug users, but it was commonly observed that such problems often developed in the context of POVERTY and rootlessness. The miseries and long stretches of boredom endemic to poverty were understood to promote frequent intoxication—even during the Prohibition years (1920–1933), when illicit ALCOHOL could be had by arrangement, as could illicit drugs. Certain "hobo" occupations that virtually demanded rootlessness, and which brought together large groups of men without families, were regarded as especially corrupting and debilitating. Railroad

gangers, cowboys, farmworkers, lumberjacks, and sailors, among others, pursued risky occupations and lived in ways that provided both motive and opportunity for dissipation. During the Depression it was widely feared that tens of thousands of homeless young people in the United States would be maimed hopping freights and would learn bad habits on the road that would transform them into lifelong tramps.

Finally, and related to their understanding of homelessness as an insalubrious and demoralizing experience, early observers paid a great deal of attention to the milieu of homelessness, which is to say, the urban areas where homeless people congregated and the constellation of institutions with which they were involved. Commonly called "hobohemias" before the Depression and "skid rows" thereafter, such areas were characterized by a particular way of life and a peculiar set of economic and social resources. They were honeycombed with cheap restaurants, residential hotels and lodginghouses, private and eventually public welfare agencies, and formal and informal labor exchanges that offered casual ("day") work. Skid row (and the segregated satellites that developed in minority communities) was also a world dominated by single men. Such areas were saturated with saloons (later bars) and sex workers. Some were the sites of a vigorous drug trade.

By the 1940s, winnowed by wartime labor demand, skid row was both repository and refuge, mainly for impoverished single men disabled by age, injury, and/or chronic illness. They survived on private charity, meager public welfare allowances, modest pensions, and undemanding work. Note, however, that they were housed. In the most literal sense, skid-row denizens were not homeless, and from the 1940s through the 1970s they were more often described as "unattached" or "disaffiliated." They were homeless in the broader, social sense discussed above. Further, and contrary to the enduring stereotype, the residents of skid row were not usually heavy drinkers or habitual drug users. Although perhaps one-third could be so described, and while public intoxication was common and visible, heavy drinking or drug taking was, as today, the exception not the rule.

With the sustained prosperity of the period between 1941 and 1973, and the simultaneous elaboration of the American welfare stale, many observers believed that skid row would wither away. The older men would die off, or—helped by federal Old Age Security, and later by Medicare, state and federal disability benefits, and subsidized housing—would move to better neighborhoods. Or they would remain on a skid row that would be uplifted and transformed by urban renewal projects and effective rehabilitation programs for heavy drinkers and drug users.

In a limited sense, these optimists were correct. The expansion of the welfare state dramatically improved the economic circumstances of the elderly, and they are greatly underrepresented among today's homeless. Aided by federal funds, some cities bulldozed their skid-row areas, thus causing their bricks and mortar, at least, to disappear. But homelessness did not disappear; instead, it underwent an astonishing and tragic transformation. If literal houselessness is used as the definition and measure of the problem, only the Depression produced the prodigious dispossession we see today.

As opposed to the domiciled isolation of skid row, something like today's houseless poverty was beginning to be reported in news magazines and the occasional scholarly publication as early as 1973. But it was not until the early 1980s that a new generation of younger homeless people achieved widespread notice. At first, most observers were struck by the apparently very high rates of mental illness, heavy drinking, and drug use among those new homeless people. Explanations of the problem tended to point toward nationwide changes in policies that governed commitment to and retention in mental hospitals and incarceration for public drunkenness and minor drug offenses. During the 1960s and 1970s many states "deinstitutionalized" both mentally ill people and "alcoholics" and "addicts." That is, state hospital patients were discharged in wholesale fashion, and new commitment laws made initial involuntary commitments difficult; they severely limited the duration of involuntary treatment. Many states also "decriminalized" public drunkenness, referring public inebriates to places where they could sober up rather than housing them in jail for thirty days to six months. Similarly, many minor drug offenders were diverted from jails. During the early 1980s many observers, notably those within the Reagan administration, characterized the resurgence of homelessness as a problem related to mental disorder, excessive drinking, habitual drug use,

and the new policies that kept people with such problems from their customary lodgings in state hospitals and county jails. Homelessness was described mainly as a problem in the rehabilitation and control of troubled and troublesome people who were not only houseless but barred from their traditional institutional shelters and estranged from family and friends who might take them in.

CURRENT VIEWS

Although not discounting this view entirely, most scholars now find it too simple and not supported by the evidence. Although some popular treatments of the subject continue to claim that perhaps 85 percent of homeless people are substance abusers and/or mentally ill, such huge figures are drawn from old studies and that were seriously flawed by two related methodological problems. The first requires little explanation: These studies for their estimates relied on *lifetime* rather than *current* measures of problems. In any group not in treatment or recently discharged, a lifetime measure (a determination of whetehr a person has ever had a sever mental illness or substance-use disorder) will always produce much higher prevalence rates than a measure of current disorder (customarily defined as present within the previous six months or one year).

The second problem is a matter of how homeless respondents were sampled for these studies and concerns the distinction epidemiologists make between "point prevalence" and period prevalence." The first term refers to counts of some condition conducted at a single moment in time (a snapshot), whereas the latter refers to counts taken over some expanse of time (a motion picture). Longitudinal ("period") counts of homeless people will produce much higher numbers than cross-sectional ("point-in-time") enumerations, for many more people are homeless during a year than on a given night. To the extent that people without problems of substance abuse and mental illness move out of homelessness more rapidly than those who suffer from then, they will be overrepresented in snapshot studies because they are more likely to be counted. Recent longitudinal studies demonstrate conclusively that a fairly small group of people with very high rates of disorder (usually single men under forty years old) account for a very large percentage of "shelter nights" in most cities. Since most studies of homeless populations conducted in the 1980s sampled from shelters on a cross-sectional basis, their estimates of substance abuse and mental illness were correspondingly inflated.

With these caveats in mind, it is probably fair to say that among all adults homeless during the previous year, something like half had a substance-use disorder or a major mental illness, alone or in combination. These rates are substantially higher among single men and significantly lower among adults who are homeless in family groups, most often single women.

Even so, sound prevalence estimates do not explain the casual relationship between homelessness and substance abuse and mental illness. Clearly, most people with such problems never become homeless. To explain why some do, current scholarship has returned—often unwittingly—to themes first sounded a century ago: the relationship of homelessness to changes in the economy and the nature and supply of housing; to the availability (or "coverage") and sufficiency of income supports and medical care; and to the tolerance and support capacity of kin. Heavy drinking, habitual drug use, and mental illness are considered in this larger context. Such problems are understood to be among many "risk factors" which make it more likely that some people will become homeless repeatedly or remain so for a long time. Moreover, current scholars are concerned increasingly with how such experience wears people down, introduces or rekindles bad habits or poor health, and makes "exits" from homelessness less likely or short-lived.

Briefly and simply, current scholarship suggests the following relationship between homelessness and heavy drinking and habitual drug use.

The problem of poverty has worsened considerably since the mid-1970s. Changes in the economy have added high-skill, well-paid technical jobs and low-skill, poorly paid service positions, but these changes have simultaneously produced job losses among semiskilled but highly paid workers, primarily in manufacturing. This process of "deindustrialization"—the historic passage from a manufacturing to a service economy—has been especially hard on those younger members of the huge baby-boom birth cohort (boomers are those born between 1946 and 1964), especially Hispanics and African Americans, who have entered a glutted labor market without the advantage of pro-

longed higher education or advanced technical training.

At the same time, the 1980s brought startling inflation in rental housing costs and a steep decline in the inflation-adjusted value of federal and state welfare benefits and unemployment insurance. In consequence, poor people had an increasingly difficult time forming independent households and poor families became increasingly hard put to support dependent adult members. On top of this and simultaneously, the stock of America's most rudimentary housing, the old hotels and lodginghouses of skid row and similar areas, was decimated by urban renewal.

The baby boom's maturation was crucial in another way. Although there is no good evidence that the combined *rate* of persistent and severe mental disorder, heavy drinking, or habitual drug use is significantly higher among boomers, neither is there any evidence that it is substantially lower than in previous birth cohorts. However, if a roughly constant rate (similar percentage) is applied to a much larger population, the resulting prevalence of a problem is of much greater magnitude—the numbers are much larger. Therefore, as huge numbers of boomers reached the age of greatest risk for the development of enduring mental-health, alcohol, and drug problems (roughly eighteen to twenty-five years old), the cohort generated an unprecedented number of such casualties. This situation developed just as conditions of material scarcity were becoming acute and the old policies of institutional containment were being dismantled.

Ironically, the unprecedented, sustained economic growth of the 1990s aggravated the problem of homelessness. As the decade wore on, shelter counts rose all over the country. In some part, this was because the general prosperity of the 1990s had little effect in the lowest reaches of the income distribution from which homeless people come, and cutbacks in federal, state, and local welfare eligibility compounded the problem. Further, rapid ecomonic expansion tends to have a significant inflationary effect on rents. Indeed, for the poorest 20 percent of American households, rents increased faster than incomes between 1995 and 1997. Moreover, the number of units renting renting for $300 per month (in inflation-adjusted dollars) decreased by 13 percent from 1996 to 1998, resulting in the loss of almost one million such units nationwide. At the same time, the number of households assisted by subsidies from the Department of Housing and Urban Development dropped by 65,000 between 1994 and 1998. In sum, the crisis in affordable housing became worse during the great boom.

CONCLUSION

Poor people have been badly squeezed since the early 1970s. As a consequence, perhaps 3 percent of all American adults, about 5.5 million people, experienced at least one spell of homelessness between the beginning of 1985 and the end of 1990. Some, however, experience frequent and prolonged episodes of homelessness, and it is among these people that rates of heavy drinking and habitual drug use are very high. It is not simply the case, however, that their drinking and drugging have caused their homelessness. The health problems and troublesome behavior often associated with such habits may have played an important role in job loss, familial estrangement, or displacement from housing—but this is not a new phenomenon, as we have seen.

Now, though, the absorptive mechanisms of earlier generations have gone awry. Deinstitutionalization has been a factor in this breakdown, mainly because its presumed consequence of community care never has been equal to the unprecedented generational need. Nonetheless, more important factors in the creation of widespread houseless poverty among heavy drinkers and habitual drug users have been the disappearance of casual labor, the erosion of public benefits and the capacities of kinship, and the virtual destruction of the tough but viable refuge of skid-row housing. In 1970, impoverished heavy drinkers and habitual drug users could almost always find some port in the storm, often by moving from one decrepit hotel to another, frequently pooling resources to rent a room by the week. Since the 1980s, they can no longer. Thus they have become a large and highly visible proportion of those who inhabit our public places and persist in our shelters month after month.

(SEE ALSO: *Alcohol: History of Drinking; Alcohol- and Drug-Free Housing; Halfway Houses; Treatment: History of in the U.S.*)

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HONG KONG AND DRUGS *See* Opioids and Opioid Control: History of

HYDROMORPHONE Hydromorphone is a semisynthetic OPIOID analgesic (painkiller) derived from thebaine, an ALKALOID of the OPIUM poppy (PAPAVER SOMNIFERUM). It is one of the most widely used and effective analgesics for moderate to severe PAIN and is often referred to as Dilaudid, one of the brand names under which it is sold. Its potency is almost eightfold greater than is morphine's. Structurally, it is quite similar to MORPHINE but most like dihydromorphine, differing only in the replacement of the hydroxyl (–0H) group at the 6-position with a ketone (=0). Thus, it is not surprising that hydromorphone has many of the same side effects—including sedation, constipation, and depression of breathing. Chronic use will produce TOLERANCE AND PHYSICAL DEPENDENCE, much like morphine. This drug is reported to have high abuse potential, perhaps due, in part, to its very high potency.

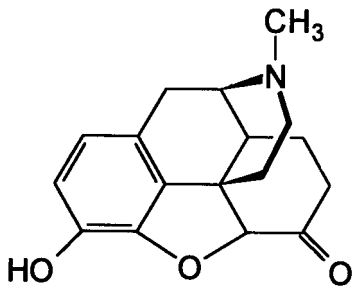

Figure 1
Hydromorphone

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HYPERACTIVITY *See* Attention Deficit Disorder; Conduct Disorder in Children

HYPNOSIS *See* Treatment Types: Hypnosis

HYPNOTICS *See* Sedative-Hypnotic

I

IATROGENIC ADDICTION The potential for ADDICTION or ABUSE influences the licit medical use of many drugs, including OPIOIDS, BENZODIAZEPINES, BARBITURATES, and others. This influence can be evaluated from two perspectives—(1) the risk that addiction or abuse will result from medical treatment of patients with no such prior history, and (2) the possibility that overconcern about this risk leads to inappropriate undertreatment of certain medical conditions. Although these issues can be discussed with reference to any of these drug classes, the opioids are most illuminating and are emphasized below.

THE RISK OF ADDICTION OR ABUSE

Like any other potential adverse outcome of drug therapy, the prevalence of iatrogenic addiction (drug addiction or abuse during medical treatment) must be determined so that the risk can be assessed by both the practitioner and the patient. An accurate understanding of prevalence, in turn, requires the application of clinically relevant definitions of these phenomena. Unfortunately, there has been little effort to define the addiction syndrome as it occurs in patients, and there is abundant evidence that clinicians commonly use definitions that are inappropriate.

Definition of Addiction in Medical Patients. Accepted definitions of addiction and abuse (Jaffe, 1985; Rinaldi et al., 1988) have been derived from experience with addict populations. These definitions emphasize that addiction is a psychological and behavioral syndrome characterized by psychological dependence on the drug and aberrant drug-related behaviors. There is loss of control over drug use and evidence of compulsive use. Use of the drug continues, and often escalates, despite overt harm to the user or others. The definitions for abuse project a similar sense and stress the persistence of harmful drug use (Rinaldi et al., 1988) or its deviation from accepted societal or cultural norms (Jaffe, 1985).

The validity of these definitions has not been evaluated in medical populations. Although specific behaviors must be used to establish the diagnoses of addiction or abuse, there have been no studies that assess the predictive value of those behaviors that commonly raise concern in clinicians (Table 1). Some behaviors, such as dose escalation, that strongly support a diagnosis of addiction in an individual who does not have an appropriate medical condition or obtains the drug from nonmedical sources may be more difficult to interpret in patients who acquire the drug from a physician to manage an appropriate problem. Some patients with unrelieved cancer pain, for example, have been said to demonstrate pseudo-addiction—behaviors that suggest addiction but disappear as soon as analgesia (pain relief) improves (Weissman & Haddox, 1989).

In the absence of adequate studies of addiction and abuse in medical patients, the evaluation of drug use in the clinical setting is based on observed

TABLE 1
Behaviors that Raise the Suspicion of Addition or Abuse of Prescription Drugs

Probably More Predictive	*Probably Less Predictive*
Selling prescription drugs	Aggressive complaining about the need for higher doses
Prescription forgery	Drug hoarding during periods of reduced symptoms
Stealing or "borrowing" drug from another patient	Requesting specific drugs
Injecting oral formulations	Acquisition of similar drugs from other medical sources
Obtaining prescription drugs from nonmedical sources	Unsanctioned dose escalation once or twice
Concurrent abuse of related illicit drugs	Unapproved use of the drug to treat another symptom
Multiple dose escalations despite warnings	Reporting psychic effects not intended by the clinician
Multiple episodes of prescription "loss"	

NOTE: There have been no studies to assess the relative predictive value of these behaviors, but separation into the two categories of "more" or "less" predictive is supported by clinical experience.

situations. Although some behaviors may provide compelling evidence (selling prescription drugs), most will require astute and often repeated assessments. Any suggestion of aberrant drug-related behavior should impel a comprehensive assessment by the clinician of all aspects related to the patient's medical disorder and treatment plan (Portenoy & Payne, 1992).

The Problem of Mislabeling. Clinicians often compound the problem of definition by mislabeling patients as addicts without the evidence to support this diagnosis. Such mislabeling increases the perceived prevalence of iatrogenic addiction and unnecessarily stigmatizes the patient.

The most common type of mislabeling confuses PHYSICAL DEPENDENCE with addiction. Physical dependence is a pharmacologic property characterized by the occurrence of an abstinence syndrome following abrupt dose reduction or administration of an ANTAGONIST. Since physical dependence is not apparent unless an abstinence syndrome occurs, and abstinence can be easily prevented, physical dependence is generally regarded as a minor problem in the clinical setting. Although it has been postulated that abstinence symptoms can become conditioning stimuli that contribute to the genesis of addiction (Wikler, 1980), it is evident that physical dependence alone does not produce addiction or abuse. Opioid addicts, for example, may or may not be physically dependent, and cancer patients, who are almost certainly physically dependent after receiving high opioid doses for prolonged periods, almost never develop the aberrant drug-related behaviors consistent with addiction or abuse (Kanner & Foley, 1981).

Studies of Addiction or Abuse in Medical Patients. Thus, the risk of iatrogenic addiction or abuse can only be determined if proper definitions are developed and applied to patient populations. Few studies have met these criteria, but those that have are reassuring, indicating a very low risk of these outcomes during medical treatment with drugs of abuse.

Surveys of opioid use are most illustrative. Although older studies of opioid addicts suggested considerable risk of iatrogenic addiction, these data have been replaced by more recent surveys of pain patients. Addiction and abuse are vanishingly rare outcomes of opioid therapy for acute and chronic cancer pain (Kanner & Foley, 1981; Chapman & Hill, 1989). Most experts have concluded that the risk of addiction during opioid treatment for cancer pain is so remote that this outcome should not even be considered in the decision to use these drugs. Similarly, the Boston Collaborative Drug Surveillance Project could document only four cases of addiction among 11,882 patients with no prior history of substance abuse who were administered an opioid during hospitalization (Porter & Jick, 1980); a national survey of burn units could not identify a single case of addiction among 10,000 patients who had no history of substance abuse and received opioids for burn pain. Finally, surveys of selected patients with chronic nonmalignant pain also suggest that aberrant drug-related behavior is distinctly uncommon among those with no such history who are administered opioids on a long-term basis (Portenoy, 1990).

Other drugs have not been evaluated as extensively as the opioids. Recent analyses of BENZODIAZEPINE use, however, conclude similarly that addiction or abuse as defined here is a rare outcome among patients with ANXIETY disorders

who are administered these drugs by physicians (Woods et al., 1988; Balter & Uhlenhuth, 1991), although many develop physical dependence.

Together, these data indicate that medical patients with no prior history of substance abuse have a very low risk of iatrogenic addiction or abuse when they are medically administered drugs with a potential for these outcomes. This conclusion is consistent with an understanding of addiction as a disorder related to the use of specific drugs, but not inherent in the pharmacology of any. Addiction is presumably determined by an interaction between the reinforcing qualities of some drugs and a constellation of individual factors, including a genetic propensity, psychosocial aspects, and the specifics of drug availability (Jaffe, 1990, 1992; Chapman & Hill, 1989). The evidence suggests that patients who do not demonstrate a proclivity to addiction or abuse by adulthood are extremely unlikely to develop these outcomes during medical treatment thereafter. Furthermore, it is probable that this small risk could be reduced further by strict adherence to guidelines that set parameters of appropriate patient behavior and follow-up assessments. Such guidelines would also facilitate the identification of those occasional patients who develop any addiction problems.

UNDERTREATMENT

Although the conclusion that iatrogenic addiction and abuse are rare, still this appears to be inconsistent with the attitudes held by many healthcare providers and patients. Fear of addiction is commonplace. Consequently, there is evidence that overconcern about addiction adversely influences prescription practices.

The negative effects on patient care produced by an inaccurate estimate of addiction liability are most clearly documented in pain management—inadequate treatment with opioid drugs results in an unnecessarily high prevalence of unrelieved acute pain, especially cancer pain. Concerns about addiction are among the salient factors that contribute to undertreatment (Portenoy, 1995).

CONCLUSION

The data extant indicate that addiction and abuse are rare outcomes during the therapeutic use of opioids and other drugs in populations with no prior history of substance abuse. The intense concern expressed by clinicians and patients alike and the impact of this concern on prescribing practice appear to be disproportionate to the actual risk. To some extent, this may relate to the difficulties encountered in evaluating addiction and abuse in medical populations, or perhaps more likely to the tendency to mislabel outcomes as addiction that do not fulfill criteria for the diagnosis. Although good clinical practice must recognize the potential for addiction and abuse, optimal therapy depends on an accurate understanding of these phenomena and the limited role they play in clinical practice.

(SEE ALSO: *Abuse Liability of Drugs: Testing in Humans; Addiction: Concepts and Definitions; Controlled Substances Act of 1970; Diagnostic and Statistical Manual [DSM]; Disease Concept of Alcoholism and Drug Abuse; Opioids and Opioid Control; Pain; Prescription Drug Abuse; Vulnerability as Cause of Substance Abuse*)

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IATROGENIC ADDICTION: NONOPIOIDS *See* Prescription Drug Abuse

IBOGAINE The roots of the shrub *Tabernanthe iboga* first aroused pharmacological interest in 1864 when a French naval surgeon brought some back from Gabon, West Africa. The root was eaten by various Gabonese tribes as part of initiation ceremonies of puberty and was said to produce intoxication, visions, and a reduced need for sleep.

An active alkaloid, ibogaine ($C_{20}H_{26}N_2O$), was isolated in 1901 from the roots, bark, and leaves of *Tabernanthe iboga*. In the early 1900s, some medical researchers in France recommended ibogaine for use in treating neurasthenia and asthenia (syndromes that would probably be diagnosed in the 1990s as depression or fatigue syndrome). Although the drug was part of a proprietary medication marketed in Europe in the late 1930s and throughout the 1940s, ibogaine attracted little medical or scientific attention until the emergence of interest in indole alkaloids that accompanied the use of reserpine in the 1950s. During the 1960s, when there was considerable research on the use of LYSERGIC ACID DIETHYLAMIDE (LSD) and other psychedelic agents (HALLUCINOGENS) in psychotherapy, ibogaine was also studied, since it appeared to produce mental effects similar in some ways to other hallucinogens. At about the time of these studies, 1967–1968, the World Health Organization and the U.S. Food and Drug Administration (FDA) classified ibogaine as a hallucinogen, along with LSD, MESCALINE, and PSILOCYBIN.

In 1962, Howard Lotsof, who was at the time addicted to heroin, ingested ibogaine in search of a different drug experience. Lotsof came out of a long psychedelic experience, during which he had not taken any heroin, and found that he had no withdrawal symptoms and did not crave drugs. At the time, he noticed that ibogaine had a similar effect on several other heroin addicts. He subsequently remained drug free, completed law school, eventually obtained a patent on the use of ibogaine for the treatment of addiction (brand name ENDABUSE), and became active in seeking funding to further develop the drug and to obtain FDA approval for its medical use in treatment of addiction.

As a Schedule I drug under the CONTROLLED SUBSTANCES ACT, ibogaine is considered to be highly subject to abuse and without any approved medical use. To be approved by the FDA, an agent must be shown to be safe and effective. Throughout the early 1990s the only reports of the efficacy of ibogaine have been anecdotal ones from individuals in Europe who were addicted to heroin, COCAINE, and TOBACCO. Those who take ibogaine are generally highly motivated since the drug is expensive, costing up to several thousand dollars. While many reported a decrease in drug CRAVING after taking ibogaine, relapse to drug use within a few months was also observed.

As a result of pressure from activists, the U.S. government funded animal studies of ibogaine's actions on opioid and cocaine withdrawal, opioid and cocaine self-administration, and neurotoxicity. Studies in animals have not been entirely consistent. High doses of ibogaine reduced some manifestations of opioid withdrawal in monkeys. Studies in opioid-dependent rodents have shown that ibogaine decreases withdrawal, but other studies have not. Some rodent studies have shown a decrease in drug self-administration. Studies of ibogaine toxicity have also produced mixed results. Some studies in monkeys produced no obvious nervous system toxicity, but a study in rats produced damage to neurons in the cerebellum, the part of the brain known best for its role in control and coordination of movement. Other research studies indicate that ibogaine is not similar to opioids such as MORPHINE and heroin nor to hallucinogens such as LSD in terms of actions at drug RECEPTORS.

Despite these inconclusive research findings, in the early 1990s an FDA advisory committee recommended approval of limited trials in humans aimed at establishing safety and efficacy in treating drug dependence. At least one death has been attributed to the use of ibogaine in the treatment of heroin addiction.

(SEE ALSO: *Ayahuasca; Hallucinogenic Plants; Hallucinogens; Pharmacotherapy; Treatment*)

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ICD-9/10 *See* International Classification of Diseases

ICE *See* Methamphetamine; Slang and Jargon

ILLEGAL AND ILLICIT DRUGS *See* Controls: Scheduled Drugs/Drug Schedules, U.S.

IMAGING TECHNIQUES: VISUALIZING THE LIVING BRAIN Images of the human BRAIN constructed using sophisticated computer systems have proven valuable for studying the effects of abused drugs. Nuclear medicine techniques, such as positron emission tomography (PET) and single photon emission computed tomography (SPECT), allow noninvasive studies of brain function in human volunteers by the administration of small amounts of radioisotopes. These procedures allow visualization and quantification of biochemical processes in the living brain. Functional MRI (magnetic resonance imaging) is a recently developed technique that makes it possible to construct functional brain images without radiation.

PET scanning uses radioisotopes that decay by emitting positrons (positively charged particles), which collide with electrons (negatively charged particles that surround atomic nuclei). In each collision, both the electron and positron are annihilated and energy is released in the form of two photons (quanta of light) that move in opposite directions. The detectors of a PET scanner surround the tissue being studied and register the arrival of photons. The associated computer system can calculate the location of each collision and reconstruct an image of the concentration of radioactivity in different parts of the tissue.

The most common applications of PET scanning involve functional measurements of cerebral (brain) metabolism or cerebral blood flow. PET is also used to map and quantify specific RECEPTORS for drugs and NEUROTRANSMITTERS in the brain. Cerebral glucose consumption (metabolism) and cerebral blood flow both reflect the activity of brain cells. Under normal circumstances, the cerebral metabolism and blood flow are tightly coupled. The most active brain cells require the most glucose, a sugar that is the primary energy source of the adult brain. Brain regions that contain the active cells also require high rates of blood flow for the delivery of nutrients and oxygen. In some conditions, however—including those caused by some drugs—cerebral metabolism and blood flow rates may be dissociated.

Rates of consumption of glucose in the whole brain or in specific brain regions have been measured using fluorodeoxyglucose (FDG) labeled with the positron-emitting isotope fluorine-18 (^{18}F). Cerebral blood flow has been measured using oxy-

gen-15 (^{15}O), either inhaled in $C^{15}O_2$ or injected in ^{15}O-labeled water.

In SPECT, radionuclides that emit single photons are used, including iodine-123 (^{123}I) and technetium-99m (^{99m}Tc), and the photons are measured using a rotating gamma camera. The isotopes used in SPECT have longer half-lives (thirteen hours for ^{123}I and six hours for ^{99m}Tc than those used in PET (110 minutes for ^{18}F and 10 minutes for ^{15}O). Therefore, whereas PET generally requires an on-site cyclotron to produce radioisotopes, SPECT radioactive tracers can be made elsewhere and brought in for use. Although SPECT produces useful images, it does not provide either the quantitative precision or the spatial resolution of PET. Currently available PET scanners can resolve differences in the radioactivity of objects only 4 to 5 millimeters (mm) apart, while the resolution of new SPECT scanners is for 6 to 8 mm.

Before the advent of PET and SPECT, blood flow was measured using xenon-133, given by brief inhalation or intracarotid artery injection. Xenon-133 has a gamma emission with a half-life of 5.27 days, and the radioactivity is monitored outside the skull by an array of detectors that each record a beam of particles from a specific location. Unlike PET, the xenon-133 methods do not provide tomographic information—they do not produce images of "slices" of the brain. Therefore, activity in deep brain structures cannot be measured this way.

Recent advances in magnetic resonance imaging (MRI) technology have permitted functional measurement of cerebral blood volume, which is closely related to cerebral blood flow. Functional MRI assessments are based upon the difference between the paramagnetic properties of oxygenated and unoxygenated hemoglobin. Activation of a brain area causes increased blood flow to the region. Oxygen carried to the activated region is delivered in excess of that which is required by the increased activity. Therefore, it accumulates, as does oxyhemoglobin. Functional MRI produces brain images of very spatial and temporal resolution.

Since researchers are interested in the activity of specific brain structures, data are obtained by functional imaging techniques often adjusted (normalized) to remove the effects of differences between individuals in whole brain activity measurements considered irrelevant to the question under study. Normalized data may be expressed numerically as the quotient of the activity in a region of interest divided by the activity in the whole brain or in the slice containing the region. Such data are not always easy to interpret, since changes in the denominator can obscure the direction and magnitude of change in a region.

ACUTE EFFECTS OF DRUGS

Alcohol. Acute administration of ALCOHOL (ethanol)—a depressant—reduces cerebral glucose utilization, as we learned from measurements taken by the FDG technique. Modest decreases of 15 percent or less are seen in the whole brain in response to a dose of 1 gram/kilogram (g/kg) of ethanol (about 2 oz. of 100 proof whiskey for a 150-lb. person). Slightly more dramatic reductions in metabolism have been noted in the brain's cortex, particularly in the frontal and the occipital regions.

In contrast, acute ethanol administration does not reduce cerebral blood flow. Therefore, ethanol appears to dissociate cerebral blood flow from glucose metabolism. Studies with xenon-133 have indicated that ethanol (0.75 g/kg) increases cerebral blood flow by about 20 percent overall. Furthermore, normalized data obtained by PET scanning, using ^{15}O-labeled water, indicate regional effects of ethanol on cerebral blood flow. The largest changes were noted in the cerebellum (decrease), the prefrontal cortex (increase), and the temporal cortex (increase).

Stimulants. Studies with STIMULANTS have indicated that drugs of this class—including COCAINE and AMPHETAMINE—like the DEPRESSANT alcohol, reduce cerebral glucose utilization. Oral AMPHETAMINE at a dose of 0.5 milligrams/kilogram (mg/kg) decreases cerebral glucose metabolism by an average of about 6 percent of values in the unperturbed state, with no variation in the effect of the drug in different brain regions. A euphorigenic intravenous dose of cocaine (40 mg iv) also reduces cerebral glucose metabolism globally, averaging about a 14 percent decrease overall. The largest reductions occur in the left temporal pole and in the left lateral occipital gyrus.

Benzodiazepines. The effects of diazepam (Valium), a benzodiazepine anxiolytic, on cerebral metabolism and blood flow have also been studied, and results indicate that both of these parameters of brain function are reduced. Glucose metabolism is reduced by taking doses as low as 0.07 milli-

grams/kilogram orally (about 5 mg, the dose that might be given for anxiety), and the effect does not show regional specificity. Small reductions in cerebral blood flow, as measured with xenon-133, are also seen in response to intravenous diazepam (0.1 mg/kg). The reductions average about 6 percent overall, with the largest reduction seen in the right frontal cortex.

Opioids. The acute effects of HEROIN on cerebral metabolism or blood flow have not been reported, but a euphorigenic intramuscular dose of MORPHINE (30 mg) reduces cerebral metabolism globally, averaging about a 10 percent decrease overall. The largest reduction is found in the left superior frontal gyrus.

Marijuana. The active ingredient in MARIJUANA, delta-9-TETRAHYDROCANNABINOL (THC), produces variable effects on global cerebral glucose consumption but increases normalized metabolism in the cerebellum, as is consistent with the localization of cannabinoid receptors to this region. The metabolic effect is correlated with self-reported intoxication and with the plasma concentration of THC.

Effects of Abused Drugs. Taken together, these results indicate that all drugs of abuse that have consistent effects on cerebral metabolism produce decreases, but the magnitude of the decrease varies. This discrepancy is due, at least in part, to differences in dose and route of administration. The regional distribution of drug effects also varies, but the regional differences in percent change are not large in any of these studies. It seems that drugs of abuse—whether classified as depressants (alcohol), stimulants (cocaine), tranquilizers (benzodiazepines), or ANALGESICS (OPIOIDS)—reduce cerebral glucose metabolism globally.

Effects of abused drugs on global cerebral blood flow are less consistent, with decreases by the tranquilizer diazepam but increases by the depressant alcohol. Differences in regional effects of drugs on cerebral blood flow are minimal or absent, and the effects are generally global. Drugs of abuse may influence cerebral blood flow by direct effects on the cerebral blood vessels. Such direct vascular effects do not reflect changes in blood flow to meet the energy demand of the brain—in contrast, measurements of glucose metabolic rates are less sensitive to vascular responses that are seen as alterations in cerebral blood flow. In this respect, glucose metabolism can be a better measure of brain function than cerebral blood flow.

CHRONIC EFFECTS OF ABUSED DRUGS

Long-term drinking (chronic ethanol abuse) has toxic effects on the brain, and imaging techniques have added to the understanding of these effects. Brain glucose metabolism is decreased in recovering alcoholics (abstinent at least seven days), even if they do not show brain damage severe enough to be diagnosed as organic brain syndrome. The largest differences from controls were found in frontal lobe structures. Cerebral blood flow, measured using xenon-133, is also decreased in chronic alcoholics, with the largest differences in frontal and temporal lobe structures. To some extent, the changes are reversible with abstinence. Low cerebral blood flow is related to heavy drinking history, with the lowest flow rates in patients with brain damage (organic brain syndrome) due to alcohol.

Chronic use of cocaine has also been associated with persistent effects on functional markers in the brain. Whether measured by PET or SECT, cerebral blood flow in recovering cocaine addicts (abstinent four to fourteen days) shows focal abnormalities and lower flow rates than controls, particularly in frontal cortex. The etiology of abnormalities in cerebral blood flow in those with histories of cocaine abuse is not clear. In some cases, focal decrements may be related to the use of alcohol or other drugs of abuse or to the dysphoria related to the withdrawal of cocaine. Heroin addicts showed perfusion abnormalities as measured by SPECT during withdrawal (one week of abstinence), but cerebral blood flow had improved by three weeks of abstinence.

Taken together, studies using imaging techniques suggest that chronic use of alcohol and cocaine may damage certain structures in the frontal lobe of the brain. The frontal lobe is thought to be involved in decision making, planning, and other executive functions necessary for self-control. Thus chronic abuse of these drugs may injure the very brain structures that are required for a person to terminate drug use.

Current imaging techniques offer the promise of delineating the anatomical substrates of the acute and chronic effects of drugs of abuse. Such information may contribute to a further understanding

of the causes and the consequences of substance abuse and, ultimately, may lead to more effective prevention and treatment strategies.

(SEE ALSO: *Brain Structures and Drugs; Complications; Reward Pathways and Drugs*)

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IMMUNOASSAY Immunology is a laboratory science that studies the body's immunity to disease. The basic mechanism of immunity is the binding of drugs or other chemical compounds to antibodies (large proteins produced by the body's immune system). An assay is a general term for an analytical laboratory procedure designed to detect the presence of and/or the quantity of a drug in a biological fluid such as urine or serum (the fluid component of the blood obtained after removal of the blood cells and fibrin clot). An immunoassay, therefore, is an analytical procedure which has as its basis the principles of immunology—specifically the binding of drugs to antibodies.

Several different types of immunoassay are routinely performed in the laboratory. Although they differ in the types of reagents and instrumentation used, they are all based on the same scientific principle (the binding of drugs to antibodies). The three types of immunoassay that are commonly used for drug testing are the radioimmunoassay (RIA), enzyme multiplied immunoassay (EMIT), and fluorescence polarization immunoassay (FPIA).

It may facilitate the reader's understanding of immunoassay to envision the reactions that occur in the body following a vaccination (e.g., polio). The vaccine contains a weak or a killed solution of (polio) virus. When the vaccine is injected into the body, the immune system recognizes the presence of a foreigner (the polio virus), and it generates antibodies to that virus. These antibodies circulate in the blood, and they constitute the body's protection; if at some later date a live (polio) virus invades the body, the antibodies recognize it by its unique size and shape (similar to the fit of a lock and key); they spontaneously bind to the virus, leading to its inactivation and removal from the body.

This binding of antibodies to drugs forms the basis for immunoassay. In the development of an immunoassay, the first step is to inject an animal (host) with the drug that we ultimately wish to analyze. The host immune system, recognizing the drug as a "foreigner," generates antibodies to this drug, and these antibodies can then be harvested from the serum of the animal. In the test-tube environment of the laboratory (*in vitro*), these antibodies can be recombined with the appropriate drug. Just as it did inside the body (*in vivo*), the antibody will recognize the drug based on the lock-and-key fit and will spontaneously bind to it.

The second step in the development of an immunoassay is to synthesize a "labeled" drug. This involves the chemical addition of a "marker" to the drug. This marker can be small, such as an atom of radioactive iodine, or it can be large, such as an enzyme, which is a fairly large protein. Irrespective of its size, this marker is added in such a way that it does not interfere with the lock-and-key recognition between the antibody and the drug.

Commercially available immunoassay kits contain the antibody (which the company has prepared as described above) and the labeled drug (which has been chemically synthesized) necessary to perform the assay. In the laboratory, a fixed amount of antibody and a fixed amount of labeled

drug are placed into a reaction vessel (test tube). If these were the only two ingredients, all the binding sites on the antibody would react with (bind to) the labeled drug. A third ingredient added to the assay is, however, the unlabeled drug (i.e., the urine, saliva, or serum specimen containing the drug that is being measured). Because the label on the labeled drug is placed in a position that does not interfere with binding to the antibody (i.e., it is "hidden"), the antibody cannot distinguish between the labeled and unlabeled drug.

Immunoassays are always designed so that there are fewer antibody-binding sites present in the reaction mixture than there are molecules of (labeled plus unlabeled) drug. Because the labeled and unlabeled drug appear the same to the antibody, they will compete equally for the limited number of available binding sites on the antibody. By measuring the amount of labeled drug bound to the antibody, the analyst can calculate the amount of unlabeled drug in the biological specimen.

All immunoassays work in the same basic fashion. They differ in the types of labels that are added to the labeled drug and in the analytical methods by which the amount of binding of labeled drug to the antibody is measured.

RADIOIMMUNOASSAY

Radioimmunoassay (known as RIA) was the earliest of the immunoassay techniques. It was developed during the 1950s by a pair of research immunologists in New York City, Dr. Solomon A. Berson and Dr. Rosalyn S. Yalow. Their initial RIA was designed to detect very low blood levels of insulin and they published their findings in 1959. Their development of this technique was considered of such importance to science that Dr. Yalow was awarded a Nobel prize in 1977 for their work (since Dr. Berson died in 1972 and Nobels are not awarded posthumously, Berson's contribution was remembered in Yalow's acceptance speech).

In RIA, the marker is an isotope of a radioactive element, hence the name *radio*immunoassay. In most RIAs performed in the laboratory today, the radioactive isotope used as the marker is iodine 125, although tritium (hydrogen 2), carbon 14, and cobalt 57 are used in some assays. RIAs can be used in two different fashions to give information about the drug in a sample: (1) they can be used qualitatively—to determine whether a drug is present or absent (e.g., in urine drug testing); (2) they can be used quantitatively—to determine how much of a drug is present (e.g., to measure serum levels of drugs such as digoxin, a heart medication, or theophylline, an asthma medication).

RIA is an extremely powerful tool. One of its main advantages is the sensitivity that can be achieved. Drug levels in serum and urine that are as low as 10 to 100 parts per billion are routinely measured. Two of the most sensitive of the radioimmunoassays are the urine LSD assay and the serum digoxin assay, both of which can detect less than one part per billion. RIA is also an extremely versatile tool. It is used to measure a wide range of drugs of abuse in blood, serum, saliva, and urine, as well as therapeutic (physician administered) drugs in blood or serum. It is also used as a diagnostic tool to detect and quantify numerous naturally occurring chemicals in human serum and urine. Another characteristic that makes RIA such a powerful tool is the specificity of the assay. The antibodies are highly specific for the drugs analyzed and they rarely make a mistake in recognizing the lock-and-key fit between antibody and drug.

One of the major limitations of the radioimmunoassay is that it generates radioactive waste. To avoid spreading the radioactive compounds and contaminating the environment, the laboratory must conform to very strict regulations, including very elaborate procedures for waste disposal—and undergo frequent inspections. Because of a short half-life for some isotopes, another limitation is that the reagents with a radioactive label have a short shelf life. For instance, the majority are RIAs labeled with iodine 125; they have a shelf life of only approximately sixty days.

Some very sophisticated automated equipment is available for performing RIA or, if need be, the assays can be performed manually. All RIAs require the use of an instrument called a gamma counter, which measures the amount of gamma radiation given off by the radioactive drug bound to the antibody. In the 1990s, gamma counters can be purchased for as little as a few thousand dollars; but the reagents are moderately expensive (costing from less than fifty cents/test to two to three dollars/test, depending on the specific assay and the volume of reagents purchased).

ENZYME MULTIPLIED IMMUNOASSAY

The enzyme multiplied immunoassay technique, also known as EMIT™, is a variation of the general immunoassay technique, in which the marker used to prepare the labeled drug is an enzyme, rather than a radioactive isotope. EMIT is a two-stage assay. As in the other immunoassays, the sample, which contains some amount of the drug being measured, is combined with the antibody plus a fixed amount of the enzyme-labeled drug. In the first reaction, the labeled and the unlabeled drug compete for the available binding sites on the antibody (standard immunoassay reaction). A secondary reaction is then performed, which involves only the enzyme portion of the labeled drug. The results of this secondary reaction are used to calculate the amount of enzyme-labeled drug that is bound to the antibody and thus how much (unlabeled) drug there was in the original urine or serum specimen.

As with other forms of immunoassay, the EMIT can be used either qualitatively or quantitatively. In urine specimens, it is used to detect the presence of drugs, such as THC (MARIJUANA), COCAINE, PCP, OPIATES (HEROIN), AMPHETAMINES, and BARBITURATES. In serum specimens, EMIT is used to determine the amount present of drugs used for therapeutic (medical) purposes. Such drugs include acetaminophen (Tylenol), salicylate (aspirin), theophylline (widely used to treat asthma), several drugs used to treat epilepsy, and several drugs used to treat heart abnormalities.

Advantages that the EMIT technology has over the RIA are (1) that no radioactivity is involved, so the waste is more readily disposable; (2) the reagents are relatively stable, which may be particularly attractive to a small laboratory, which runs only a few specimens. The EMIT reagents are also less costly than the RIA reagents. The basic instrumentation requires less capital outlay than does the RIA, however the expense grows as more sophisticated automation is acquired.

Some limitations of the EMIT technique are (1) that it is somewhat less sensitive than the RIA (in particular, the LSD assay requires detection of such minute levels of the drug in urine that it can only be done by RIA); (2) also, EMIT is less specific than RIA and is subject to some interferences that do not affect the RIA—for example, the EMIT assay for amphetamines in urine gives a positive response with several other drugs that are similar in structure to amphetamines.

FLUORESCENCE POLARIZATION IMMUNOASSAY

Fluorescence polarization immunoassay (known as FPIA) is a technique that was developed by Abbott laboratories and marketed under the trade name TD_X. As the name FPIA implies, the marker for the labeled drug is a molecule of a naturally fluorescent compound called fluorescein. The amount of labeled drug that binds to the antibody is measured by a sophisticated instrument called a spectrofluorometer. As with the other immunoassays, this measurement is used to calculate the amount of labeled drug bound to the antibody and thus the amount of drug in the original urine or serum specimen.

The instrumentation necessary to perform the FPIA is only made by Abbott. It is expensive to purchase (upwards of $50,000) but can be leased from the manufacturer. The reagents are more expensive than EMIT reagents, being roughly comparable in cost to RIA reagents. They come in a liquid form and have a more limited shelf life than those for EMIT, but they tend to be more stable than RIA reagents.

The attractiveness of FPIA is in the speed and ease of operation of the instrument. The reagents come in a kit that is bar coded and is placed right into the instrument. All the operator has to do is fill the sample cups with serum or urine, place the reagent pack inside the instrument, and push a button marked "run." The instrument reads the bar code, enters the necessary programs into its memory, performs the assay, and prints out the results. For the routine hospital lab or small drug-testing lab, it is as fast or faster than EMIT or RIA and a lot easier; however, the instrument can only run twenty specimens at a time. For the large drug-testing laboratory, more rapid results can be achieved with the automated instrumentation available for the EMIT or RIA techniques.

FPIA is nearly as sensitive as RIA; digoxin can be run by FPIA, although LSD is still not available. The specificity of FPIA is also comparable to that of RIA.

(SEE ALSO: *Drug Testing and Analysis; Hair Analysis as a Test for Drug Use*)

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JEFFREY A. GERE

IMPAIRED PHYSICIANS AND MEDICAL WORKERS Concern about impairment from alcohol and drugs in health-care professionals in the United States and in other countries has waxed and waned during the twentieth century. Until the 1960s, ALCOHOL, the OPIATES, and other PRESCRIPTION DRUGS were the primary concerns. More recently, the concern was extended to MARIJUANA and COCAINE.

Although there are many estimates of addiction rates among physicians, the prevalence of alcohol and other drug problems within the entire health-care profession is unknown. Brewster (1986) reviewed published estimates of U.S. addiction rates among physicians and found that available reports were not adequate to support firm conclusions about the prevalence rates. Adding to the difficulty is the physician's ability of self-medication, and the fact that much of the detection of abuse to begin rehabilitation can come only after a voluntary confession.

Physicians (since we do have data on them) are as likely as their age and gender peers to have experimented with drugs—both licit and illicit. They are, however, less likely to be current users of illicit substances (Hughes et al., 1992). Self-medication by physicians has changed little since the 1960s, whereas the use of cocaine and marijuana has greatly increased (McAuliffe et al., 1986).

Figure 1 shows the results of three surveys of drug use among U.S. medical students. Substantial numbers of medical students come to medical training having had some experience with illicit drugs.

Disciplinary or diversion actions by health professionals' licensing boards and studies of health professionals who receive treatment for alcohol or drug dependency are additional sources of information about the kinds of problems caused by drugs and alcohol and their relative frequency.

It is widely believed that health-care professionals are especially vulnerable to problems of alcohol and drug abuse because of familiarity with and ready access to drugs, the high STRESS associated with patient-care responsibilities, and their

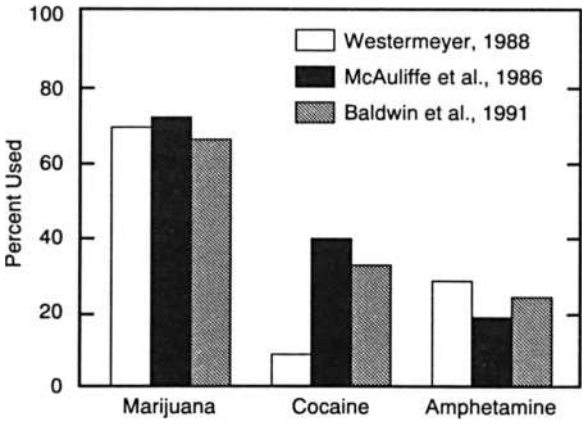

Figure 1

Lifetime Drug Use by Medical Students. Westermeyer (1988) surveyed first-year medical students (n = 195) at the University of Minnesota. During 1984–1985, McAuliffe et al. (1986) mailed anonymous questionnaires to a random sample of medical students in New England (n = 381). In 1987, Baldwin et al. (1991) mailed questionnaires to senior medical students at 23 schools located throughout the country (n = 2,046).

NOTE: n is the number of medical students who returned questionnaires.

own family problems may also contribute. Many physicians self-prescribe medications for relief of PAIN and ANXIETY. A 1989/90 survey of U.S. physicians found that 11.4 percent had used BENZODIAZEPINES and 17.5 percent had used minor opiates during the preceding year without medical supervision (Hughes et al., 1992a).

While the problem of drug addiction among health-care professionals is now widely acknowledged, such awareness has not always been the case. Impaired physicians and other health-care workers have been fearful of seeking help because they have not known how patients and colleagues might respond, which has become even more complicated since courts in numerous states have determined that physicians are not obligated to reveal their drug and alcohol practices to patients. Doctors also do not reveal their difficulties because they fear loss of practice privileges and licenses. Like many other professionals, physicians often feel uncomfortable about confronting drug or alcohol abuse in a colleague. They want to believe that a colleague in trouble will know when to seek help and will voluntarily seek it. The reluctance of physicians to report colleagues has been called a conspiracy of silence.

In 1972, the American Medical Association (AMA) Board of Trustees accepted the report of its Council on Mental Health and officially ended the conspiracy of silence by making physicians ethically responsible to recognize colleagues' inability to adequately practice medicine—an inadequacy that includes difficulties caused by drug or alcohol abuse. The council recommended a series of steps that should be taken if the impaired physician does not curtail practice: referral of the problem to the medical staff of hospitals in which the physician practices; referral to the state or county medical association; or, if other steps fail, referral to the licensing agency. In 1974, the AMA drafted model legislation allowing states' licensing agencies to require treatment and rehabilitation of impaired physicians as a condition of maintaining licensure. Before that time, the only possible response of the licensing agency was to discipline the physician. Since then, many state medical societies and licensing bodies have established programs for health professionals with alcohol or drug addiction.

In response to increasing malpractice, the U.S. Department of Health and Human Services established the National Practitioner Data Bank to collect information about malpractice and state-board licensing actions, hospital restrictions, revocation or denial of privileges, or denial of membership by a professional society. The purpose of the data bank is to prevent physicians from moving from state to state and continuing to practice without disclosing previous adverse actions against them. Hospitals must request information from the data bank when a physician applies for clinical privileges. The data bank prevents physicians with untreated alcohol or drug dependencies who have been disciplined in one state from practicing without restrictions in another state.

As a means of detecting drug and alcohol abuse, random urine testing is sometimes proposed for physicians and other health-care professionals. The AMA opposes routine urine testing because it intrudes on personal privacy and because a positive test does not establish impairment. Furthermore, drug- and alcohol-induced impairments are complex psychosocial and neurobehavioral problems that require a comprehensive clinical assessment, and neurobehavioral testing may better reflect the degree of impairment. Urine testing is useful for other purposes, though It is, for example, one of the best ways to document abstinence, which is an indicator of treatment progress.

DIFFERENCES IN PREVALENCE BY SPECIALTY

The choice of a particular drug and route of administration is influenced by accessibility and familiarity. Among anesthesiologists, for example, injectable fentanyl and its analogs are the most frequently abused opioids (see Table 1).

Although opioid addiction—and addiction treatment—among anesthesiologists has received frequent notice, the addiction rate among anesthesiologists may not be higher than among other physicians. Opiate abuse among anesthesiologists may be discovered more frequently because of the hospital environment in which they must practice. Interpersonal stress and the isolation of an office practice are believed to make psychiatrists particularly vulnerable to alcohol and drug abuse. The privacy of a solo office practice also makes detection difficult.

TABLE 1
Drugs of Abuse by Anesthesiologists

Drug of Abuse	*California Diversion Program* n = *42* *(percent)*	*Resident Anesthesiologists* n = *132* *(percent)*
fentanyl/sufentanyl	40	80
meperidine (Demerol) and other opiates	29	17
alcohol	17	11
cocaine	10	3
diazepam (Valium)	2	12
inhalation anesthetics (e.g., nitrous oxide)	0	8
ketamine	0	6
other	2	6

Data from the Diversion Program of the Medical Board of California shows the *primary* drug of abuse of anesthesiologists who were participating in the program during 1989 (Ikeda & Pelton, 1990). The data on resident anesthesiologists show their drug or drugs of abuse (Menk et al., 1990). Some residents abused more than one drug.

DETECTION OF ADDICTION

In hospitals, drug use by health-care professionals is often uncovered during inventory audits of medications, and the concealment efforts of impaired health-care professionals are often reflected in their treatment of or attitude toward patients. Some physicians who abuse prescription medications routinely overprescribe opiates or other drugs to patients in an effort to hide their self-prescription; others may prescribe unusually conservatively to avoid drawing attention to themselves.

TREATMENT

Many health professionals are pessimistic about the treatability of substance abuse, and if they develop an alcohol or drug problem, they may discount the value of treatment for themselves. Those who train or work in public-sector hospitals or clinics often observe that the treatment of their patients is rarely successful. Their perception is unduly pessimistic, however, because such clinics often treat recalcitrant, end-stage substance abusers. Furthermore, recent observation has seen medical care givers surpass all other professions as the most successful with intervention programs. This is possibly attributable to the ability of doctors to notice the difficulties in colleagues and the consequential early response.

The resistance to seeking treatment on their own often necessitates some form of coercion to force health professionals into treatment. One method of breaking down denial and forcing a person to seek treatment is called an *intervention*. The process consists of a group confrontation of the drug-abusing professional by concerned friends, family, and colleagues. A peer professional experienced in conducting interventions often assists in setting up the confrontation. The interventionist rehearses those who will be involved. When the stage is set, participants each tell the abuser what they have observed concerning the drug abuse and how it has adversely affected them. The confrontation, which may include threats to notify the abuser's employer, hospital, or state licensing board, may motivate an abuser to go for treatment. Such motivation is often fleeting, so it is important for the addict to go immediately into a treatment setting.

TREATMENT MODALITIES

Most treatment for impaired health professionals is drug-free and recovery-oriented, emphasizing follow-up participation in ALCOHOLICS ANONYMOUS (AA), COCAINE ANONYMOUS (CA), or

other peer-led groups. Recovering physicians rate participation in AA, for example, as an important factor in their recovery. In most respects, treatment of addiction for health professionals differs little from that used for other middle- and upper-class patients. Health-care professionals who abuse prescription drugs often see themselves as being different from street-drug users. Some programs deal with this form of resistance by insisting on a uniform treatment for all patients. There are, however, special problems that must be addressed. For example, addicted physicians, unlike street addicts, often underreport their degree of PHYSICAL DEPENDENCE on a substance in an effort to project a false sense of being in control. A period of inpatient treatment is often required.

METHADONE MAINTENANCE, which has been employed successfully for some HEROIN (and other OPIOID) addicts, is generally not an option for practicing health professionals, since most licensing boards will not allow them to practice while taking methadone. NALTREXONE has been particularly successful with health-care professionals and is the only medication for treatment of opioid dependency acceptable to most licensing boards. The ingestion of naltrexone reassures licensing boards and hospitals that the recovering health professional is not impaired from abuse of opioids. Its lack of mood-altering effect also fits well with the drug-free treatment philosophy.

WORK REENTRY

Work reentry can be difficult for recovering health professionals. Those who have abused prescription medication face reexposure to their drugs of abuse, which could lead to relapse. Hospital and other professional privileges are not easily regained. Licensing boards often opt to revoke or restrict the impaired health professional's license to practice, and insurance companies often refuse malpractice coverage to recovering addicts.

Some of these obstacles can be overcome with planning and peer support. For example, a nurse may find employment in a blood bank or other setting in which there is no access to drugs. Also, a physician may make arrangements to have a colleague see all the patients that require a NARCOTIC, thus avoiding having to write narcotic-containing prescriptions. Reentry may involve redirecting the health-care practitioner's professional activities to a different location or area of treatment, restricting the recovering health professional's scope of practice, or removing him or her from the previous practice environment altogether. For many health professionals, return to full practice after a period of monitored abstinence and compliance with treatment is possible.

One matter that remains unnegotiable, however, is the safety of the public. Medical boards do find it is their responsibility to aid the physician in reentering the workforce, but not at the expense of the health of patints.

RESPONSE TO TREATMENT

Prognosis for physicians treated for ALCOHOLISM or drug dependency is generally favorable. A study comparing physicians with other middle-class patients similarly treated in an inpatient program showed that physicians did better. The California Physicians' Diversion Program reported a 69 percent success rate for anesthesiologists and an overall success rate of 73 percent. This success is attributed to regular attendance at group meetings, regular testing for sobriety, and immediate corrective action whenever a slip or relapse occurs.

Such high rates of success are not uniformly attained. In a survey of training programs for anesthesiologists, it was found that of the seventy-nine anesthesiology residents who returned to their specialty following treatment, only twenty-seven (34%) did not relapse—and of the fifty-two who relapsed, thirteen (25%) died of drug overdose (Menk et al., 1990).

Some medical specialties are more stringent than others in allowing recovering trainees to return. Minor slips that are often dealt with by additional treatment in some specialties are usually not acceptable in anesthesiology training programs. Therefore, comparison of recovery rates between treatment programs and different subgroups of physicians is difficult to impossible.

(SEE ALSO: *Coerced Treatment; Contingency Management; Drug Testing and Analysis; Industry and Workplace, Drug Use in*)

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INDIA, DRUG USE IN *See* Asia, Drug Use in; Bhang

INDUSTRY AND WORKPLACE, DRUG USE IN The 1991 NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE (NHSDA) indicated that roughly ten million employed Americans were "current users" of illegal drugs. Drug use by employees and workers has become an important issue for American business. In the 1990s, employers of all types (large and small businesses, nonprofit organizations, government) are attempting to contain the negative impact of illegal drug use on job performance, PRODUCTIVITY, safety, and health (Walsh & Gust, 1989).

Drug use "in the workplace" is perhaps a misnomer in that today's employer policies focus on drug use by the "worker," whether that use is at the work site or off the job. In the United States, drug abuse in the workplace was recognized as a serious problem in the early 1980s, and in the next decade a slow but progressive response by both labor and management yielded model programs and policies to deal with the issue. Typically, by 1993 most

organizations had comprehensive programs that included the following basic components: a written policy; supervisory training; employee education; employee assistance resource; and DRUG TESTING.

With such a comprehensive approach, the workplace has proven to be one of the most effective venues for drug prevention, treatment, and rehabilitation efforts (Gust & Walsh, 1989; Gust et al., 1991). Figure 1 shows the NHSDA data for "current users" (those reporting use of an illegal drug within thirty days prior to the survey), with figures broken down by employment status. Many observers believe that comprehensive workplace-based programs that reach out not only to the workers but also to their spouses and their student children can have an impact on at least 80 percent of all current users.

These endeavors have not been undertaken without controversy, especially with regard to the use of employee "drug testing," but the controversy is perhaps the most interesting part of the story. The development, current status, and future of these policies and programs will be discussed in detail later in this article. The evolution of workplace-based antidrug programs provide insights into the way unique social problems create a need for and the eventual development of innovative public policy (for additional information, see Walsh & Yohay, 1987).

Workplace "antidrug" policies date back to the 1960s, particularly in the transportation and other safety-sensitive industries. These policies were not then very effective because detection methods were poor and the signs and symptoms of drug use are often subtle and difficult to identify. Not until 1980, when new technology became available that provided reliable, inexpensive detection methods for MARIJUANA and other commonly abused drugs, did workplace detection efforts begin to be effective. Interestingly, the "workplace" that triggered the birth of these antidrug initiatives was the U.S. military.

In 1971, President Richard M. Nixon, as commander in chief, changed the Uniform Code of Military Justice so that testing positive for an illicit drug was no longer a court-martial offense. He ordered the military to start a program of urine testing among U.S. troops in VIETNAM and to offer treatment to those who tested positive for drug use. This urine-testing program was then expanded to service personnel worldwide. In the mid-1970s, the

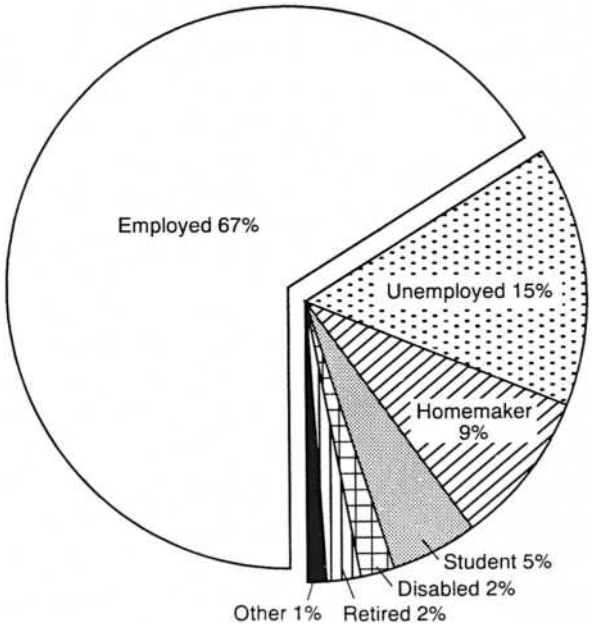

Figure 1
Employment Status of Illegal Drug Users

program was discontinued as a result of court challenges. At the time, the only drugs that could easily be tested for were OPIOIDS and some STIMULANTS.

The remarketing and reintroduction of drug-testing technology in 1981 occurred at roughly the same time as the Department of Defense (Burt & Biegel, 1980) and the Congress (House Select Committee on Narcotics, 1981) independently reported the survey results of drug use by MILITARY personnel. The results of these two surveys indicated high rates of drug use by military personnel and brought about considerable congressional scrutiny. The accident on the aircraft carrier U.S.S. *Nimitz* in May 1981, in which drug use was discovered by the postmortems of the crew members, increased political pressure on the military to do something about the drug-abuse problem. The juxtaposition of these events—the availability of drug-testing technology and congressional demands for the Defense Department to address drug taking in the military—was pivotal in justifying the widespread application of drug testing and the formulation of strict policies forbidding the illegal use of drugs on or off the job.

The development of such policies in the military received wide media coverage and generated much discussion in 1981. Shortly thereafter (1982–1983), similar policies began to be adopted in the transportation and utility industries for employees

in safety-sensitive positions. The National Transportation Safety Board documentation of drug involvement in railroad and airline accidents more than justified the increasing concerns about workplace drug abuse.

Early military and private-sector policies were punitive in nature; employees found to be using drugs were summarily dismissed. This created a dilemma for many major corporations that recognized they had a drug problem but didn't feel comfortable firing employees, especially when there was no safety or security nexus. The rationale for workplace drug policies and the use of drug testing has evolved considerably since 1981. The philosophy of why to test and what to do with the results changed dramatically during the 1980s and early 1990s. At the outset, the primary purpose of a drug policy was to identify users and to fire them without evaluating the circumstances of the drug use. Subsequently a more positive, helping-hand philosophy evolved.

The basic purpose of today's model corporate drug policy is twofold:

1. to minimize the risk of hiring drug users by denying employment to applicants who use illegal drugs (as manifested by a positive preemployment drug test); and
2. to provide active programs to get the substance-abusing employee into treatment, to afford the opportunity to get help, and to get the individual back on the job.

This philosophical change to a more politically acceptable, socially responsible policy in dealing with drug abuse evoked about 1986 and allowed major corporations and professional organizations to involve themselves in antidrug workplace initiatives.

The federal government facilitated, encouraged, and in some instances required the development of private- and public-sector workplace antidrug programs. The Federal Railroad Administration began hearings on drug rules for the railroad industry in 1984 and issued regulations requiring written policies and the testing of employees; after a number of legal delays, the regulations went into effect in 1986. In September 1986, President Ronald W. Reagan issued an executive order (EO 12564) that required all federal agencies to develop drug-free workplace programs to ensure that the more than 2 million federal employees were not illegally using drugs on or off the job. In 1988 the Department of Transportation issued regulations for the airline, maritime, trucking, railroad, pipeline, mass transit, and other transportation industries, requiring (1) written policies prohibiting the illegal use of drugs on or off the job and (2) preemployment, reasonable-suspicion, postaccident, and "random" drug testing without cause for employees in specified safety-sensitive occupations. By 1992 the regulations were extended to cover intrastate as well as interstate transportation, a move that increased the number of transportation workers affected by the regulations to ten million, or nearly 10 percent of the total U.S. workforce. The Nuclear Regulatory Commission also issued regulations requiring written policies and extensive testing of personnel at nuclear sites.

Congress got on the bandwagon and passed the Drug-Free Workplace Act of 1988, which requires all federal grant recipients and federal contractors (whose contracts exceed $25,000) to certify that they will provide a drug-free workplace. The final rules describing the requirements for such grantees and contractors were published in the *Federal Register* on May 25, 1990. In general, the law requires covered employers to:

1. Develop and publish a written policy and ensure that employees read and consent to the policy as a condition of employment;
2. Initiate an awareness program to educate employees about:
   the dangers of drug abuse
   the company's drug-free workplace policy
   any available drug counseling, rehabilitation, and employee-assistance programs
   the penalties that may be imposed on employees for drug-abuse violations;
3. Require that all employees notify the employer or contractor of any conviction for a drug offense in the workplace;
4. Make an ongoing effort to maintain a drug-free workplace.

In 1988, the Bureau of Labor Statistics (BLS) surveyed business establishments throughout the United States about their policies on drug abuse (BLS, 1989). The survey found that half the nation's nonagricultural workforce was employed by organizations with a formal policy on drugs, and that 20 percent of payroll workers were employed in establishments with some type of drug-testing

program. More than 90 percent of the establishments surveyed had an EMPLOYEE-ASSISTANCE PROGRAM available to employees. In the years since the BLS survey, the number of corporate and other employers and the employees covered by these policies continued to grow exponentially. The American Management Association (Greenberg, 1993) has surveyed its membership about their workplace drug policies annually since 1987. The 1993 survey indicated that 84 percent of respondents believe that drug testing is an effective way to deal with workplace drug abuse, compared with 50 percent in 1987. The share of surveyed firms that test for drugs rose to 85 percent in 1993. Since 1987, drug testing has increased nearly 300 percent. From the drug-treatment perspective, more than half of all companies (54%) have indicated that employees who test positive are referred for counseling and treatment.

As indicated above, progress in using the work site to intervene in individual substance abuse has not happened without controversy. Generally, employees and workers have no problem with supervisory training or employee education. However, the utilization of drug testing to make employment decisions and the involvement of employee-assistance programs (EAPs) in what many feel is a policing action generates an emotional, gut-level response from both labor and management. The drug testing and EAP components are so critical to any workplace effort that a detailed discussion of the issues is required.

DRUG TESTING

When drug testing is considered, it is important to be familiar with the basic issues with which management and labor have been struggling (a full range of issues are discussed in Walsh & Trumble, 1991). The question of whether to utilize drug-testing technology evokes a complex array of moral, social, ethical, medical, scientific, and legal issues for many Americans. Although most citizens do not condone drug abuse, their concerns about the erosion of civil liberties generate feelings of uncertainty as to whether the end justifies the means. "Where will it stop? Where do you draw the line?" are questions raised by unions, civil libertarians, and others who worry that employee AIDS testing and pregnancy testing will be the next battlegrounds.

Many Americans view the drug-testing process (i.e., collection of urine) as degrading and dehumanizing. Government employees, unions, and civil libertarians argue strongly that drug testing is an invasion of privacy, that it constitutes an illegal search and seizure (i.e., of body fluids) and therefore violates individual rights guaranteed by the Constitution. In general, the constitutional protections apply only to testing conducted by the government (federal, state, and local). Therefore, test-

TABLE 1
Some Recent Attempts to Define Alcoholism and/or Drug Dependence

Drug dependence. A state, psychic and sometimes also physical, resulting from the interaction between a living organism and a drug, characterized by behavioural and other responses that always include a compulsion to take the drug on a continuous or periodic basis in order to experience its psychic effects, and sometimes to avoid the discomfort of its absence. Tolerance may or may not be present. A person may be dependent on more than one drug. (*World Health Organization Technical Report Series*, 1969, no. 407, p. 6.) This definition was reaffirmed in the WHO Expert Committee on Drug Dependence Nineteenth Report, *World Health Organization Technical Report Series*, 1973, no. 526, p. 16.

Alcoholism is a chronic, progressive, and potentially fatal disease. It is characterized by tolerance and physical dependency or pathologic organ changes or both, all of which the direct or indirect consequences of the alcohol ingested. (National Council on Alcoholism/American Medical Society on Alcoholism, 1976.) (See Flavin & Morse, 1991.)

The 1976 definition was revised and broadened in 1991 to include the concept of *denial:*

Alcoholism is a primary, chronic disease with genetic, psychosocial, and environmental factors influencing its development and manifestations. The disease is often progressive and fatal. It is characterized by continuous or periodic impaired control over drinking, preoccupation with the drug alcohol, use of alcohol despite adverse consequences, and distortions in thinking, most notably denial. (National Council on Alcoholism and Drug Dependence/American Medical Society on Alcoholism, 1976) (See Flavin & Morse, 1991.)

TABLE 2
A Comparison of ICD-10 and DSM-IV Criteria for Dependence

ICD-10 Dependence Syndrome	*DSM-IV Substance Dependence*
A cluster of physiological, behavioural, and cognitive phenomena in which the use of a substance or a class of substances takes a higher priority for an individual than other behaviours that once had greater value. A central characteristic of the syndrome is the desire (often strong, sometimes overpowering) to take psychoactive drugs (medically prescribed or not), alcohol, or tobacco. There may be evidence that return to substance use after a period of abstinence leads to a more rapid reappearance of other features of the syndrome than occurs with nondependent individuals. *Diagnostic guidelines* A definite diagnosis of dependence should usually be made only if three or more of the following have been experienced or exhibited during the previous year: (a) a strong desire or sense of compulsion to take the substance; (b) difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use; (c) a physiological withdrawal state . . . when substance use has ceased or been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms; (d) evidence of tolerance, such that increased doses of the substance are required to achieve effects originally produced by lower doses (examples are alcohol- and opiate-dependent individuals who may take doses sufficient to incapacitate or kill nontolerant users); (e) progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects; (f) persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use, or drug-related impairment of cognitive functioning; determination should be made of the user's actual or expected awareness of the nature and extent of the harm. Narrowing of the personal repertoire of patterns of psychoactive substance use has also been described as a characteristic feature (e.g. a tendency to drink alcoholic drinks in the same way on weekdays and weekends, regardless of social constraints that determine appropriate drinking behaviour).	A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three or more of the following occurring at any time in the same twelve-month period: (1) tolerance, as defined by either of the following: (a) need for markedly increased amounts of the substance to achieve intoxication or desired effect (b) markedly diminished effect with continued use of the same amount of the substance (2) withdrawal, as manifested by either of the following: (a) the characteristic withdrawal syndrome for the substance . . . (b) the same (or closely related) substance is taken to relieve or avoid withdrawal symptoms (3) the substance is often taken in larger amounts or over a longer period than was intended (4) a persistent desire or unsuccessful efforts to cut down or control substance use (5) a great deal of time is spent in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances), use the substance (e.g., chain-smoking), or recover from its effects. (6) important social, occupational, or recreational activities given up or reduced because of substance use (7) continued substance use despite knowledge of having had a persistent or recurrent physical or psychological problem that was likely to have been caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced depression, or continued drinking despite recognition that an ulcer was made worse by alcohol consumption) Specify if: *with physiological dependence:* Evidence of tolerance or withdrawal (i.e., either item [1] or [2] is present): *without physiological dependence:* No evidence of tolerance or withdrawal (i.e., neither item [1] nor [2] is present).

ing conducted by private employers is not covered by the constitutional safeguards. However, government-mandated drug testing of private-sector employees—for example, in the federally regulated transportation and nuclear-power industries—must also pass constitutional muster. Although several of these constitutional questions have been brought before the Supreme Court and have generally been upheld, many specific issues may not be resolved by the current cases and will likely continue to be the subject of litigation for some time. This legal uncertainty— whether testing will be upheld and programs go forward or will be found unconstitutional and therefore be restricted—has created confusion for policymakers as well as for employees and unions.

Medical and scientific questions about the accuracy and reliability of drug testing were and are continually raised by those who oppose testing. Concerns have been voiced that many laboratories offering drug-testing services do not have the expertise or capability to perform the assays required. In addition, many employers may be using inappropriate technology and falsely accusing employees of drug use. Congressional support for these concerns has been manifested by the passage of legislation (P.L. 100-71, sec. 503, July 11, 1987) that requires stringent technical and scientific procedures for federal workplace drug-testing programs, as well as standards for the certification of laboratories engaged in drug testing for federal agencies. Similar legislation has been introduced in both the U.S. Senate and House of Representatives that would require such standards and lab certification for the private sector.

In response to concerns about the accuracy and reliability of drug testing, the U.S. Department of Health and Human Services (HHS) issued *Technical and Scientific Guidelines for Federal Drug Testing Programs* (Walsh, 1988). These guidelines are mandatory for federal programs and have rapidly become the gold standard for private-sector programs as well. By 1993, the rigor of the federal standards virtually eliminated concerns regarding accuracy and reliability. The issue of the quality of laboratories has also been addressed by HHS through the establishment of a national laboratory certification program. The College of American Pathologists has also established a forensic urine drug-testing certification program making certified labs available in virtually every state. The use of a certified lab has become the standard by which drug-testing programs are measured. A consensus report from HHS on the scientific issues of drug testing provides detailed information (Finkle et al., 1990).

A discussion of the pros and cons of drug testing provides no clear answers (Walsh & Trumble, 1991). The American Civil Liberties Union (ACLU) has been among the most vocal organizations actively lobbying against drug testing. In addition to constitutional issues, a major concern has been the potential for abuse by managers and supervisors to discriminate against and harass employees. The focus of the ACLU argument is that a positive urinalysis does not prove intoxication or impairment of performance; therefore it cannot be used to draw a nexus between drug use and job performance.

For their part, employers have wrestled with competing objectives and values to develop substance-abuse policies that fulfill multifaceted obligations. On the one hand, many employers feel a moral obligation to do all they can to achieve a drug-free workplace. They have corporate responsibilities to provide a healthy and safe workplace for all employees and to protect shareholders from losses resulting from drug abuse. On the other hand, employers have obligations to their workers—to respect the individual rights and civil liberties of loyal and trustworthy employees (who for the most part are not involved with drugs).

This is an exceedingly difficult balancing act, and, as workplace policies are designed, the balance will shift depending on the individual work site and the nature of the particular job.

EMPLOYEE-ASSISTANCE PROGRAMS

EAPs have become the key component of model workplace policies (Masi, 1984). Although drug testing has provided the major turning point in the evolution of workplace antidrug programs, the EAPs have expanded, grown more sophisticated, and become a vital part of the antidrug initiative. EAP programs were developed in the 1970s to focus on ALCOHOL abuse and to assist employees in dealing with the stresses of employment and personal life. Typically EAP programs provide short-term counseling and serve as a referral source for those employees who need treatment or long-term counseling. So-called broad-brush EAP programs provide a variety of services, in addition to crisis

intervention, including management training and health workshops and seminars (e.g., SMOKING CESSATION, weight reduction).

As managers began to develop antidrug policies, the question was raised: What will we do if we find an employee using drugs? Generally, corporate lawyers and security officers would suggest termination, while corporate medical and EAP staff would recommend treatment. The issue proved difficult to resolve for many corporations when the cost of treatment and the uncertainty of success weighed heavily on the minds of financial officers responsible for making a profit in a bad economy. Fortunately, most corporations have EAP resources to implement the "helping-hand" approach that management sought.

The involvement of the EAP program in the antidrug effort was also not without problems. Initially some EAP providers had difficulty expanding their programs to deal with illegal drug users, a different type of client from ones with whom they had previously worked. The illegal aspect of drug behavior was troublesome in a field where confidentiality is the cornerstone of the therapeutic relationship. Also the advent of drug testing created an ethical dilemma for the EAP provider who was accustomed to being an ombudsman between management and labor. A good percentage of EAP referrals were coming from the drug-testing program in a last-chance situation in which the pressure was on the EAP to "cure" the problem—or management would fire the employee. In the past, many employees using EAP services had sought assistance on their own, and management was never aware of the employee's initiative.

Despite these problems, the EAP field has expanded its efforts to treat substance abuse and has proven to be integral to the entire program. Employers have recognized that EAP programs not only help employees but are cost-effective. New materials, training programs in substance abuse, and certification programs have developed that have made the EAP provider more skilled in dealing with the drug-using employee.

SUMMARY

Although drug abuse in the workplace is still a significant concern of American employers, substantial progress has been made since the early 1980s. Companies with comprehensive programs report significant reductions in accidents, absenteeism, and positive drug tests. There continues to be progressive growth in small and mid-size businesses, as resources for EAP, testing, management training, and legal services are being made available through local business consortia. The business community has developed a consensus that the workplace is an appropriate site for confronting drug abuse and has sent a clear message to the workforce and to the community that drug use will not be tolerated.

For the future, we are likely to see continued growth and expansion of workplace programs. As the country has gained confidence in the accuracy and reliability of drug testing, lower thresholds will be permitted that will make it much more difficult for the casual user to escape detection. We will probably see federal legislation setting additional standards for workplace programs, including standards for testing and for protection of employees.

Educating high school and college students that they must be drug-free to get and hold a job will in the long run contribute significantly to the reduction of drug abuse in the student population. And finally, because the workplace efforts are the most organized drug education, prevention, and treatment initiatives in the country today, they represent the best prospect for turning around the drug problem in America.

(SEE ALSO: *Accidents and Injuries From Drugs; Drug Metabolism; Hair Analysis as a Test for Drug Use; Prevention*)

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MICHAEL WALSH

INHALANTS Inhalants are solvents or volatile anesthetics that are subject to abuse by inhalation. Most are central nervous system (CNS) depressants, but some are convulsants. As a class they are characterized by high vapor pressure and significant solubility in fat at room temperature. Vapors and gases have been inhaled since ancient times for religious or other purposes, as at the oracle at Delphi. Experimentation with inhalants did not occur to any significant extent until after the discovery of nitrous oxide and the search for volatile anesthetics commenced in earnest. Arguably the most toxic of abused substances, inhalants can produce a wide range of injuries, depending on the chemical constituents of what is inhaled. Many are very complex mixtures formulated for a specific purpose, or are used because they are the least expensive alternative, or both. Thus their purity and safety are in no way comparable with those achieved by pharmaceutical companies manufacturing medications for human consumption.

Inhalants are typically abused by achieving a high airborne concentration of a substance and deliberately inhaling it. With solvents, this typically involves putting the solvent in a closed container, or saturating a piece of cloth and inhaling through it. Compressed gases are sometimes released into balloons and inhaled; directly releasing these substances into the mouth may freeze the larynx, causing laryngospasm and death by asphyxiation. Once the chemical is inhaled, its uptake and duration of action are determined by its solubility in blood and brain, and by the respiratory rate and cardiac output.

The mechanism of action of this class of agents is less well understood than those of other drugs and medications. As CNS depressants, they have been thought to exert their actions by dissolving in membranes and altering their function in a nonspecific way; the potency of these compounds is frequently related to their solubility in membranes. Many consider this relationship to better predict the access of the agent to the site of action, and to be unrelated to the mechanism by which the solvents exert their effects. Solvents impair conduction in isolated nerves, and affect nerves with smaller diameters first. This suggests that parts of the nervous system such as the cortex would be affected before systems consisting of large fibers. There is significant interest in the GABA receptor complex as the site of action of many of these compounds. There is not yet evidence for specific interactions with a receptor, in the sense of a "lock and key" mechanism. However, these agents may "lubricate" or "obstruct" such mechanisms.

Although inhalant abuse has been implicated in a variety of organic diseases, its effects on the nervous system have been of the greatest concern. Such injuries range from paralysis and loss of bowel and bladder control, to permanent impairment of the higher cognitive functions and fine motor control. Those who become involved in inhalant abuse vary across culture and, as in many

Inhalants are typically "huffed" from a rag soaked in the substance and placed in a plastic bag so the vapors can concentrate. (Drug Enforcement Administration)

other types of drug abuse, the vulnerability to becoming dependent on these substances may depend on present economic well-being and perceptions of the possibility of future well-being. Their ability to act as a reward has been demonstrated in laboratory animals, so there is no doubt that they exert powerful actions on the nervous system. Preventive actions are of two types: education about the adverse effects of solvents on bodily function, and the possible formulation of consumer products with less intrinsic toxicity. Some manufacturers have attempted to minimize the abuse of their products by adulterating them with irritants. Intervention strategies for those habitually using inhalants are not different from those employed for other CNS depressant dependence disorders. Frank withdrawal symptoms are rarely seen with organic solvents. They do, however, accumulate under some conditions of use, and can be associated with prolonged delirium and behavioral disturbances.

ALKANES

Alkanes are hydrocarbons of the general formula $C_nH_{2n}+2;$. The potency of this family of straight-chain chemicals increases with the number of carbons. The smaller molecules (methane, ethane, butane, propane) are gases at room temperature; their deliberate inhalation produces cardiac arrhythmias and sudden death. Pentane, hexane, and longer alkanes are liquids that become progressively less volatile. Hexane produces a devastating neurotoxicity. Alkanes are paraffins; cycloparaffins are rings without alternating double bonds; and alkylcycloparaffins have a short substituent on the ring. Alkylcycloparaffins such as methylcyclopentane and methylcyclohexane (hexahydrotoluene) are convulsants.

AMYL NITRITE

Amyl nitrite is a volatile, oily liquid with a sweet, banana-like odor. It is sold by prescription in glass ampules for the treatment of angina pectoris, chest pain caused by the narrowing of vessels in the heart. When the glass ampules are broken, they "pop"; hence they are sometimes called "poppers." Amyl nitrite relaxes the vessels of the heart by relaxing the muscles of the veins as well as all other smooth muscles in the body. When the veins throughout the body dilate, blood pressure falls. Because a minimum blood pressure is required to maintain blood supply to vital organs such as the brain, a reflex protects the brain by increasing heart rate and blood flow. This produces a "rush" as the heart pounds, and there is a throbbing sensation in the head. Users also experience a warm flush as the blood accumulates near the skin because of the dilation of veins. Vision also may "redden" as the retinal vessels dilate. The user may faint if the heart cannot maintain blood flow to the brain. If this occurs, the user falls to the floor, and blood flows to the brain, restoring consciousness. Use in a situation where it is impossible to become horizontal may result in brain damage.

The duration of action of the drug is very brief, and as the effect wears off, the user may experience headache, nausea, vomiting, and a chill. The drop in body temperature occurs because of the loss of heat when the veins dilate and the skin flushes. Use of the drug for prolonged periods, or swallowing the liquid, may produce fatal methemoglobinemia, a "chocolate" blood condition in which the blood is brown and cannot carry oxygen to the brain. The drug produces a thick, crusty brown rash if it is spilled on the skin, and is irritat-

ing to the lungs. It is flammable and explosive. Volatile nitrites are converted to nitrosamines in the body, and most nitrosamines are very potent cancer-causing chemicals. There is an association of the use of volatile nitrites with Kaposi's sarcoma, an AIDS-related skin cancer. Volatile nitrites impair the function of the immune system. The physiology of sexual intercourse involves smooth muscle; the nitrites relax those muscles as well and so will affect sexual function.

The prescription requirement for amyl nitrite was eliminated in 1960, and its use became popular; in 1964 prescription requirements were reestablished. "Designer" nitrites, such as butyl and isobutyl nitrites, were then bottled and sold as "room deodorizers" with such names as RUSH, Locker Room, and Aroma of Men, so named because it smelled like a locker room. Since these products were not controlled substances or sold as medicines, they were once legal products.

ANESTHETICS

Anesthetics are used in medicine to permit surgical procedures without pain or consciousness. They are of two types: local and general. A local anesthetic is usually injected near nerves to prevent pain in a limited area, such as a Novocaine injection to anesthetize a tooth. General anesthetics are administered to the whole body and depress the CNS to such an extent that major surgery can be performed without killing the patient from the shock resulting from procedures that otherwise would be unendurable. General anesthetics were developed in the mid-nineteenth century by doctors experimenting, usually on themselves, with the organic solvents available at the time. These experiments were sometimes done by groups of people who inhaled the vapors and described the effects, or passed out. Later, careful experimental work identified volatile chemicals that are used to save lives by permitting surgery that would otherwise be impossible to perform, and that are safe to use and have relatively low toxicity.

Some anesthetics can be given by injection. Short-acting anesthetics are used for brief procedures in medicine and dentistry where inhalation anesthesia is inappropriate or difficult, or for starting anesthesia before longer-acting agents are given to the patient. Drugs used for this purpose include barbiturates such as sodium methohexital and sodium thiopental, and benzodiazepines such as midazolam. Fentanyl and related compounds are used for a longer duration of action. A dissociative anesthetic, ketamine, is used for treating burn patients and small children. These agents affect the brain in a more selective way than other anesthetics, so that there is more muscle tone and better circulation in the head and neck. A related veterinary drug, phencyclidine (PCP), has a longer duration of action; when given to humans, however, it has produced terrifying hallucinations upon recovery. It is subject to abuse.

VOLATILE ANESTHETICS

Volatile anesthetics induce unconsciousness and loss of reflexes for surgical procedures. This CNS depression can be induced by a wide variety of different chemicals; those used in clinical medicine are selected for reasons that include low toxicity, ease of maintaining and adjusting a given depth of anesthesia, and freedom from adverse effects upon recovery. Many compounds were examined in the search for modern anesthetic agents.

The depth of anesthesia depends on how much of the medication is present in the CNS. This in turn depends on how much is in the air, to what extent the anesthetic passes between air and blood, and how much passes from blood to brain (or fat, since the brain is largely fat). An agent that is highly insoluble in blood achieves a plateau, or saturation, concentration very rapidly; an example is nitrous oxide. More soluble agents take a longer time to come to plateau, and take a longer time to be exhaled as well, so recovery from them takes longer. Nitrous oxide and cyclopropane have the same solubility in blood, and take the same amount of time to come to a steady concentration in blood; cyclopropane is more soluble in brain and fat, however, so it takes a much lower concentration to achieve the same effect. (Cycloproane is explosive, and therefore is not used in the operating room.) The way an anesthetic functions in a given individual depends on a number of variables, including the amount of fat in the individual's body, the volume of air inspired per minute, the amount of blood pumped through the lungs per minute, and various preexisting medical conditions.

AROMATIC HYDROCARBON SOLVENTS

Aromatic hydrocarbon solvents have a structure that includes a benzene ring. The simplest form is benzene, a six-membered ring with double bonds and six hydrogen atoms. All other aromatic hydrocarbons have alkyl substituents around the ring; for example, toluene has one methyl group and xylene has two methyl groups.

BENZENE

Benzene is a volatile aromatic hydrocarbon (see above). Its presence in consumer products and in the workplace has been reduced because it causes a form of leukemia. Its chemical formula is C_6H_6; it is a six-membered ring with alternating double bonds and a hydrogen on each carbon. The ring opens when metabolized, causing the formation of reactive and toxic chemicals. Benzine, a name applied to automotive fuel in Europe, is a solvent mixture.

BLACK JACK

This is a trade name for several inhalant products that contain either volatile nitrites or ethyl chloride.

CHLORINATED HYDROCARBONS

These substances comprise a large class of industrial chemicals. Those which are highly volatile are sometimes subject to abuse. Chlorinated hydrocarbons undergo significant metabolism in the body, and these changes in chemical structure usually result in an increase of the solvent's toxicity. Because many of these metabolic products are reactive chemicals, they can produce injuries to the kidneys, the liver, and the blood-forming organs. Chlorinated hydrocarbon inhalation is also associated with lethal disorders of heart rhythm, ventricular arrhythmias.

CHLOROFLUOROCARBON PROPELLANTS

Halogenated hydrocarbons are relatively nonreactive chemicals with very high vapor pressure that have been used to blow products out of containers through a tiny hole. Their widespread use in the early 1960s was followed by an epidemic of aerosol sniffing that led to cardiac arrhythmias and death among young people. The halogens—chlorine, fluorine, and bromine—are used to make various chemicals for purposes ranging from propellants and refrigerants to fire extinguishers. Their use has been severely limited since the recognition that their release into the atmosphere depletes the upper layers of ozone, exposing the earth to excessive amounts of ultraviolet radiation. Freon is a brand name for a family of commercial products.

CHLOROFORM

Chloroform, $CHCl_3$, was one of the earliest solvents put to use as an anesthetic agent. It has been replaced with agents that are much less toxic. Its use in cough and cold medications is obsolete. Chloroform was widely abused in the nineteenth century.

ETHYL CHLORIDE

This is a local anesthetic, CNS depressant, and refrigerant that has been subject to abuse by inhalation. Ethyl chloride has a very high vapor pressure, and spraying it directly into the mouth may freeze the tissues of the throat and cause fatal laryngospasm (contraction of the muscles of the throat and larynx), and the shutoff of air to the lungs. Ethyl chloride has been sold in canisters and spray cans (e.g., Black Jack). A related chemical, methyl chloride, has similar effects and was used in refrigerators until it was recognized as highly poisonous in closed spaces.

ETHYL ETHER

A volatile anesthetic agent subject to abuse by inhalation, ethyl ether was used as an inhalation anesthetic for many years. It has been supplanted by other agents with fewer recovery side effects, such as headache, nausea, and vomiting. It is explosive. Ethyl ether was drunk during the Whiskey Rebellion of the eighteenth century, when heavy taxes were imposed on whiskey. Consumed by this route, ether "tanned" (hardened dramatically) the soft palate. When swallowed, profound intoxication follows, but recovery is faster than from alcohol. Alcohol is metabolized at a fixed number of grams per hour, except under extreme conditions; ethyl ether is eliminated by exhalation.

FREON

Freon is a brand name applied to a class of aerosol propellants. See Chlorofluorocarbon Propellants, above.

GASOLINE

Gasoline, a fuel that powers internal combustion engines, is a complex petroleum product that is subject to abuse by inhalation. The toxicity produced from gasoline exposure depends on the constituents of the mixture and the route of administration. Oral ingestion of gasoline is usually followed by vomiting; subsequent aspiration of gasoline liquid into the lungs is followed by a frequently fatal chemical pneumonia. Deliberate inhalation of leaded gasoline fumes can lead to brain injury related to absorption of tetraethyl lead, a very toxic chemical.

GLUE

Glues are made by dissolving a sticky or adhesive material in a solvent. When the solvent evaporates, the adhesive material remains attached to the surfaces to which it is applied, sticking them together. Glues are complex mixtures formulated for specific purposes. They are not designed for human consumption. When inhaled, they may produce severe injury or death. Most of the solvents used in glues are flammable, and fires have resulted from their inappropriate use. The solvent mixtures in glues and glue thinners are designed to dissolve the solid glue material and to evaporate evenly at a rate appropriate for the product. Solvents of relatively low industrial purity are used in these products; they are usually complex mixtures whose formulation changes with market price. Their toxicity can be great when concentrated and inhaled. Some manufacturers label their products or add irritants in an attempt to dissuade youths from deliberately inhaling these products.

HEXANE

Hexane is a volatile solvent that contains six carbons in a straight chain and has the chemical formula C_6H_{14}. It can cause severe damage to the peripheral nervous system, producing death of the long myelinated nerves (distal axonopathy). This condition results in an inability to walk, loss of muscle mass in all limbs, and sometimes loss of bowel and bladder control. This injury occurs because hexane is metabolized to a gamma-diketone. Another solvent subject to abuse that undergoes the same change in the body is methylbutylketone.

NITROUS OXIDE

Nitrous oxide is a volatile analgesic and anesthetic agent. It was discovered at the beginning of the nineteenth century by Sir Humphry Davy, who was looking for gases and vapors that might have some therapeutic use. Nitrous oxide quickly produces an inebriation that many found pleasurable, and it rapidly became the subject of much experimentation and merrymaking. Nitrous oxide parties became very fashionable, but could not long be limited to the upper classes. Popular demonstrations were conducted, and at one such demonstration Horace Wells noticed that a participant had injured his leg, yet seemed oblivious to the pain. Although Davy had noted that nitrous oxide deadened the pain of his toothaches, it was Wells who underwent the first tooth extraction using nitrous oxide for pain relief. The first widespread use of nitrous oxide for clinically significant pain relief was its use in childbirth by S. Klikovich. Nitrous oxide inhalation is about as effective as 30 mg of morphine for pain relief.

Nitrous oxide is not very soluble in either blood or brain tissue, and consequently it has a short duration of action and requires very high levels to produce effects, on the order of 15 to 30 percent by volume. Because the use of gases at this high a concentration might result in asphyxiation, special equipment is used to guard against this possibility in medical settings. Because it displaces oxygen, nitrous oxide frequently kills those who inhale it for pleasure in closed rooms or automobiles.

Nitrous oxide was long thought to be a relatively innocuous anesthetic, almost as safe as inert gases. Recent work has demonstrated, however, that its inhalation irreversibly inactivates methionine synthetase, and this enzyme inhibition produces a vitamin deficiency that can injure the peripheral nervous system. This was first observed in dentists and others with access to nitrous oxide and who inhaled it habitually. This nervous system injury is associated with numbness and clumsiness of the hands, and with Lhermitte's sign, a lightning-like shooting

sensation that occurs when the patient bends the neck.

Nitrous oxide is used in dentistry because it has both analgesic and anxiety-relieving properties. It is used as a carrier gas and inducing agent in major surgery, facilitating induction of anesthesia maintained by other agents. Because it is not very soluble in blood, oxygen must be provided to patients at the end of the surgery, because the nitrous oxide can displace oxygen as it rushes out of the patient's body (diffusion hypoxia).

PERCHLOROETHYLENE

This chlorinated hydrocarbon solvent, used in the dry-cleaning industry, is also known as PERC (see Chlorinated Hydrocarbons, above).

TOLUENE

Toluene (methyl benzene, toluol) is an aromatic hydrocarbon solvent widely used in industrial processes, fuels, and consumer products. It is among the least irritating of the aromatic hydrocarbon solvents. When inhaled, it can produce CNS depression, like alcohol and other solvents. Its pharmacologic effects resemble those of other CNS depressant drugs, displaying actions like those of medications used for the treatment of epilepsy or for the clinical management of anxiety.

Toluene is removed from the body by exhalation and by metabolism. It is metabolized to methylhippuric acid, and is excreted by the kidneys. Overexposure to toluene can produce distal tubular acidosis of the kidney, an injury attributable to excess acidity that is reversible upon termination of exposure. Toluene has been demonstrated to produce loss of high-frequency hearing in laboratory animals following repeated high exposure, such as occurs during solvent abuse. Toluene also has been implicated in severe injuries to the nervous system in a large number of patients who deliberately inhaled toluene-containing solvents. These injuries are characterized by injury and loss of brain tissue. Patients display flattened emotional responses, impaired cognitive abilities, and a wide, shuffling gait associated with injury to the cerebellum. Animal studies have not yet conclusively demonstrated that toluene alone is responsible for this severe brain injury syndrome; nonetheless, solvent abusers who inhale toluene-containing mixtures run a very high risk of irreversible brain injury.

1,1,1 TRICHLOROETHANE (TCE)

This is a chlorinated hydrocarbon solvent with very high vapor pressure. It is useful in products that need to dry quickly, such as liquid paper products used to cover errors. The deliberate inhalation of these products has been associated with sudden death from ventricular arrhythmias (see Chlorinated Hydrocarbons, above).

TRICHLOROETHYLENE

A chlorinated hydrocarbon solvent used as a degreaser and dry-cleaning agent, it is subject to abuse by inhalation. When alcohol is consumed after exposure to trichloroethylene, profound blushing of the face occurs, the "degreaser's flush." One of the metabolites of trichloroethylene is chloral hydrate, an anesthetic agent used in "Mickey Finns," drinks used criminally to anesthetize robbery victims.

WHIPPETS

Whippets are small canisters of nitrous oxide used at soda fountains to make whipped cream. They have been incorporated into various products, such as balloon inflators, "carburetor pipes," and other drug paraphernalia (see Nitrous Oxide, above).

(SEE ALSO: *Complications; Ethnicity and Drugs; High School Senior Survey; Inhalants: Extent of Use and Complications*)

RONALD W. WOOD

INHALANTS: EXTENT OF USE AND COMPLICATIONS About 12 1/2 million ADOLESCENTS in this country say that they have sniffed INHALANTS—usually volatile solvents such as spray paint, glue, or cigarette lighter fluid—at least once in their lives, according to the National Institute on Drug Abuse (NIDA) in its 1997 MONITORING THE FUTURE study, a national survey of 8th-, 10th-, and 12th-grade students (also called the HIGH SCHOOL SENIOR SURVEY). In fact, results from a number of surveys suggest that among children under 18, the

level of use of inhalants is comparable to that of stimulants and is exceeded only by the level of use of MARIJUANA, ALCOHOL, and CIGARETTES.

The abuse of inhalants, which include a broad array of cheap and easily obtainable household products, is frequently viewed by the public as a relatively harmless habit and not in the same high-risk category as drugs such as alcohol, COCAINE, and HEROIN. Some people tend to view inhalant "sniffing," "snorting," "bagging" (when fumes are inhaled from a plastic bag), or "huffing" (when an inhalant-soaked rag is stuffed in the mouth) as a kind of childish fad to be equated with youthful experiments with cigarettes. But inhalant abuse is deadly serious. Sniffing volatile solvents, which include most inhalants, can cause severe damage to the brain and nervous system. By starving the body of oxygen or forcing the heart to beat more rapidly and erratically, inhalants have killed sniffers, most of whom are adolescents.

The difficulty people face in recognizing the scope and magnitude of the problem lies in the dearth of documenting information. Survey data on the prevalence of inhalant abuse are difficult to obtain for a number of reasons—and what information does exist may underemphasize the severity of the situation. No one knows how many adolescents and young people die each year from inhalant abuse, in part because medical examiners often attribute deaths from inhalant abuse to heart problems, suffocation, SUICIDE, or ACCIDENTS. What is more, no national system exists for gathering data on the extent of inhalant-related injuries, although medical journals have described the situation as serious. As serious as the situation may be, some researchers warn that doctors and emergency medical personnel are not adequately trained to recognize and report symptoms of inhalant abuse.

SCOPE OF THE PROBLEM

Inhalant abuse came to public attention in the 1950s when the news media reported that young people who were seeking a cheap "high" were sniffing glue. The term *glue sniffing* is still widely used, often to include inhalation of a broad range of common products besides glue.

With so many substances lumped together as inhalants, research data describing frequency and trends of inhalant abuse are uneven and sometimes contradictory. However, evidence indicates that inhalant abuse is far more common among all socioeconomic levels of U.S. youth than is typically recognized by parents and the public. For example, the National Institute on Drug Abuse's (NIDA's) Monitoring the Future survey shows that in 1997, 21.0 percent of 8th graders had used an inhalant in his or her lifetime.

Inhalants were used by equally high percentages of 10th and 12th graders, according to the NIDA survey. Lifetime inhalant use among 12th graders, which had increased steadily for most of the 1980s, leveled off somewhat at 16.1 percent in 1997; 10th graders also reported a lifetime inhalant use of 18.3 percent.

Inhalants are most commonly used by adolescents in their early teens, with usage dropping off as students grow older, unlike the case for other drugs. For example, while 5.6 percent of 8th graders reported using inhalants within the past 30 days, known as "current" use, only 2.5 percent of seniors reported current use of inhalants.

One major roadblock to recognizing the size of the inhalant problem is the ready availability of products that are inhaled. Inhalants are cheap, or even free, and can be purchased legally in retail stores in a variety of seemingly harmless products. As a result, adolescents who sniff inhalants to get high do not face the drug procurement obstacles that confront abusers of other drugs. Youthful inhalant abusers can easily buy airplane glue, hair spray, spray paint, cigarette lighter fluid, nail polish remover, or typing correction fluid.

DANGERS OF INHALANT ABUSE

Despite the dangers associated with inhalant abuse, no central system exists in the United States for reporting deaths and injuries from abusing inhalants. A study by Dr. James C. Garriott, the chief toxicologist in San Antonio and Bexar County, Texas, examined all deaths in the county between 1982 and 1988 that were attributed to inhalant abuse. Most of the thirty-nine inhalant-related deaths involved teenagers, with twenty-one deaths occurring among people less than twenty years old. Deaths of males outnumbered those of females thirty-four to five. Many of the abusers met with a violent death possibly related to but not directly caused by the use of volatile solvents. Eleven deaths were caused by suicide (ten by hanging), nine by

homicide, and ten by accident, including falls, auto accidents, and overdoses.

Most of those people who died in Bexar County had used toluene-containing products, such as spray paints and lacquers, Dr. Garriott reported. The next most frequent cause of death in the Texas study was the use of a combination of chemicals found in typewriter correction fluids and other solvents. Other abused substances that resulted in death included gasoline, nitrous oxide, and refrigerants, such as fluorocarbons (Freon). Freon now has been replaced with butane or propane products in most aerosols.

As reported in the Texas study, the solvent toluene is identified frequently in inhalant-abuse deaths and injuries because it is a common component of many paints, lacquers, glues, inks, and cleaning fluids. A 1986 study of twenty chronic abusers of toluene-containing spray paints found that after one month of abstinence from sniffing the paint, 65 percent of the abusers had damage to the nervous system. Such damage can lead to impaired perception, reasoning, and memory, as well as defective muscular coordination and, eventually, dementia.

In England, where national statistics on inhalant deaths are recorded, the largest number of deaths in 1991 resulted from exposure to butane and propane, which are used as fuels or propellants. Many researchers believe that abuse of butane, which is readily available in cigarette lighters, is on the increase in the United States.

A recent report of this particular inhalant problem in the Cincinnati region indicates that butane gas is the cause of enough deaths to foster national concern about the abuse of fuel gases, whether or not it is a passing form of inhalant abuse. Sniffers seem to go out of their way to get their favorite product; in certain parts of the country, Texas 'shoeshine'—a shoe-shining spray containing toluene—and silver or gold spray paints are local or current favorites.

Since the banishing of fluorocarbons, the most common sniffing death hazards among U.S. students probably are due to butane and propane. Doctors and emergency room staffs need to be aware that the profile of the teenager who inhales volatile solvents is not limited to ethnic lower socioeconomic classes. Many sources lead us to believe that abuse of these readily available inhalants has reached epidemic proportions, indicating an urgent need for preventive efforts.

WHO ABUSES INHALANTS?

One possible reason for the increased use of volatile solvents is that more girls are joining boys in sniffing solvents. Studies in New York State and Texas report that males are using solvents at only slightly higher rates than females. Among Native Americans, whose solvent-abuse rates are the highest of any ethnic group, lifetime prevalence rates for males and females were nearly identical, according to 1991 NIDA data.

There is a public perception that inhalant abuse is more common among HISPANIC youth than among other ethnic groups. However, surveys have not found high rates of abuse by Hispanics in all geographic areas. Rates for Hispanics may be related to socioeconomic conditions. Hispanic youths in poor environments may use solvents heavily, but the usage rates in less stressful environments are lower.

In fact, inhalant abuse shows an episodic pattern, with short-term abuse outbreaks developing in a particular school or region as a specific inhalant practice or product becomes popular in a fashion typical of teenage fads. This episodic pattern can be reflected in survey results and can overstate the magnitude of a continually fluctuating level of abuse.

Inhalant abusers typically use other drugs as well. Children as young as fourth graders who use volatile solvents will also start experimenting with other drugs—usually alcohol and marijuana. Adolescent solvent abusers are POLYDRUG users prone to use whatever is available, although they show a preference for solvents. Solvent abuse is held in low regard by older adolescents, who consider it unsophisticated, a childish habit.

It is not just juveniles who are abusing inhalants. Reports in the mid-1990s indicate that college-age and older adults are the primary abusers of butane and nitrous oxide.

(SEE ALSO: *Poverty and Drug Use*)

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NEIL SWAN
REVISED BY DONNA GRAFT

INJECTING DRUG USERS AND HIV

One of the major risk behaviors for infection by the HUMAN IMMUNODEFICIENCY VIRUS (HIV) is injecting drug use; the others are unprotected male homosexual sex (Centers for Disease Control, 1991a) and unprotected heterosexual sex with an HIV-infected partner. The NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) estimated that there were between 1.1 and 1.3 million injecting drug users (IDUs) in the United States in the late 1980s (Centers for Disease Control, 1987). Although the number of IDUs increased between 1990 and 1997, participation in needle exchange programs also increased, as did participation in HIV testing and counseling (Des Jarlais et al., 2000).

In 1990, 30 percent of ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) cases were heterosexual injecting drug users; in addition, 30 to 50 percent of new cases identified were related to IDU (Iguchi et al., 1990). Injecting drug use was also related to most instances of heterosexual transmission of the virus (Centers for Disease Control, 1992). Also, whether directly or indirectly, injecting drug use accounted for 70 percent of AIDS cases among women and children (Centers for Disease Control, 1989). In these cases, either the woman or her sex partner was an IDU (Gayle, Selic, & Chu, 1990). Since 1995 Belarus, Moldova, Kazakhstan, Russia, and Ukraine have seen rapid HIV increases, with at least 50 percent among IDUs (Henderson, 2000a).

The transmission of HIV among IDUs occurs directly through blood transmission of the virus, as when drug users share used, nonsterilized hypodermic needles and syringes, cotton, cookers, rags, and water that has been contaminated with the infected blood of other users. It is also transmitted when bodily fluids (e.g., semen, saliva, blood) are exchanged during sexual acts. The virus can also be transmitted to a fetus by a pregnant, HIV-positive woman. However, the risk of transmission to the fetus can be sharply reduced if the HIV-positive woman takes the antiviral drug AZT during pregnancy. Various studies have found that prior to the HIV epidemic, between 70 and 100 percent of IDUs shared injection paraphernalia (Lange et al., 1988; Des Jarlais et al., 1988). These percentages have been decreasing, since the connection with AIDS has been widely publicized since the 1980s. Still, dirty syringes cause 80,000 to 160,000 HIV infections worldwide annually (Henderson, 2000b).

Historically the most commonly injected drug has been HEROIN; however, the increased availability of COCAINE has resulted in an increased use by IDUs since the late 1980s. Injecting cocaine has elevated the risk of HIV spread because the shorter duration of a cocaine "high" leads to more frequent injecting (Gottlieb & Hutman, 1990). It has also been reported that cocaine injectors, when the number of injections was statistically controlled, were at higher risk than other drug-injecting populations for HIV because cocaine use is associated with increased unprotected sexual activity (Chaisson et al., 1989).

BACKGROUND

The prevalence of HIV/AIDS among injectors varies widely from region to region in the United States. The highest rates of IDU and HIV are found along the east coast and west coast, in the southwest, Florida, Puerto Rico, and in major metropolitan areas. Overall, of the 48,269 new cases of HIV reported in 1998, more than 50 percent were IDU-associated (Centers for Disease Control and Prevention, 1999). The prevalence of HIV infection is also related to the social context of needle sharing. In areas where injectors go to "shooting galleries"—where anyone using a previously used needle may not know who else used the needle—there are generally high rates of HIV infection. Conversely, in areas where the IDU social network is well known and only a limited number share works with one another, the infection rate is lower (Leukefeld et al., 1991).

While IDUs with HIV infection are predominantly males of color (Hispanics and African-Americans) in their late twenties and early thirties,

variations and exceptions are noted and reflect dynamics in individual metropolitan areas. In 1989, the highest prevalence of IDUs in drug treatment centers who tested positive for HIV were in the Middle Atlantic states (New York, New Jersey, and Pennsylvania), where the overall rate for HIV-positive intravenous drug using men and women in treatment was 44 percent (Centers for Disease Control, 1990b).

REDUCING RISK-TAKING BEHAVIOR

Drug-abuse treatment and prevention can be effective in controlling the spread of AIDS among IDUs and for reducing the risk of exposure to the HIV virus. The goals of drug treatment and prevention are different. The goal of treatment is to eliminate injecting drug use as a risk factor in the spread of HIV. The goal of prevention is to reduce and eliminate harmful behaviors, like sharing needles, that place the IDU at risk for either becoming infected or infecting others with HIV. Prevention does not necessarily focus on changing drug-seeking and needle-using behavior. Four areas are considered to be of prime importance: (1) increasing the number of drug abusers in treatment, (2) enhancing the effect of treatment, (3) developing outreach and counseling strategies, and (4) developing prevention strategies for reducing the risk-taking behavior among IDUs.

Drug Treatment. Several organizations and groups have suggested that drug-abuse treatment is important in helping to decrease and prevent the spread of AIDS. These ogranizations include the World Health Organization (WHO), the American Medical Association (AMA), the National Academy of Sciences/Institute of Medicine and the Presidential Commission on the HIV Epidemic.

Drug-abuse treatment can play an important role in preventing HIV transmission. Treatment reduces the number of people engaging in risky behavior. In addition to reducing the number of active drug addicts, treatment can also reduce the number of people out recruiting new drug addicts (Brown, 1991). Barriers to treatment now exist for IDUs with HIV. IDUs themselves avoid people they suspect have HIV or AIDS, and some treatment programs will not allow HIV-infected people to participate in their programs (Brown, 1991). But the most serious barrier to drug-abuse treatment is the lack of treatment availability and programs. More specifically, some IDUs, including those known to be HIV infected, are not admitted into drug treatment for as long as six months due to a lack of available openings in treatment programs (Gotlieb & Hutman, 1990). In some community-outreach programs designed specifically to target IDUs to prevent HIV transmission, the majority of IDUs contacted have never been in treatment (Schrager et al., 1991). There is evidence that drug-abuse treatment reduces needle sharing by eliminating or reducing needle using behaviors.

Drug-abuse treatment incorporates several modalities (approaches), which include: (1) drug-free outpatient services, (2) METHADONE MAINTENANCE PROGRAMS, and (3) therapeutic communities (Leukefeld, 1988), as well as a number of programs that do not fit into these categories. Ideally, HIV and drug treatment should be integrated to increase social supports, which should increase adherence to medication schedules and resistance to drugs (Stein et al., 2000).

Outreach and Counseling. One way to increase the number of IDUs in treatment is to increase the number of outreach and counseling programs. The National AIDS Drug Abuse Research Demonstration Program is an example of outreach and counseling (National Institute on Drug Abuse, 1988). This demonstration program, initiated in 1987, provided an opportunity to assess the characteristics and risk-taking behaviors of injecting drug abusers not in treatment. Additional purposes included focusing on sexual partners of IDUs at high risk for AIDS, determining and monitoring HIV seroprevalence (rate a given population tests positive) across cities, and evaluating prevention strategies. The overall goal was to reduce the spread of HIV infection by reducing and eliminating drug-use practices and certain high-risk sexual practices. Counseling and outreach approaches were applied, tested, and evaluated at each community site. Projects were targeted on three levels: (1) high-risk individuals, (2) family and social networks of IDUs, and (3) the larger community. Although intervention components varied across sites, the focus and objectives were similar (Chitwood et al., 1991; Leukefeld, 1988). These projects provided information about protective behaviors, and IDUs were encouraged to enroll in drug-abuse treatment programs. Trained indigenous outreach workers distributed and discussed materials using informal groups or through one-on-

one interactions. Sixty-three communities were involved in this demonstration project (McCoy & Khoury, 1990; Leukefeld, 1988).

Strategies for community outreach differ between the IDU, their sex partners, and prostitutes. Reaching the IDU means that outreach workers go to places where IDUs hang out and buy their drugs, as well as going into criminal-justice settings (jails, PRISONS, courts), drug-treatment centers, and the health-care system. Although there is inherent danger in many of these settings, recovering drug users—savvy men and women of the same backgrounds as IDUs—have achieved success in contacting IDUs in these settings (Serrano, 1990; Brown, 1990).

Many male IDUs hang out on the street or can be found in places where other IDUs hang out. However, female sex partners of IDUs frequently stay close to home with children and they frequently work (Margolis, 1991). While women may purchase drugs for their partners, they do not generally hang out at those locations. Thus, targeting female partners of IDUs requires different strategies than those used for contacting the IDU. The YES project of San Francisco is an example of a program targeted toward female sex partners of IDUs. It began by supporting high-risk women in meeting their basic needs by helping them get general assistance, food, clothing, and health care. A second strategy was to rent a hotel room, called "A Room of Her Own" in which education, counseling, and service could be provided to the female partner of the IDU. Another project (serving Bridgeport, Connecticut, San Juan, Puerto Rico, and Juarez, Mexico) contacted the female sex partners of male IDUs; it examined an approach that attracted women to a safe setting established by the program—a clothing boutique where women could pick up new clothes and then stay for an AIDS information video. Another strategy as part of this project was to have outreach staff available in the afternoons and evenings, hours when the women were available (Moini, 1991). In another project in Long Beach, California, a drop-in center was established for youth and women (Yankovich, Archuleta, & Simental, 1991).

Prostitutes, another high risk group, require strategies appropriate to their setting. Contacting prostitutes can be difficult, since their pimps can severely restrict contact with social-service workers. In one study, contact was made when the pimp was not around and through the Salvation Army mobile canteen that served coffee to prostitutes in the late night/early morning hours (Moini, 1991). Another study reported that prostitutes are aware of AIDS, know how it is transmitted, and are aware that their drug use and unsafe sexual behavior are putting them at risk (Shedlin, 1990). However, barriers to behavioral changes in prostitutes include low self-esteem and low levels of education, along with POVERTY, addiction, hopelessness, lack of knowledge, and lack of support services.

Prevention Strategies. Prevention is of central importance in controlling the spread of HIV among IDUs. Abstinence from drug use and needle use is the overall approach for preventing the spread of HIV. Preventing infection is a self-preservation issue (protecting self), while preventing the spread of HIV is an altruistic issue (protecting others) (Moini, 1991). It has been reported that among IDUs there is greater resistance to changing sexual behaviors (using condoms) than drug-use behaviors (sharing needles) (Sorenson, 1990). Thus, it is important to target not only IDUs but also their sex partners and prostitutes who engage in unsafe sex practices. These people may also be exchanging drugs for sex and may be IDUs themselves (Centers for Disease Control, 1991b). Three prevention strategies have been developed: education, NEEDLE-EXCHANGE PROGRAMS, and community-based interventions.

Education. In addition to the community-outreach programs, three overarching prevention-education strategies have been developed: (1) prevention education for HIV-antibody-negative individuals, (2) AIDS pre- and post-test counseling, and (3) prevention and support for HIV-antibody-positive individuals (Schensul & Weeks, 1991). AIDS prevention education involves delivery of information related to HIV spread, risk behaviors, and preventing the spread of the virus. Educational activities have been targeted on the general public, school-aged populations, and populations at risk, like IDUs. The U.S. Centers for Disease Control (CDC) National Public Information Campaign has produced numerous educational materials for the radio, television, and print media. Education targeted to individuals at risk for HIV infection has included counseling, testing, the teaching of behavioral responses to risky behaviors, and providing support for low or no-risk behaviors (Roper, 1991).

Prevention education for IDUs includes several informational components. Of primary importance to active drug users are issues related to needle sharing as a risk behavior for HIV transmission. Also of critical importance to needle-sharing IDUs in preventing HIV transmission are describing ways to effectively sterilize shared paraphernalia. Of importance to IDUs, the sex partners of IDUs, and prostitutes are safe-sex issues and knowledge of HIV transmission through unsafe sex. Of importance to potential partners—both men and women who have relationships with IDUs and who may be IDUs—is knowledge about the transmission of the virus from mother to fetus (Strawn, 1991). Early to mid-1990s research indicates that the use of AZT (an anti-HIV drug) by pregnant women who are HIV-positive sharply reduces the probability that the baby will be infected with the virus.

Pre- and post-test AIDS counseling is another strategy for HIV prevention. In the early 1980s, at the beginning of the AIDS epidemic, testing was controversial because of the fear of discrimination, concern about the accuracy of tests, the usefulness of the results, and the psychological distress associated with a positive result. However, with more effective treatment for symptomatic AIDS and early treatment for HIV-infected individuals, the resistance is diminishing (Strawn, 1991).

Generally, individuals seek HIV testing for one of two reasons: (1) an agency or person, (like a plasma center, a penal institution, or a medical professional) requests it, or (2) the individual seeks to be tested because of identified high-risk behaviors (Roggenburg et al., 1991). HIV testing can represent a crisis in the life of an individual being tested. Receiving the results can be difficult due to the anxiety of the situation, even if the results are negative. Pre- and posttest counseling is necessary to assess the psychological well-being of the individual being tested. Some people believe that being informed of a positive test result can make some people suicidal (Strawn, 1991).

A controversial prevention approach in the United States for preventing HIV infection is the provision of clean needles to IDUs. In needle-exchange programs, a clean needle and sometimes injection equipment (works) are exchanged for used ones. Proponents of these programs argue that needle exchanges help prevent HIV transmission and offer opportunities for education and referral to drug-treatment programs. It has been reported that in areas where needle-exchange programs have been in operation, the incidence of sharing used needles has diminished (Karpen, 1990). Some needle-exchange programs are conducted illegally by AIDS activists (Karpen, 1990). Occasionally, in the United States, needle exchanges are managed legally by health departments. To conduct a needle-exchange program legally, in many regions the PARAPHERNALIA LAWS related to drug-use equipment would need to be modified (Wood, 1990).

Opponents of needle-exchange programs point out that needles and syringes are only two of the many drug-use implements that can be contaminated with blood and transmit HIV. For example, cotton, cookers, and the water used to rinse out syringes can transmit HIV if they have been contaminated with infected blood. In addition, some injecting rituals can transmit HIV even if a clean needle and syringe are used. Sharing an injection can be part of a ritual between addicts. For example, in a "rinse" or a "geezer" one addict injects another person and then injects him- or herself with the remnant in the syringe (Primm, 1990). Few rigorous U.S. studies have examined needle-exchange programs and their effects on HIV transmission. One group of researchers interviewed IDUs participating in needle-exchange programs to help determine needs for prevention programs (Des Jarlais, 1999). Although some areas showed low rates of HIV, others showed no marked decrease in cases. The researchers believed that more complete reporting of risk behavior was necessary.

As above, one component of the National AIDS Demonstration Project has been to compare the CDC basic outreach and counseling intervention with an enhanced intervention. The CDC basic intervention includes factual information about AIDS transmission, prevention, and self-assessed risk. Enhanced community-based educational-intervention programs have involved several strategies: counseling individuals, couples, and groups; developing behavioral skills; and using applied ethnography with outreach workers to disseminate information (Chitwood et al., 1991). Using these strategies helped the rate of sharing between IDUs to decrease by up to 59 percent in a five-city study. In the same study, IDU condom use increased by up to 16 percent (Iguchi et al., 1990).

CONCLUSION

Preventing the spread of AIDS for IDUs and their sex partners requires a multidisciplinary, multiple-strategy approach. Community-intervention strategies have proven to be partially effective in reducing IDU risk behaviors (Leukefeld, Battjes, & Amsel, 1990). Much remains to be accomplished, however. Targeting HIV-prevention approaches and interventions will receive additional emphasis as the epidemic progresses (Leukefeld & Battjes, 1991). Research needs to continue to examine methods to reduce HIV in IDUs, to reinforce IDU behavior changes, to increase the effectiveness of drug-abuse treatment, and to provide psychosocial and other supports focused on HIV-infected IDUs.

(SEE ALSO: *Complications; Heroin: The British System; Prevention; Substance Abuse and AIDS; Vulnerability as Cause of Substance Abuse*)

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INSTITUTE ON BLACK CHEMICAL ABUSE (IBCA) *See* Treatment Programs/Centers/Organizations: An Historical Perspective

INTERNATIONAL CLASSIFICATION OF DISEASES (ICD) This is the official classification system of the World Health Organization (WHO). As a general system for the classification of diseases, injuries, causes of death, and related health problems, the ICD is used throughout the world as a common frame of reference for statistical reporting, clinical practice, and education. The ICD is a system of categories to which specific disease entities can be assigned consistently in different parts of the world. Recognizing the growing importance of alcohol and drug misuse, the ninth revision of ICD was published in 1975 (ICD-9), and it introduced the terms *dependence* and *abuse* into the international nomenclature. *Drug dependence* was defined as "a state, psychic and sometimes also physical, resulting from taking a drug, and characterized by behavioural and other responses that always include a compulsion to take the drug on a continuous or periodic basis in order to experience its psychic effects, and sometimes to avoid the discomfort of its absence" (WHO, 1977, 198). *Alcohol dependence* was defined in a similar way. The category Non-Dependent Abuse of Drugs was designed for cases where a person "has come under medical care because of the maladaptive effect of a drug on which he is not dependent and that he has taken on his own initiative to the detriment of his health or social functioning" (WHO, 1978, 43–44).

In 1993, the tenth revision, ICD-10, was introduced—replacing ICD-9 as the official classification system for international use (WHO, 1992a). Chapter 5, which describes mental and behavioral conditions (WHO, 1992b), includes a section for the classification of disorders based on ten kinds of PSYCHOACTIVE substances: ALCOHOL, SEDATIVE-HYPNOTICS, CANNABIS (MARIJUANA), COCAINE, other STIMULANTS, OPIOIDS, HALLUCINOGENS, TOBACCO, VOLATILE SOLVENTS, and multiple drugs. The major disorders associated with these substances are acute intoxication, harmful use, dependence syndrome, withdrawal state, amnesic syndrome, and psychotic disorders (WHO, 1992b). The identification of the substance used may be made on the basis of an interview with the patient, laboratory analysis of blood or urine specimens, or other evidence (such as clinical signs and symptoms or reports from third parties).

Acute intoxication is a transient condition following the ingestion of alcohol or other psychoactive substances. It results in disturbances in consciousness, cognition, perception, mood, or behavior. According to ICD-10, psychoactive substances are capable of producing different types of effect at different dose levels. For example, alcohol may have stimulant effects at low doses, lead to agitation and aggression with increasing dose levels, and produce clear sedation at very high levels. The term *pathological intoxication* in ICD-10 refers to the sudden onset of violent behavior that is not typical of the individual when sober. This occurs very soon after amounts of alcohol are drunk that would not produce intoxication in most people.

A central feature of the ICD-10 approach to substance-use disorders is the concept of a dependence syndrome, which is distinguished from disabilities caused by harmful substance use (Edwards, Arif, & Hodgson, 1981). The *dependence syndrome* is defined as "a cluster of physiological, behavioural, and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviours that once had greater value" (WHO, 1992b, 75). A central characteristic of the dependence syndrome is the strong and persistent desire to take psychoactive drugs, alcohol, or tobacco. Another feature is the rapid reappearance of the syndrome soon after alcohol or drug use is resumed after a period of abstinence. A definite diagnosis of dependence is made only if three or more of the following have been experienced during the previous year: (1) a strong desire or sense of compulsion to take the substance; (2) difficulties in controlling substance-taking behavior in terms of its onset, termination, or levels of use; (3) a physiological withdrawal state; (4) evidence of tolerance; (5) progressive neglect of alternative pleasures or interests because of substance use; and (6) persisting with substance use despite clear evidence of overtly harmful consequences.

Harmful use, a new term introduced in ICD-10, is a pattern of using one or more psychoactive substances that causes damage to health. The damage may be: (1) physical (physiological), such as fatty liver, injuries associated with alcohol intoxication, or hepatitis from needle-injected drugs; or

(2) mental (psychological), such as depression related to heavy drinking or drug use. Adverse social consequences often accompany substance use, but they are not in themselves sufficient to result in a diagnosis of harmful use.

Chapter 5 of ICD-10 is available in several different versions. The *Clinical Descriptions and Diagnostic Guidelines* is intended for general clinical, educational, and service use. *Diagnostic Criteria for Research* is designed for use in scientific investigations and epidemiological studies. A shorter and simpler version of the classification is available for use by primary health-care workers.

(SEE ALSO: *Addiction: Concepts and Definitions; Alcoholism: Origin of the Term; Diagnostic and Statistical Manual [DSM]; Disease Concept of Alcoholism and Drug Abuse*)

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INTERNATIONAL DRUG CONTROL *See* Anslinger, Harry J., and U.S. Drug Policy; Psychotropic Substances Convention of 1971; Single Convention on Narcotic Drugs

INTERNATIONAL DRUG SUPPLY SYSTEMS The majority of illicit drugs consumed in the United States are of foreign origin—including all the COCAINE and HEROIN and significant amounts of MARIJUANA. In the early 1990s, the U.S. National Narcotics Intelligence Consumer Committee (NNICC) report estimates that Latin American countries supplied approximately 25 to 30 percent of the heroin, perhaps 60 to 80 percent of the marijuana, and all the cocaine. Southeast Asian and Middle Eastern countries supplied the remaining 70 to 75 percent of the heroin.

Drug use and drug abuse have been a part of many cultures for centuries. Although once considered a problem only for countries with massive demand and consequent loss of labor and life, drugs are now recognized as a policy concern for all countries involved—the producing, TRANSIT, and consuming countries alike. No country is insulated from the destabilizing forces of illicit drugs. For SOURCE (producing) countries, drug trafficking appears to provide short-term economic benefits, but mainly for those involved in the business. Eventually, long-term negative economic consequences ensue, with foreign investment, tourism, and domestic production diminished—and with off-shore money laundering and the concentration of wealth in the hands of a few. The drug trade does not stimulate regional economies through jobs, capital appreciation, and investment.

Since 1971, when modern international drug-control efforts began, a number of major shifts have occurred in the drug-producing capabilities of various countries. For example, in the early 1970s, after the so-called French Connection was broken (Turkish OPIUM was processed into heroin in France), MEXICO replaced Turkey as a major source of U.S. heroin; Pakistan then supplanted Mexico after 1979, when the Islamic political revolution in Iran created a population of refugees. At about the same time, the Soviet Union occupied Afghanistan, and the resistance movements there increased their income-generating opium cultivation practices.

In the 1980s, cocaine production in the Andean countries of Peru, BOLIVIA, and COLOMBIA expanded significantly into nontraditional growing zones (the Bolivian Chapare region and Peruvian Upper Huallaga Valley, or UHV), augmenting the more traditional licit production areas of the Bolivian Yungas and Peruvian Cuzco regions. In the early 1980s, U.S. demand for Mexican marijuana decreased dramatically, because of consumer con-

TABLE 1
Cocaine Production Estimates

U.S. MEASURE

Source Country		*Net Coca Cultivation (acres)*	*Estimated Coca Leaf Yield (tons)*	*Potential Cocaine HCl Capacity (tons)*
Peru	1990	299,611	216,590	627–671
	1991	298,376	244,970	710–759
Bolivia	1990	124,241	84,480	270–457
	1991	118,313	86,240	275–462
Colombia	1990	99,047	33,310	72
	1991	92,625	33,000	66

Potential
Cocaine HCl Production
1990 = 969–1,199 tons 1991 = 1,051–1,287

METRIC MEASURE

Source Country		*Net Coca Cultivation (hectares)*	*Estimated Coca Leaf Yield (metric tons)*	*Potential Cocaine HCl Capacity (metric tons)*
Peru	1990	121,300	196,900	570–610
	1991	120,800	222,700	645–690
Bolivia	1990	50,300	76,800	245–415
	1991	47,900	78,400	250–430
Colombia	1990	40,100	32,100	65
	1991	37,500	30,000	60

Potential
Cocaine HCl Production
1990 = 880–1,090 metric tons 1991 = 955–1,170 metric tons

NOTE: The figures reflected here are consistent with the *International Narcotics Control Strategy Report (INCSR) 1992.* The INCSR states cultivation in hectares and yields in metric tons. All figures have been converted to acres or short tons, as appropriate, in the chart on the left. A new procedure introduced in the 1991 INCSR is used for calculating coca leaf production. Previous methods did not deduct immature, non-producing fields from net cultivation before calculating production. Multiple harvests of coca do not begin until plants are at least two years old. Here, only mature cultivation was used to calculate production. Estimates included here for 1990 have been revised to reflect the use of the mature cultivation methodology cited. These figures do appear in the NNICC Report 1990 charts in parenthesis.
According to past UHV Reduction Agency (CORAH) and U.S. Agency for International Development (USAID) reports, the dry leaf yield of mature coca in the UHV ranges between 2.0 and 2.7 metric tons per hectare. A mean yield factor of 2.3 metric tons was used for this area. Other areas of Peru have lower yields similar to the Yungas in Bolivia. Last year's reported yield of 1.14 metric tons was for areas of Peru outside the UHV. According to the Bolivia's Coca Eradication Directorate (DIRECO), mature coca leaf yield averages 2.7 metric tons in the Chapare and 1.0 metric tons in the Yungas. The conversion rate for calculating potential cocaine production from dry leaf is 322–345:1 in Peru and 195–330:1 in Bolivia. Production in Colombia is determined by multiplying a yield factor of .8 metric tons per hectare by net cultivation. All Colombian cultivation was assumed to be mature. The conversion rate for Colombia is 500:1.

SOURCE: *International Narcotics Control Strategy Report 1992.*

cern about Mexico's drug-elimination program, where marijuana was sprayed with the herbicide paraquat, some of which is reported to have killed U.S. users. Consequently, Colombia replaced Mexico as the preferred source of high quality marijuana. Colombia and Guatemala also began to cultivate substantial amounts of opium in the early 1990s.

Traffickers have also adjusted their smuggling routes in response to government law-enforcement pressures. For example, in the mid-to-late 1980s, Colombian drug traffickers began to shift their routes away from the Florida peninsula and toward Central America and Mexico. By the early 1990s, the U.S. government estimated that up to 50 percent of the Colombian cocaine consumed in the United States entered via Mexico. Wide variations in source-country response to these shifts in production have also been chronicled, ranging from government complicity and corruption to modest attempts to reduce crop production and trafficking to intensified organized efforts to eliminate or hamper seriously the drug trade.

PRINCIPAL DRUG-PRODUCING COUNTRIES

Coca/Cocaine. As of the early 1990s, all the cocaine, about 30 percent of the heroin, and a significant amount of marijuana entering the United States is produced in the Western Hemisphere—in Mexico, Central, and South America. They are smuggled in through the southern borders of the United States. All of the cocaine consumed in

TABLE 2
Illicit Opium Estimates

CULTIVATION			
Major Source Country		*Net Cultivation (acres)*	*Net Cultivation (hectares)*
Burma	1990	370,747	150,100
	1991	395,200	160,000
Thailand	1990	8,484	3,435
	1991	7,410	3,000
Laos	1990	75,533	30,580
	1991	73,174	29,625
Mexico	1990	13,482	5,450
	1991	9,300	3,765
Guatemala	1990	2,087	845
	1991	2,828	1,145
Colombia	1990	Unknown	Unknown
	1991	2,865	1,160
Afghanistan	1990	30,566	12,375
	1991	42,459	17,190
Pakistan	1990	20,266	8,205
	1991	19,834	8,030
Lebanon	1990	7,904	3,200
	1991	8,398	3,400
Iran	1990	Unknown	Unknown
	1991	Unknown	Unknown

PRODUCTION			
Major Source Country		*Production (tons)*	*Production (metric tons)*
Burma	1990	2,475	2,250
	1991	2,585	2,350
Thailand	1990	44	40
	1991	39	35
Laos	1990	303	275
	1991	292	265
Mexico	1990	68	62
	1991	45	41
Guatemala	1990	14	13
	1991	19	17
Colombia	1990	Unknown	Unknown
	1991	30	27
Afghanistan	1990	457	415*
	1991	627	570**
Pakistan	1990	182	165
	1991	198	180
Lebanon	1990	35	32
	1991	37	34
Iran	1990	330	300
	1991	330	300
Total:	1990 = 4,202 tons (3,819 metric tons)		
	1991 = 3,908 tons (3,552 metric tons)		

NOTE: Opium generally converts to heroin hydrochloride at ratio of 10:1. Point estimates cited above reflect a mathematical mean point estimate and are not intended to imply a degree of accuracy or certitude which cannot be obtained due to the nature of illicit drug cultivation and production.

*The U.S. Drug Enforcement Agency believes that multi-cropping and the use of fertilizers in Afghanistan renders the 1990 estimate of 457 tons (415 metric tons) to the lower end of a potentially higher production range in Afghanistan.

**The U.S. Drug Enforcement Agency believes that higher yields may exist in Afghanistan and that production could potentially be above 990 tons (900 metric tons.)

SOURCE: *International Narcotics Control Strategy Report 1992.*

the world is grown and processed in the Andean countries of Peru, Bolivia, Ecuador, and Colombia. Some 60 percent of COCA PLANTS (*Eryroxylon coca*) are cultivated in Peru, about 15 percent in Colombia, and about 25 percent in Bolivia.

Peru. Traditional legal cultivation of coca is licensed for cultivation in Cuzco, Peru, but the majority of Peru's illicit crop comes from the Upper Huallaga Valley (UHV), which includes portions of Huanuco, San Martin, and Ucayali departments. Other illicit cultivation occurs in La Convencion and Lares valleys in Cuzco and in the Ayachucho department. Much of the coca leaf is processed into COCA PASTE and cocaine base in crude maceration (steeping) pits positioned near cultivation sites. Clandestine labs then process the paste into base. Normally the base is then shipped to hydrochloride (HCl) laboratories in Colombia, although cocaine HCl production in Peru is rising. Reportedly, traffickers have been moving their laboratories from isolated jungle sites nearer to towns, where corrupt officials can offer protection. The chemicals (kerosene, lime, ether, acetone, hydrochloride acid) needed to process coca leaves into paste, base, and hydrochloride are diverted from legitimate chemical shipments that reach Peru by sea.

Although the Colombian traffickers control most of the cultivation and the processing of coca into

paste and base in Peru, some 20 Peruvian trafficking organizations have also been identified. By early 1991, self-limiting by coca growers increased the price for coca derivatives in the UHV; this was largely because of *Sendero Luminoso* (SL—Shining Path), Maoist political insurgents, who demanded a greater share of the cocaine-base profits. The SL extended their area of influence; charged a tax on coca leaf, paste, and base; and attempted to set prices among the Colombian traffickers, growers, and lab operators—therefore, the prices for coca products varied widely in 1990, showing an average 100 percent increase.

The majority of cocaine base is moved from UHV staging areas by air and by river to Colombia for conversion to cocaine HCl. Drug-control efforts in Peru have been ineffective; violence, political factions, rivalry between the Peruvian police and military, and widespread corruption in a severely depressed economy have contributed to Peru's lack of effectiveness.

Bolivia. By the early 1990s, almost 75 percent of illicit coca was grown in the Chapare, Carrasco, and Arani provinces, in Cochabamba department, Bolivia. Legal cultivation of some 35,000 acres (14,000 ha) occurs only in the Yungas. Small farmers and unemployed migrants cultivate the coca in the Chapare on plots that average one to two acres (less than 1 hectare). When the market price drops below their cost of production, farmers choose not to sell the leaf. Most leaf that is sold to middlemen (*intermediarios*) is processed in the Chapare and then refined into base or cocaine HCl in the Beni, Cochabamba, or Santa Cruz departments. Due to increased enforcement in the early 1990s, some traffickers moved their base of operations to less accessible locations, and more paste is refined into cocaine base or HCl by about 35 Bolivian trafficking organizations.

Colombian and Bolivian traffickers have integrated some operations vertically, from wholesale paste purchase through cocaine base and HCl refining and export. The U.S. government estimates that as much as 35 percent of Bolivian coca paste may be processed in Bolivia prior to export. Chemicals arrive by truck, train, and aircraft from Brazil, Chile, Argentina, and Paraguay. The base is smuggled to Colombia in private aircraft from the Beni. Increasing its law-enforcement efforts, the Bolivian government eradicated about 10 percent of the cultivation, dismantled a number of laboratories, and disrupted several major trafficking organizations (e.g., Meco Dominquez, Mario Ariaz-Morales, Martin Morales-Daczer).

Colombia. Proximity to a large cash-based U.S. marketplace, powerful criminal organizations, indigenous entrepreneurial spirit, vast tracts of uncontrollable land, and a long tradition of smuggling have made Colombia an ideal source for cocaine. The U.S. government estimated that in 1991 92,000 acres (about 37,500 ha) of the world's 526,500 acres (213,000 ha) of coca were cultivated in Colombia—mainly in the Llanos (plains) region, which encompasses almost 50 percent of eastern Colombia. There is also coca cultivation in Caqueta, Guaviare, Putumayo, and Vaupes departments, with crop expansion into Bolivar department and into south and southwest Colombia. Colombia's drug cartels are the world's leading producers of both cocaine HCL (which is sniffed or snorted) and CRACK (which is smoked).

Colombian cocaine-trafficking organizations are sophisticated and well-organized industries, which derive their strength from control of cocaine laboratories and the smuggling routes to North America. After financing the cultivation of coca plants in Bolivia and Peru, Colombian traffickers often oversee the processing of the leaves into coca paste and sometimes base, which may then be shipped to laboratories in Colombia where the traffickers refine the coca paste—first into coca base and then into cocaine HCl by the ton. Recently, Peru and Bolivia have stopped shipping some of their coca products to Colombia and have begun to refine them into cocaine HCl in laboratories near their own fields, but as of the early 1990s Colombia operates the greatest number of base and HCl labs.

Cocaine is a major threat to weakening Colombia's democratic institutions and directly or indirectly affecting everyone in the country. Colombians increasingly recognize that the violence and corruption that accompany drug trafficking are harming their economy and society. By the early 1990s, under President Cesar Gaviria, the Colombian government security forces began enforcement procedures against cocaine traffickers. The Colombian police have also eradicated virtually all marijuana cultivation in the traditional growing areas along the North Coast and Guajira peninsula. The government of Colombia consequently damaged the leadership structure of the Medellin cartel by jailing its leader, Pablo Escobar. Some feared

TABLE 3
Marijuana Production Estimates

U.S. MEASURE

Source Country		*Net Cultivation (acres)*	*Net Production (tons)*
Mexico	1990	86,574	21,687
	1991	44,250	8,553
Colombia	1990	3,705	1,650
	1991	4,940	1,650
Jamaica	1990	3,013	908
	1991	2,347	705
Belize	1990	181	66
	1991	133	54
Others	1990	NA	3,850
	1991	NA	4,950
Domestic U.S.	1990	NA	5,500–6,600
	1991	NA	3,977–5,077

METRIC MEASURE

Source Country		*Net Cultivation (hectares)*	*Net Production (metric tons)*	*Percentage* of Total Supply*
Mexico	1990	35,050	19,715	42%
	1991	17,915	7,775	
Colombia	1990	1,500	1,500	8%
	1991	2,000	1,500	
Jamaica	1990	1,220	825	3%
	1991	950	841	
Belize	1990	65	60	>2%
	1991	54	49	
Others	1990	NA	3,500	24%
	1991	NA	4,500	
Domestic U.S.	1990	NA	5,000–6,000	22%
	1991	NA	3,815–4,615	

Summary	*1990*	*1991*
Gross Marijuana Available	33,660–34,760 (tons)	19,889–20,989 (tons)
Less U.S. Seizures,** Seizures in Transit, and Losses	3,850–4,950 (tons)	3,850–4,950 (tons)
Net Marijuana Available	28,710–30,910 (tons)	14,939–17,139 (tons)

Summary	*1990*	*1991*
Total Marijuana Available	30,600–31,600 (metric tons)	18,080–19,080 (metric tons)
Less U.S. Seizures,** Seizures in Transit, and Losses	3,500–4,500 (metric tons)	3,500–4,500 (metric tons)
Net Marijuana Available	26,100–28,100 (metric tons)	13,580–15,580 (metric tons)

*Percentages were rounded off and reflect midpoints of the quantity ranges in this table. For purposes of calculation and comparison, all the marijuana produced overseas was assumed to be potentially available for import to the United States.

**U.S. seizures included coastal, border and internal (not domestic eradicated sites). Seizures in transit included those on the high seas, in transit countries, from aircraft, etc. The loss factor included marijuana lost because of abandoned shipments, undistributed stockpiles and inefficient handling and transport, etc.

SOURCE: *International Narcotics Control Strategy Report 1992* and the U.S. Drug Enforcement Agency.

that jailing Escobar would not curtail his cocaine trafficking, but it did have a symbolic effect on the Medellin cocaine business. (Escobar was later killed after escaping from jail.)

A signatory of the 1961, 1971, and 1988 United Nations International Narcotics Control Conventions, Colombia demonstrates its political will and commitment to investigate and immobilize major cocaine traffickers and to eradicate marijuana and opium. Colombia has also created public-order courts and begun to share evidence, reform its judiciary, and track the substantial money flows into the country—requiring the banking institutions to keep records on cash transactions over $10,000.

In the realm of CROP CONTROL, despite widespread testing of various coca herbicides, the government has not begun a major coca-eradication effort; this is largely because it is not a focus of antidrug efforts—given the location of the fields in terrorist controlled land, it is dangerous for ground forces and almost impossible for air attack. Fearing a new and burgeoning heroin business, in 1992 the Colombian government agreed to spray the common garden herbicide glyphosate (Roundup) to kill the source—the opium poppy fields—after a widespread manual eradication effort in 1991. Since the mid-1980s, marijuana production continues to be minimal because of an effective herbicidal campaign.

The Colombian national police, the military, and the security forces have conducted major operations against the Medellin and Cali cocaine cartels with the assistance of U.S. technical and information support. Colombia's government has, however, paid a heavy price for its action, suffering almost 500 deaths by assassination or during enforcement operations. Colombia has also threatened to use, or has used, the tool of extradition to incarcerate or immobilize major traffickers. In late 1990, President Cesar Gaviria's offer of amnesty (a plea-bargaining opportunity for major traffickers) resulted in decreased violence throughout the country and the surrender and imprisonment of five traffickers and one terrorist, including Pablo Escobar and the three Ochoa brothers (Jorge Luis, Juan David, and Fabio).

Opium/Heroin. The opium poppy (*PAPAVER SOMNIFERUM*) is the source of heroin. It is grown in three principal geographic regions: Southeast ASIA, Southwest Asia, and Latin America. The Southeast Asian GOLDEN TRIANGLE countries of Myanmar (Burma until 1989), Laos, and Thailand in 1991 cultivated approximately 81 percent of the world's total, 488,000 acres (195,000 ha), yielding 2,500 metric tons of opium, which would yield 250 metric

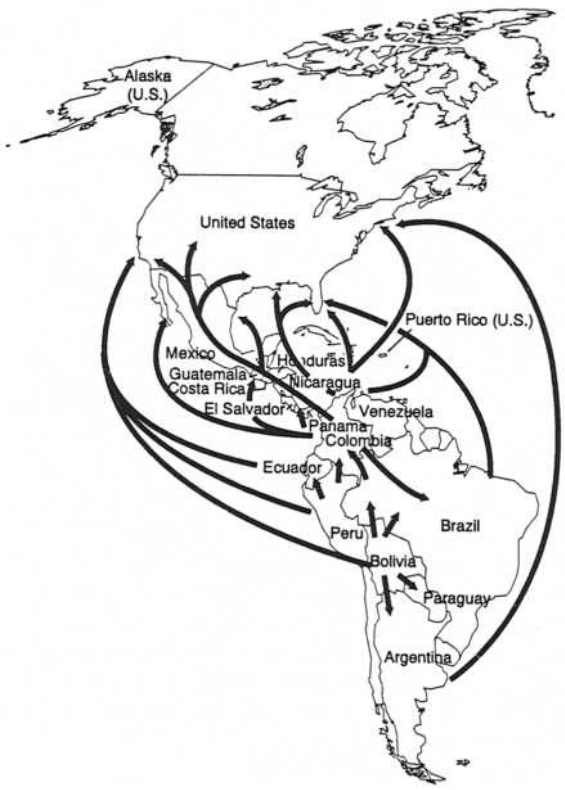

Figure 1
Cocaine Distribution

tons of heroin. The Golden Crescent countries of Afghanistan, Iran, and Pakistan cultivated approximately 11 percent, and the Latin American countries of Mexico, Guatemala, and Colombia (plus the Middle Eastern country of Lebanon) produced approximately 8 percent. India is the world's largest cultivator of licit opium, producing about 35,000 acres (14,000 ha) annually for the international medicinal market.

Southeast Asia's Golden Triangle: Myanmar. The largest supply of illicit opium—56 percent of U.S. availability—comes from the Golden Triangle of Southeast Asia. Fields of opium poppy are planted on hillsides that have been prepared by ancient slash-and-burn agricultural methods. Nearly 90 percent of Southeast Asian opium comes from the Union of Myanmar (Burma), where cultivation areas are largely controlled by antigovernment insurgents in the Shan state. Heavy cultivation exists east of the Salween river and in the eastern and southern parts of the Shan state, at an average elevation of 3,300 feet (1,000 m). Fields are small, averaging about an acre (0.5 ha). The climate is ideal for growing poppy. The growers depend on opium for survival, receiving subsistence prices for and selling entire stocks to the political insurgents, who use the proceeds for food, arms, and ammunition. The opium is also consumed locally by large numbers of addicts.

Most processing of opium and heroin in Southeast Asia occurs in Myanmar, with only small amounts in Thailand and Laos. The Shan United Army and the Wa insurgent groups control refineries along the Thai/Myanmar border; the Kokang, Wa and Kachin ethnic groups also operate large heroin refineries along the China/Myanmar border. Increasing amounts of heroin are smuggled via southern China to Hong Kong, south through Malaysia and Singapore, and west through India and Bangladesh.

With the overthrow of the long-standing government of Burma by a military junta in 1988—and ongoing political strife in the new Union of Myanmar—suspension of aerial opium eradication and diminished enforcement contributed to increases in opium cultivation, heroin refining, and drug trafficking. A signatory to the 1961 SINGLE CONVENTION ON NARCOTIC DRUGS, but not to the 1972 Protocol to the Convention or the 1971 PSYCHOTROPIC SUBSTANCES CONVENTION, MYANMAR had acceded to the 1988 UN Convention but now disputes the validity of extradition and submission of disputes to the International Court of Justice.

Thailand. Only a small amount of land is used to grow opium in Thailand, but it remains a net importer of opium, consuming far more than it produces. Developed transportation systems make Thailand the primary transit route to the opium/heroin world markets, shipping by air, sea, and overland. Since the mid 1800s, opium has been grown in the northern highlands by nomadic hill tribes, who are not tied to Thailand culturally, religiously or politically. Opium cultivation in Thailand is illegal, so the government has sponsored both eradication and crop-substitution efforts in the north.

Thailand is a party to the 1961 Single Convention on Narcotic Drugs and the 1972 Protocol to the Single Convention. In 1991, Thailand passed conspiracy and asset-forfeiture laws and a new extradition treaty with the United States; both are working on a mutual legal-assistance treaty.

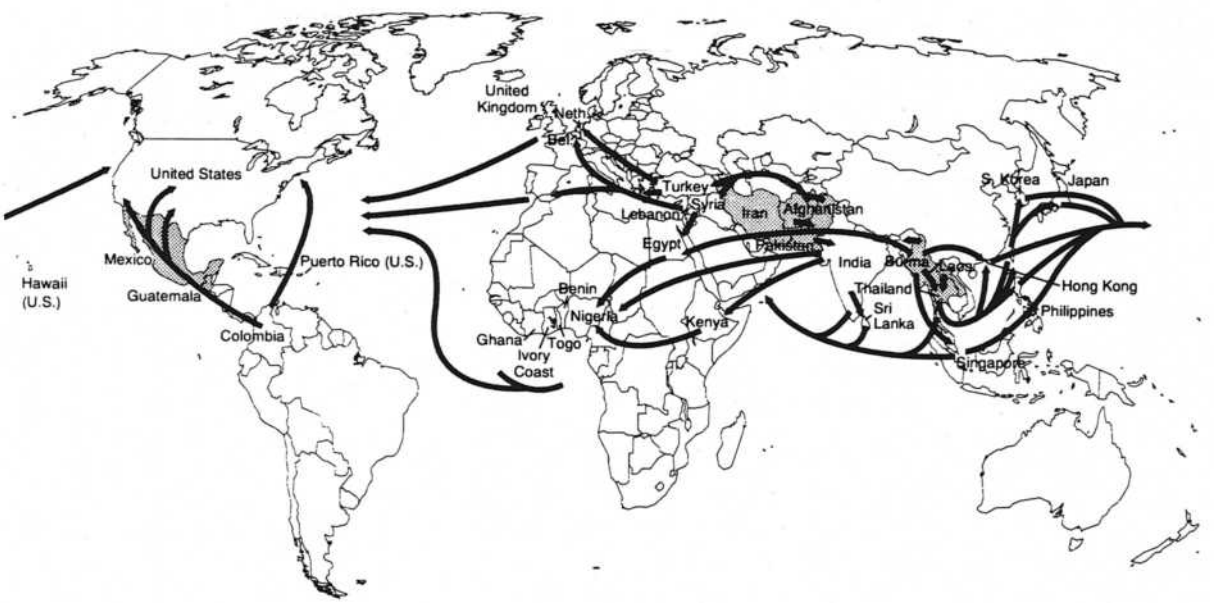

Figure 2
Opium/Heroin Distribution.

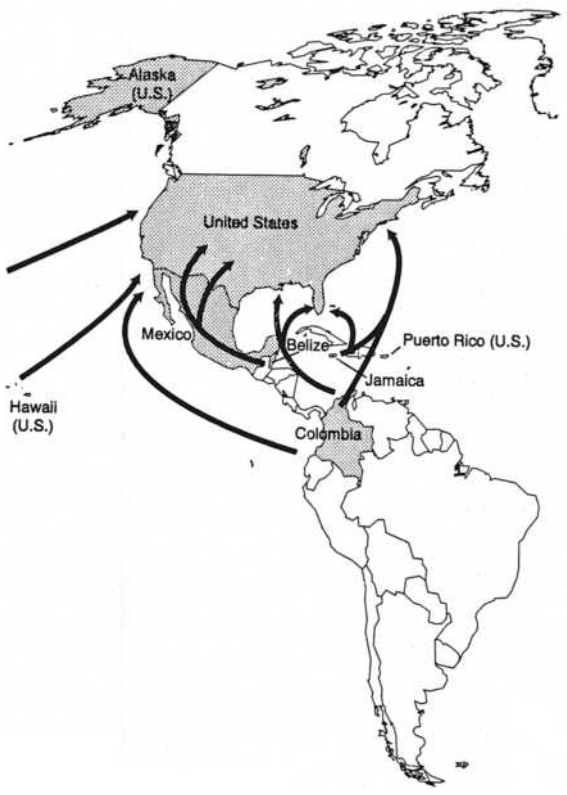

Figure 3
Marijuana Distribution.

Laos. Recent changes in the world's political order have resulted in cooperation by the Laotian government to reduce opium cultivation. Widespread reports of Lao military corruption and involvement with the traffickers, however, have limited the success. A landlocked country, Laos has been isolated and ignored by the West since 1975 when the Communist Pathet Lao seized power; opium poppies have been grown in its nine northern provinces, yielding in the early 1990s about 20 percent of Burmese production. Three crop-substitution projects have had limited success—one in Houaphanh province, one in Vientiane province, and one in Xiang Khouang province.

The Lao government does not have a mutual legal-assistance treaty or an extradition treaty with the United States, but it does have a formal memorandum of understanding and informal agreements with U.S. agencies to cooperate more fully in drug-control efforts.

China and the Golden Triangle. In its 1992 International Narcotics Control Strategy Report, the U.S. government stated that opium cultivation may be emerging as a major problem in the People's Republic of China. China has become a major narcotics transit point with its open border to Myanmar, its location adjacent to the Golden Triangle, and its excellent transportation and commu-

nication links with the trade ports of Hong Kong and Macao. Much of the heroin processed by the Kokang Chinese in the Golden Triangle travels by road through China's Yunnan, Guangxi, and Guangdong provinces to Hong Kong for overseas distribution. In 1991, Chinese law enforcement seized more drugs and investigated more cases than at any time since the Communist takeover. A spreading domestic opium consumption appears to be accompanying the increased heroin flow.

The Golden Crescent: Pakistan, Afghanistan, and Iran. The Golden Crescent supplied about 21 percent of the heroin consumed in the United States in the early 1990s. In area under cultivation, the Golden Crescent countries produce almost 11 percent of the world's opium.

Pakistan. This is a producer and an important transit country for opiates and HASHISH. The Islamic government of Pakistan maintains a poppy ban in areas under its control and manages to maintain about the same production level from year to year, but cultivation has increased slightly in areas where government control is ineffective or only nominal. Cultivation is both rain fed and irrigated in the northwest and the tribal areas of Kyber, Mohmand, and Bajaur. Once the poppy is harvested, it is processed into opium and heroin in more than a 100 clandestine mobile laboratories in the Northwest Frontier Province (NWFP) bordering Afghanistan, which is controlled by armed tribes who maintain traditional cross-border connections.

Pakistan is party to the 1961 UN Single Convention on Narcotic Drugs, the 1971 UN Convention on Psychotropic Substances, and the 1988 UN Convention Against Illicit Traffic of Narcotic Drugs and Psychotropic Substances. Yet, with widespread corruption and government inaction, Pakistan failed to enforce its counternarcotics laws in the tribal areas, raising questions about its compliance with the 1961 Convention. Pakistan's government does however cooperate with U.S. law-enforcement agencies and has responded positively to extradition requests.

Afghanistan. After Myanmar, Afghanistan is the world's second largest producer of illicit opium. Considered an effective cash crop, opium has been grown for generations in Afghanistan, in the Helmand valley and Nangahar province, and used for medicinal and culinary purposes. The opium is processed into heroin and smuggled across the borders of Iran through Turkey. Afghanistan's government exerts little control over production or trafficking. Drug revenues continue to finance political resistance operations against the Communist government and provide a livelihood for farmers who depend on the opium crops. Unless the government is willing and able to control opium production in the countryside, both production and domestic consumption will continue to rise. The end of Soviet occupation (1979–1989) has not brought the refugees home, but their return will affect Afghanistan's overall economy and may cause an increase in drug trafficking.

Afghanistan is a party to the 1961 Single Convention but not to the 1972 Protocol amending the Convention. It is a signatory to the 1971 Convention on Psychotropic Substances but not to the 1988 Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances.

Iran. Limited data exist on drug cultivation and trafficking since the Islamic Republic of Iran was established in 1979 under the Ayatallah Khomeini. Iran outlawed opium cultivation in 1980 but growth reportedly occurs in remote areas near the Pakistan and Afghanistan borders. Allegedly, laboratories process heroin from opium in the Kurdish areas of the northwest and the Baluch region in the southeast, with significant Irani and local addict populations consuming the product. The U.S. government estimated that Iran produces about 50 percent of the amount of heroin produced in Afghanistan.

Drug trafficking is increasing along the Afghan-Iran and Afghan-Pakistan borders. Baluch and Pashtun tribesmen from all three Golden Crescent countries smuggle drugs in addition to traditional contraband. Pakistanis and Iranis could increase poppy cultivation to help rebuild their livelihoods that were interrupted by almost twelve years of war.

Mexico. In the 1970s, Mexico began to smuggle significant amounts of heroin into the United States, replacing Turkey as the principal heroin supplier for U.S. addicts. Opium is grown and harvested twice a year—winter and spring—in Mexico's states of Sinaloa, Chihuahua, and Durango. In the 1990s, harvesting has become year round, and cultivation has expanded to include Mexico's west coast from Sinaloa to the Mexican-Guatemalan border. Supplying an estimated 23 percent of the heroin consumed in the United States, Mexican

traffickers produce both traditional brown and black-tar heroin, although the predominant type smuggled into the United States is the black-tar type. Conversion from the popular "Mexican brown" in the 1970s to the black-tar variety is a result of traffickers using more cost-effective mobile laboratories. The mobile labs are much harder to detect and can move with the harvesters, as they go from field to field collecting the opium gum and producing the purer black tar preferred by U.S. addicts. Although the mobile labs are found near the fields, Mexican law-enforcement personnel are also finding them near towns and cities, where chemicals and security can be acquired more easily. The administration of President Carlos Salinas (1988–1994) instituted effective law enforcement, including strong measures to combat official corruption, a 40 percent increase in opium eradication, and increased cocaine interdiction.

Mexico is a signatory to the 1988 UN Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances and entered into a Mutual Legal Assistance Treaty (MLAT) with the United States in 1991.

Guatemala and Colombia. These two countries have both begun to cultivate substantial amounts of opium in the late 1980s. By 1991, in Guatemala's western provinces of San Marcos and Huehuetenango, farmers harvested approximately 4,300 acres (1,700 ha) of opium poppy, which had been cultivated in steep mountain valleys on small plots. Mexican traffickers provide the financial, technical, and agricultural support for local growers to harvest three crops per year; the opium, however, is sent to Mexico for processing into heroin. The Guatemalan government has conducted aerial herbicidal eradication with some success, destroying almost a third of the total cultivated, but farmers are relocating their fields to more remote areas. In Colombia, in 1991, over 6,000 acres (2,500 ha) of opium poppy were located in 12 of the 32 states—planted for the most part in the Cauca and Huila departments and financed and controlled by the Cali cartel. Colombia has agreed to begin herbicidal spraying from crop-duster aircraft, as it did during its mid-1980s marijuana-eradication program.

Cannabis. A by-product of the HEMP plant *CANNABIS SATIVA* is marijuana, which is the most commonly used illicit drug in the United States. Both the plant and its PSYCHOACTIVE ingredient TETRAHYDRO-CANNABINOL (THC) are classified as CONTROLLED SUBSTANCES by the U.S. government, which estimates that Mexico supplies the majority of U.S.-consumed marijuana—perhaps as much as 63 percent. The U.S. supply accounts for another 18 percent, Colombia for 5 percent, Jamaica for 3 percent, and the remaining 11 percent comes from Belize, Laos, the Philippines, Thailand, Lebanon, Pakistan, and Afghanistan. Brazil and Paraguay also cultivate cannabis but the majority is consumed locally or exported to neighboring South American countries.

Mexico. Although *Cannabis* grows throughout the country, major concentrations have been located historically in the western states of Chihuahua, Jalisco, San Luis Potosi, Sinaloa, Sonora, and Zacatecas; it is also found in Mexico's eastern state of Veracruz and, recently, in the southern states of Chiapas, Guerrero, Michoacan, and Oaxaca. Farmers grew two crops per year, traditionally, but in many areas it is grown and harvested year round. *Cannabis* is cultivated by subsistence farmers who often intermingle the crop with corn and beans. Traffickers have introduced irrigation and technological advances to help the farmer (*campesino*) avoid eradication attempts and survive cyclical droughts. The traffickers control the processing and transport of the product into the United States, smuggling the vast majority by road.

Colombia. Once the primary source for marijuana consumed in the United States, in the 1990s Colombia cultivates about 5,000 acres (2,000 ha) in the traditional growing areas of Sierra Nevada de Santa Marta and Serrania de Perija of northeastern Colombia. Since the dramatic success of the Colombian government's 1980s aerial-eradication program, only small amounts of low-quality cannabis have been cultivated in Colombia.

Southeast Asia. This region produces a high-grade marijuana that became popular in the late 1980s; it is cultivated in Thailand and Laos, then shipped to staging points along Thailand's southern coast, to western Cambodia, and to the coast of Vietnam. Moved by ten-wheel trucks, the product is then loaded onto trawlers and taken to motherships in the Gulf of Thailand. Oceangoing vessels, yachts, and sailing boats have all been used to smuggle the product to the United States, with trans-Pacific shipments occurring in the spring and summer. U.S. traffickers usually control the commerce of marijuana into the United States, off-loading

their cargo to smaller faster vessels off the U.S. coast.

Two crops a year are generally harvested in Southeast Asia, in December–January and April–May. Cultivators normally press the harvested marijuana into kilogram blocks, using a hydraulic press, and then package the blocks into aluminum foil or plastic bags that are vacuum-packed. They are hermetically sealed with heat and wrapped with nylon-reinforced plastic tape, then stored in tin canisters, burlap sacks, nylon or canvas gym bags, or boxes—all designed to maintain the product's composition, eliminate odor, and prevent mildew.

The THC content of Southeast Asian marijuana can be as high as 9 percent, whereas the average THC content for Mexican or U.S. marijuana is only 2 to 3 percent.

Jamaica. The successful eradication campaigns mounted since 1987 have decreased significantly Jamaica's importance as a supplier of *Cannabis* in the form of ganja. Cultivation has shifted from the accessible wetlands of west-central Jamaica to remote sites in the highlands, often in plots smaller than an acre. In the early 1990s, of 4,500 acres (1,800 ha) cultivated, almost 50 percent were reportedly eradicated. The rest was smuggled into the United States in concealed storage areas of pleasure craft, as well as in commercial fishing vessels, cargo ships, and container ships.

POLITICAL AND ECONOMIC SIGNIFICANCE

Although drug cultivators, transportation workers, processors, laboratory workers, middlemen, and smugglers receive their wages, the majority of the money made in the drug business remains in the consuming country or is invested in off-shore banking havens. Drug-producing countries do not normally offer attractive long-term investment opportunities. Countries such as Peru, Bolivia, Myanmar, and Afghanistan have troubled economies, which do not attract traffickers' investment portfolios; rather, traffickers spend money on luxury items, such as foreign real estate and automobiles, race horses, gambling houses, yachts, clothes, and jewels.

In the Golden Triangle and the Golden Crescent, drug production and trafficking offer a primary cash crop for food and the support of political (antigovernment) operations. Resistance groups in Afghanistan and Pakistan and insurgent tribes in the Golden Triangle use the profits from the sale of opium to buy rice and the arms to fight the central governments. Politically speaking, illicit-drug production and trafficking offer a viable means of acquiring wealth, which can be instrumental in buying power and influence. In some countries, the traffickers and insurgent groups may be identical (such as the Wa or the Shan United Army of Burma); in others, insurgency and trafficker goals may be diametrically opposed (such as the Cali cartel and the FARC in Colombia). Most trafficker organizations work to coopt the government and maintain the status quo, buy power and protection, and keep a low profile; insurgent groups, however, seek to be highly visible and wish to change the existing power structure. Despite the opposing objectives of both, traffickers and insurgents often function symbiotically; that is, both need hard currency, security, protection, and armed support to evade detection and apprehension.

"Drug czar" Barry McCaffrey (left) meets Mexican Foreign Minister Rosario Green (second left), Attorney General Janet Reno (center), and Mexican Attorney General Jorge Madrazo Cuellar before the opening of the U.S.-Mexico Drug Strategy Conference, December 15, 1998, in Washington, D.C. (AP Photo/Dennis Cook)

HISTORICAL SUPPLY SHIFTS

The 1960s and 1970s. Drug production and trafficking have undergone major shifts since the 1960s. After the so-called French Connection was broken between 1968 and 1972, Mexico began to supply the United States with a low-quality heroin to fill the market demand. As Mexican eradication became more successful in the mid to late 1970s

and the Iranian Islamic revolution erupted in 1979, significant amounts of Southwest Asian heroin from Afghanistan and Pakistan were smuggled, often by Iranians, through Western Europe into the United States. Throughout the 1970s, heroin from Mexico, Southeast Asia, and the Middle East was high on the U.S. drug-control policy agenda. No one denied that cocaine and marijuana abuse might be dangerous; indeed, initial attempts were made to initiate bilateral programs with the Andean cocaine source countries in their traditional growing areas, but because policymakers believed that the negative health consequences of heroin consumption were far worse, the U.S. law-enforcement emphasis was placed on cocaine and marijuana.

The 1980s. In the 1980s, targeting heroin gave way to focusing on the reduction of cocaine and marijuana use in the United States, since greater numbers of Americans were using and abusing them, creating large drugged populations. Nongovernmental institutions became very active in spreading the Just Say No message of the Reagan Administration (1981–1989). Moreover, until the early 1980s, when research had documented the negative health consequences of cocaine, the drug enjoyed a glamour and allure that heroin had never possessed. In some circles, the ability to afford cocaine was a sign that one had succeeded. Most believed that cocaine was not addictive and it became the recreational drug preferred by Hollywood, sports figures, and musicians. The 1980s was the Coke Decade—with cocaine used both by the affluent consumption-oriented yuppie "me generation," as well as the poor disenfranchised underclass who tried to emulate their heros and "make it" in their own world. Both the powder and the rock-crystal crack forms found eager markets and ready money in the so-called affluent Reagan years. The stock market crashes of 1987 and 1989 ended the boom in the national and the drug economies.

When Colombia replaced Mexico in the early 1980s as the primary source of U.S. cocaine and marijuana, smuggling vast quantities through the South Florida peninsula, the Reagan Administration turned its focus to cocaine and marijuana control in the Western Hemisphere. In the late 1980s, the Bush Administration (1989–1993) continued the same cocaine policy but decreased federal attention on marijuana supply reduction. Bolivia and Peru quickly expanded their production of illicit coca in nontraditional growing areas of the Chapare and the Upper Huallaga Valley, two areas that remain the major source for the world's illicit coca. Surrounding and potential transit countries also became more involved in the cocaine smuggling enterprise. The Caribbean nations functioned as attractive transit points for both cocaine and marijuana from South America. As the United States placed more enforcement pressure on the Caribbean, the traffickers shifted their routes through Central America and Mexico. In the mid-to-late 1980s, Mexico became a principal transit and smuggling route for an estimated 50 percent of the cocaine entering the United States. In response to smuggler shifts, both Mexico and the United States have increased interdiction efforts along the joint southwest border and the U.S. 1992 Drug Control Strategy focuses its attention in Mexico predominately on improving cocaine interdiction.

The 1990s. U.S. policymakers are faced with a number of new challenges—namely, increased heroin production and trafficking in Central Asia (China), in Central and South America (Guatemala and Colombia), and in Myanmar and Afghanistan. Another challenge is the growing cocaine business in Bolivia and Peru, where increased processing of coca into cocaine products occurs. Finally, policymakers need to consider the potentially devastating impact of increased cocaine and heroin demand and consumption in the new democracies of Eastern Europe and the new republics of the former Soviet Union.

Beginning in 1991, the U.S. government expressed its concern over an increase in worldwide heroin production, trafficking, and abuse. Record seizures have been made in China's Yunnan province—signaling major changes in trafficking routes out of the Golden Triangle through China, Hong Kong, and Taiwan to the West. Heroin traffickers have begun to use the immense container-shipping industry to smuggle large amounts of heroin from Asia into the United States. In June 1991, the single largest heroin seizure in the world was made in San Francisco, hidden in containerized freight from Taiwan. Colombia also became a significant cultivator of opium for the first time, in the 1990s—planting an estimated 6,000 acres (2,500 ha) of opium in 1991. Although opium cultivation has decreased in Mexico and the Golden Crescent, increasing demand in the United States may be met by Colombia and Myanmar.

The cocaine epidemic of the 1980s, as measured by prevalence and incidence indicators, appears to have peaked and is declining for certain cohort populations, but concern continues over the chronic intensive use of the crack form among the predominantly minority underclass; those least able to cope—the uneducated, unemployed, and disenfranchised—are the victims. With processing facilities now closer to source countries least able to implement effective drug-control programs politically and economically, these two problems present daunting challenges for U.S. public policymakers.

Finally, the massive political, economic, and social changes that occurred since 1989—the democratization and political upheaval in the Eastern bloc and the Soviet Union—may result in increased drug demand, driving up drug production, trafficking, and serious negative health consequences. Unfortunately, accompanying the economic difficulties and growing political pains in the fledgling democracies are increases in crime, violence, and drug abuse.

(SEE ALSO: *Amphetamine Epidemics; Drug Interdiction; Money Laundering; Operation Intercept; Terrorism and Drugs; Transit Countries for Illicit Drugs; U.S. Government/U.S. Government Agencies*)

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JAMES VAN WERT

INTERPOL *See* Drug Interaction; International Drug Supply Systems

INTOXICATION *See* Addiction: Concepts and Definitions

INTOXILYZER *See* Drunk Driving

ITALY, DRUG USE IN In Italy, the impact of illicit drug use was first felt on a broad scale during the mid-1960s. The patterns in Italy were similar to those seen in other European countries. They seemed to be associated with the contestation by young people of existing political and social situations. As in the United States at the same time, this phase was influenced by the cultures of the East—especially those of the Indian subcontinent, Southeast Asia, and the Middle East—in all of which some amount of drug use was not illegal. *Cannabis sativa*, the HEMP plant that produced MARIJUANA, GANJA, HASHISH, and other variants, was particularly unhampered by legislation there and was enjoyed by locals and outsiders, some of whom found ways to smuggle it into the West, where in many instances it was illegal.

In addition, the OPIOIDS (especially HEROIN) began to be used illicitly, and by the 1970s serious consequences ensued. By then, the countercultural movement and its abuse of illicit drugs had lost most of its original idealistic principles. Abusers were simply in search of ever more and ever stronger psychotropic effects. Moreover, criminal organizations took charge of the illicit drug trade, not only to increase their profits but also to control and direct the political and social development of the youth of Italy. For the most part, users became abusers who were physically dependent on their drug, so their behavior could be controlled by the suppliers.

In the 1980s, the drug scene changed, with various control measures and less heroin available. In addition, with less heroin being sold, longer intervals occurred between drug doses for many users. Such modified habits led to decreased tolerance and increased overdosing, with ensuing deaths. For these reasons, the number of heroin addicts in Italy decreased—then, in the mid-1980s, COCAINE emerged as the new illicit drug problem. The CRACK and FREEBASE forms were especially harmful among young ADOLESCENTS. More detailed data are contained in the annual reports of the National

TABLE 1
Treatment Typology Provided by Public Services (1991–1994), Italy

Typology	*Addicts (percent)*			
	1991	*1992*	*1993*	*1994*
Psychosocial and/or rehabilitating	38.1	37.3	39	41.2
Pharmacologic				
Integrated	24	27	26.5	28.2
(from which "methadone treatment")	(21)	(23.4)	(23)	(14.3)
Methadone treatment (short & long, not integrated)	9.3	9.7	12	27.6
Naltrexone	7.7	7.6	8.2	
Other antagonistic	0.14	0.20	0.13	
Other	19.8	16.3	13.4	

SOURCE: Alcohol and Drug Addiction Central Service, Ministry of Health. Data processed by the Istituto Superiore di Sanità.

Health Council (1985–1991) and the reports of the Department of Social Affairs (1991–1993).

LEGISLATION

At the beginning of the drug-abuse phenomenon of the 1960s, the legislation in force had been passed in 1954. It proved to be insufficient for coping with emerging conditions; it did not take into consideration the political-cultural trends, the scientific knowledge of the day, or the increasingly important role of public health.

New legislation in 1975 was characterized by such innovative elements as the nonpunishment of

TABLE 2
Seizures of Illicit Drugs by Raw Weight in Kilograms, Italy

	Opioids				*Stimulants*			*Hallucinogens*			
Year	*A*	*B*	*C*	*D*	*E*	*F*	*G*	*H*	*I*	*J*	*K*
1980	—	267	197	—	76	—	—	—	4,907	—	—
1981	—	82	141	—	64	—	—	—	11,204	—	
1982	12	0.5	230	1	105	5	4	968	3,908	23	0.1
1983	7	3	314	11	223	71	3	1,018	4,137	23	0.1
1984	2	0.2	457	3	73	0.4	0.4	854	5,185	14	0.3
1985	5	0.9	275	7	107	0.2	0.05	372	1,051	8	0.3
1986	2	7	333	1	129	0.4	0.8	293	15,731	3	0.2
1987	2	0.7	323	9	330	5	1	1,207	11,817	6	0.01
1988	3	1	576	5	612	0.7	0.9	256	6,912	0.0	0.3
1989	1	1	685	0.8	668	0.7	1	239	22,993	0.4	0.1
1990	—	8.8	1,003	2.2	802	0.7	—	182	7,704	1,258	0.23
1991	—	—	1,556	—	1,300	0.7	5,983[a]	499	9,223	—	4,016[b]
1992	—	—	1,353	—	1,367	15.4	22.2	584	22,620	—	12,759[b]

NOTE: A = opium B = morphine C = heroin D = others
E = cocaine F = amphetamine-like G = others[a] (includes MDA, MDMA, in number of units)
H = marijuana I = hashish J = hashish oil K = LSD and others[b] (in number of units)
SOURCE: Drug Enforcement Service, Ministry of the Interior.

TABLE 3
Seizures of Drugs (Raw and Normalized Weight, average doses*), Italy, 1988

Heroin (kg)			*Cocaine (kg)*		
raw (37%)	*normalized (100%)*	*doses no.*	*raw (88%)*	*normalized (100%)*	*doses no.*
576	213	14,200–8,500	612	539	13,500–9,000

*average dose, heroin = 0.015–0.025 gr
mean dose, cocaine = 0.040–0.060 gr

the addict found to be in possession of a moderate quantity of illicit drugs. The quantity was to be examined and quantified, and it was to be considered in relationship to the physical and psychological needs of the addict. Unfortunately, this individualistic approach was poorly applied, which made the law useless.

The regulations approved in 1990 improved the state's power of both repressive action and intervention, and it defined a daily mean dose to separate administrative offenses from crimes. The objective was to recover and rehabilitate drug addicts. A 1993 referendum, however, repealed the prohibition on personal drug use and canceled references in the regulations to the daily mean dose.

TREATMENT FACILITIES

In accordance with national policy guidelines, a network of facilities was set up and various links were established with rehabilitation, law enforcement, and judicial structures. This process was worked out with public support; the aim has been to sustain every initiative to reduce the availability and demand for drugs.

Of the addicts served by the facilities, almost all are heroin abusers, some not yet dependent. Starting by weaning them from heroin with METHADONE, the facilities provide integrated and custom-designed programs founded mainly on nonpharmacological support.

A wide range of resources are available; 576 public facilities and 276 residential communities and sociorehabilitative structures (public, private, and voluntary—most of them situated in northern Italy). Voluntary services continue to increase in importance both in number and in regional distribution. The effectiveness of the facilities has been proved, since trained personnel and good records provide such statistics on trends (see Table 1).

SEIZURES OF ILLICIT DRUGS

Various trends can be seen by studying the records of seized drugs. Some decreasing trends have been recorded for MORPHINE in 1982, for heroin in 1981 and 1985, and for cocaine in 1984. Irregular trends emerged for *Cannabis* products: a 128-percent increase in 1981, a decreasing trend until 1985, and two huge increases in 1986 and in 1989 (see Table 2). The decision to standardize descriptions of drug seizures by reference to the percent of the primary drug instead of the raw weight of the primary drug seized should improve the accuracy of record keeping (see Table 3).

TABLE 4
Drug Abuse–Related Deaths (1980–1993) and "Empiric" Mortality Rate (1986–1993), Italy

Year	*Deaths*	*Total Addicts*	*Rate (percentage)*
1980	206	NA	—
1981	237	NA	—
1982	252	NA	—
1983	237	NA	—
1984	392	NA	—
1985	237	NA	—
1986	276	32,000	0.86
1987	516	39,000	1.32
1988	809	47,000	1.72
1989	972	55,000	1.77
1990	1,161	66,700	1.74
1991	1,383	76,200	1.81
1992	1,217	89,000	1.37
1993	888	109,000	0.81

SOURCES: Ministry of the Interior; Ministry of Health.

DRUG ABUSE-RELATED DEATHS

Drug abuse-related deaths also show irregular trends. Most deaths could be attributed to heroin overdoses or to accidents while injecting it. After 1980, two large increases in the death rate occurred, first in 1982 and then in 1984, followed by a steady rise into 1986. From 1986 to 1988, the "empiric" mortality rate nearly doubled; it subsequently remained steady until 1991 and then dropped until 1994 (see Table 4), except among the elderly, for whom the rate increased.

ALCOHOL ABUSE

ALCOHOL use in Italy strongly differs from drug use for historical, traditional, behavioral, and cultural reasons; supply and distribution are also different, since alcohol is free from legal restrictions. Wine is the most frequently used alcoholic beverage. Although a gradual displacement in wine consumption occurred during the 1980s, with substitution of other liquors and beers, still the total amount of alcohol (percent of ethanol) consumed remained almost constant.

ALCOHOLISM is mainly a problem of chronic abuse by adults over the age of 40. It is mainly a problem in northern Italy. Since the 1980s, however, increasing numbers of young people are abusing liquors and beer. Alcoholism has also become complicated by the combining of alcohol with psychotropics (e.g., tranquilizers), especially by women over 40.

Driving-license regulations have, since 1988, included a test that measures the breath concentration of alcohol. The alcohol level must not be over 8 grams per liter (g/l), approximately that of other countries of the European Economic Community.

(SEE ALSO: *Britain, Drug Use in; Netherlands, Drug Use in; Sweden, Drug Use in*)

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USTIK AVICO

J

JAILS *See* Prisons and Jails

JAPAN, DRUG USE IN *See* Amphetamine Epidemics; Epidemics of Drug Abuse

JELLINEK MEMORIAL FUND In 1955 the Jellinek Memorial Fund was established to commemorate Dr. E. M. Jellinek's great contribution to the field of ALCOHOL studies. A capital fund was developed and the interest from this fund has been used to provide an annual cash award to a scientist who has made an outstanding contribution to the advancement of knowledge in the alcohol/alcoholism field. The first award was presented in 1968.

Each year the board of directors of the Jellinek Memorial Fund designates the specific area of research for which the award will be made and appoints an Expert Selection Committee to review candidates and recommend an appropriate awardee. The awardee may be selected from any country, the sole criterion being the scientific contribution that the person has made within the selected category. The award is traditionally presented at a major international conference and, if necessary, travel and accommodation expenses are provided to permit the winner to attend the conference for the presentation. The following general criteria have been accepted by the board and by previous selection committees as guidelines:

1. The award is to be given to the person deemed to have made, during the preceding years, the greatest scholarly contribution to human knowledge of problems relating to alcohol, in the designated research area.
2. The person selected for the award should be someone who would provide an example and serve as a model for others who might be attracted to work in the field.
3. Only living scientists should be considered for the award.
4. Advanced age or impending retirement would not disqualify someone from candidacy. However, if two or more scientists were considered approximately equal, the one more likely to continue longer in the field would be favored.
5. If the outstanding contribution of a candidate was made more than ten years ago, consideration for the award would require evidence of the candidate's continuing interest and active participation in alcohol research.
6. Other factors being equal, the person would be favored whose primary identification continued to be in the field.
7. If a member of the Expert Selection Committee is deemed eligible for the Jellinek Award, the chair of the selection committee should consult with the president and request the resignation of the committee member.

8. If a previous award winner becomes a candidate and appears equal to or above all other candidates on the basis of unique new achievements, he or she should not be ruled ineligible. The chair of the selection committee should consult with the president to ensure that the award is for new achievement and determine if he or she is eligible.
9. The award will normally be made to an individual researcher most highly recommended by the selection committee. In special circumstances, if the selection committee recommends two persons of equal and outstanding merit, a joint award may be made to the two.

In 1999, the award was given in the category of epidemiology and population studies jointly to Kaye Middleton Fillmore and Alexander Wagenaar. Fillmore, a School of Nursing adjunct professor in the Department of Social & Behaviorial Sciences and the Center for Health & Community, received the award for her contribution to the basic understanding of the life course of drinking and of alcohol problems in a multinational context. Wagenaar, Professor of Epidemiology and Director of the Alcohol Epidemiology Program at the School of Public Health, University of Minnesota, received the award for lifetime achievement in community intervention and policy evaluation research on alcohol.

(SEE ALSO: *Disease Concept of Alcoholism and Drug Abuse*)

H. DAVID ARCHIBALD
REVISED BY DONNA CRAFT

JEWS, DRUGS, AND ALCOHOL

Who hath woe?
Who hath sorrow?
Who hath contentions?
Who hath babbling?
Who hath wounds without cause?
Who hath redness of eyes?
They that tarry long at the wine;
they that go to seek mixed wine.
Look not thou upon the wine when it is red,
when it giveth his color in the cup,
when it moveth itself aright.
At the last it biteth like a serpent,
and stingeth like an adder.
Thine eyes shall behold strange women,
and thine heart shall utter perverse things.
Yea, thou shalt be as he that lieth
down in the midst of the sea,
or as he that lieth upon the top of a mast.
They have stricken me, shalt thou say,
and I was not sick;
they have beaten me, and I felt it not:
when shall I awake?
I will seek it yet again.

—Proverbs 23:29–35

As illustrated by this biblical description of intoxication, alcoholic blackouts, alcohol-related physical and social problems, alcoholic hallucinations, loss of control of drinking, and alcohol dependence was not unknown to the ancient ancestors of today's Jewish population. The Hebrew Bible (called by Christians the Old Testament) includes several illustrations of alcohol-related problems, such as the drunkenness of Noah, which led to family strife, and the incest between Lot and his daughters.

Modern literature about the role of ALCOHOL in the Jewish community displays two very different trends. On the one hand, Jews are regarded as a population with few alcohol problems; and a variety of cultural, spiritual, or physiological explanations are suggested to explain the relatively low rate of ALCOHOLISM among Jews. On the other hand, studies of alcoholism among Jews point out that many cases often go unrecognized, because of the myth of Jewish immunity to alcohol abuse.

Surveys of U.S. drinking practices conducted in the 1960s found that most males who considered themselves Jewish reported drinking to some extent, but few reported alcohol problems. However, the number of Jewish subjects in these studies was small. A more recent study of U.S. male college students and university employees reported that although Jewish and Christian subjects had generally similar drinking patterns, Jews were less likely to drink more than six drinks on any one occasion and less likely to report alcohol problems.

Israel reports a lower per-capita alcohol consumption than countries in Western Europe or the Americas and a lower death rate from cirrhosis of the LIVER. (Cirrhosis mortality is thought to correlate with rates of alcoholism.) A single study of a sample of 266 adult Jews in the general public in

New Haven, Connecticut, found a lifetime prevalence of alcohol abuse of 1.7 percent, significantly lower than the rate reported for Protestants, Catholics, or those without religious affiliation. The prevalence of alcohol dependence was not reported and the actual rates of alcohol dependence among Jews, either in the United States or in other parts of the world are unknown.

Explanations of Jewish sobriety go back at least as far as the German philosopher Immanuel Kant, who in 1798 theorized that Jews (like women and ministers) avoided drunkenness because their special position in European Christian society was based on the perception that they adhered to a religious law that dictated a higher code of conduct. Intoxication for a Jew would therefore be sinful as well as scandalous. Others have suggested that the traditional use of wine for religious ritual in Jewish life, rather than for hedonistic or social purposes, protects Jews from alcoholism.

In the 1950s, C. R. Snyder studied the drinking patterns of seventy-three Jewish men living in New Haven, Connecticut, and also analyzed data from Jewish and non-Jewish male college students. He concluded that SOBRIETY was a positive factor in Jewish identity, as opposed to drunkenness, which was associated with non-Jews. He also concluded that the greater the adherence to Jewish religion and its "ceremonial orthodoxy," the lower the alcohol problem risk. This finding has led many to theorize that those Jews who do develop alcohol problems are those who have rejected or left Jewish religious practices, abandoning their Jewish identity.

The finding that genetic factors may predispose to alcoholism has led to speculation that there may also be some hereditary protection for Jews. Dr. Y. D. Neumark and his colleagues in Israel studied sixty-eight Jewish families of male heroin addicts, 75 percent of whom also drank to excess. Using statistical methods they found evidence for a combination of genetic and environmental factors influencing the levels of alcohol use in family members. They also found that the presence of a specific gene (the ADH2*2 allele, a variant of the gene for an alcohol-metabolizing enzyme) was associated with lower alcohol intake in a comparison of fifty-three of the heroin-dependent heavy drinkers with a group of ninety-two Jewish male light drinkers. A 1991 study by Monteiro and colleagues found suggestive evidence that young adult Jewish males were more sensitive to the subjective effects of low levels of blood alcohol than were a control group of Christians. Although this finding awaits replication, the authors theorize that heightened sensitivity in Jews might either deter heavy drinking or help facilitate internal mechanisms for the control of alcohol consumption. Nearly all studies of Jewish sobriety concentrate on male subjects, leaving the applicability of these theories to women unknown.

In 1980, Dr. Sheila B. Blume and colleagues published a study of 100 Jewish members of ALCOHOLICS ANONYMOUS from the New York city area (58 men and 42 women). The subjects had been abstinent for an average of 4 years. The belief among clinicians that Jewish alcoholics would have a high rate of preexisting psychiatric illness (because they would have to be mentally ill to be so deviant from their cultural group) was found not to be accurate. The Jewish subject group generally resembled their fellow non-Jewish alcoholics in treatment and at Alcoholics Anonymous, with similar family histories of alcoholism, drinking histories, and rates of additional psychiatric diagnoses. They did differ in having an unusually high rate of dependence on prescribed psychoactive medications, a combined result of their attempts to obtain professional help and the frequent failure of their physicians to reach an accurate diagnosis. Although there was evidence of less adherence to orthodox Judaism later in life in these Jewish alcoholics, their subjective feelings of Jewish identity were strong and remained so throughout their alcoholism and recovery.

Many subjects reported that their families, their friends, their physicians and they themselves had dismissed the possibility that they might be suffering from alcoholism, because "Jews can't be alcoholics." They experienced great relief when they finally met another recovering person who was Jewish.

It is an interesting footnote to history that the great psychiatrist Sigmund Freud seemed to have accepted the idea of Jews' immunity to alcoholism. He once reassured a Jewish patient who expressed concern about his drinking by saying that alcohol would neither help nor harm him; alcohol was for the gentiles.

During the late 1970s and 1980s interest in helping Jewish alcoholics grew, both in the United States and in Israel. The Federation of Jewish Philanthropies, based in New York City, organized a

task force on alcoholism, which later extended its purview to all addictive diseases, including compulsive GAMBLING. In 1980, the Jewish Alcoholics, Chemically Dependent Persons and Significant Others Foundation, Inc. (JACS) was organized to serve as a forum for the sharing of recovery by Jewish addicts and their families. Both groups continue to educate the Jewish community and to encourage prevention, treatment, and the opening of synagogues and Jewish community centers to twelve-step groups such as Alcoholics Anonymous, NARCOTICS ANONYMOUS, Gamblers Anonymous, AL-ANON, Nar-Anon and Gam-Anon. In addition, JACS has sponsored the most extensive study of chemically dependent Jews and their significant others in the literature.

The literature on drug addictions other than alcoholism in the Jewish community has been less divided, because of an absence of long-standing belief concerning Jewish immunity to drug dependence. The New Haven study mentioned above found a lifetime prevalence of drug abuse of 1.3 percent in the Jewish adults, which did not differ significantly from the rates for the other religions or those reporting no religious preference. Nevertheless, denial of drug problems in many Jewish households and communities is an ongoing problem.

The JACS study collected information from 538 recovering Jewish alcholics, addicts, and significant others (i.e., those affected by the addiction of a family member or close friend). One hundred thirty seven of the subjects considered themselves both chemically dependent and significant others, 242 of the subjects were chemically dependent but not significant others, and the remaining 159 were significant others and were not addicted. Susan L. Vex and Blume reported that 71 percent of the chemically dependent subjects were dependent on more than one substance. Alcohol was the most prevalent drug of dependence. Alcohol was the primary drug of choice for 54.7 percent of the addicts and a secondary drug for 24.5 percent. The JACS data did not support the idea that alcoholism in Jews was a result of lack of education, poor income, alienation or loss of religious identity, as had been hypothesized earlier. As in the 1980 Blume et al. study, the male to female ratio was much lower than usually found in studies of alcoholism and addiction in the general United States population, but the significance of finding equal numbers of male and female alcoholics is not clear. Like the Jewish alcoholics studied twenty years earlier, the JACS subjects also reported that in their search for recovery they had found little help within the Jewish community, and felt that education of rabbis and Jewish leaders about addiction was of utmost importance.

Efforts to promote education, PREVENTION, and TREATMENT of other drug problems among Jews have gone hand-in-hand with the efforts to fight alcoholism—and have employed the same methods. Self-help fellowships based on the twelve steps of Alcoholics Anonymous can be helpful to alcoholics and to other addicts of the Jewish faith, even though the spiritual base of the twelve steps was originally adapted from the philosophy of a Protestant Christian movement. Several authors have published guides to the twelve steps as related to Judaism.

(SEE ALSO: *Ethnicity and Substance Abuse; Twelve-Step Programs*)

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SHEILA B. BLUME

JEWISH ALCOHOLICS, CHEMICALLY DEPENDENT PERSONS AND SIGNIFICANT OTHERS FOUNDATION, INC. (JACS) *See* Treatment Programs/Centers/Organizations: An Historical Perspective

JIMSONWEED A tall, coarse, poisonous plant that flowers, produces seed, and dies in one year. It belongs to the nightshade family (Solanaceae), and has foul-smelling leaves and large white or violet trumpet-shaped flowers. It produces round, prickly fruits. Jimsonweed (Datura stramonium) grows in several parts of the world. A strong intoxicant made from this plant was used by the woodland tribes of eastern North America. The plant was also used as an ingredient of wysoccan, an intoxicant employed in the puberty rites of Native Americans in what is now Virginia. Indeed, the name Jimson is another form of Jamestown, the English colony founded in Virginia in 1607.

Smoke from burning jimsonweed was breathed to relieve symptoms of asthma in India, and cigarettes containing jimsonweed have also been used for the same purpose.

As in other members of the Solanaceae family, the mind-altering substances are tropane ALKALOIDS, and the seeds and leaves contain up to 0.4 percent of these compounds. The principal alkaloid found in jimsonweed (also found in belladonna) is atropine. Atropine widens the pupils of the eyes, helps stop muscular spasms, lessens pain, and reduces bodily secretions. Large to toxic doses of atropine result in restlessness, irritability, disorientation, hallucinations, and delirium.

(SEE ALSO: *Plants, Drugs from; Scopolamine*)

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JUVENILE DELINQUENCY AND SUBSTANCE ABUSE *See* Adolescents and Drug Use; Crime and Drugs

K

KAVA A drink prepared from the root of the Australasian pepper shrub *Piper methysticum*. The word *kava*, which is Polynesian for bitter, pungent, is given to the drink because of its strong peppery taste. Several variations of this drink were once used widely as social intoxicants in the islands of the South Pacific, particularly Fiji. The quality of the drink improves with the age of the root, and the roots are generally at least four years old before they are used. After the root is cut and crushed or grated, the active components are extracted by soaking the preparation in water.

Common effects of kava include general muscular relaxation, euphoria, and loss of fatigue. Visual and auditory effects are also common. In large quantities kava can induce muscular incoordination and ultimately stupor.

While no ALKALOIDS or glycosides have been found in kava, several aromatically substituted α-pyrones, including kawain, dihydrokawain, methysticin, and yangonin, have been isolated from the extracted root. Other as-yet-unidentified components of kava may also be important in the effects of the drink.

(SEE ALSO: *Plants, Drugs from*)

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ROBERT ZACZEK

KHAT This is a shrub or small tree that grows wild and is largely cultivated in the uplands of Yemen and East Africa. The plant is known under many names; it is called *qat* in Yemen, *tschad* in Ethiopia, and *miraa* in Kenya. The botanical name is *Catha edulis*. Khat is a habituating stimulant containing ALKALOIDS released by chewing the leaves, buds, and sprouts. The leaves are about two to three inches long, with a serrated edge (see Figure 1), are brownish-green, somewhat leathery, and have a glossy upper surface. Since these plants lack more specific botanical features, a chromatographic test for their identification has been developed.

Use. Khat leaves can be made into a tea, but generally they are chewed for their stimulating effect. They are thoroughly masticated one by one; the juice is swallowed while their residue is stored in the cheek and later ejected. Young leaves are the most tender and potent; the leaves must be fresh to be effective. A portion is about 100 to 200 grams of leaves; they are predominantly consumed in a social setting. In Yemen, the habit is part of the cultural tradition and of great importance to social life; many houses have a room specifically arranged for the khat session, for which men meet almost every day. During the session, the group may also smoke from a water-pipe, and there is a supply of beverages. Khat use by women is less formal and much less frequent. In East Africa, khat use is more recreational in nature, with the leaves being consumed at times together with ALCOHOL or other

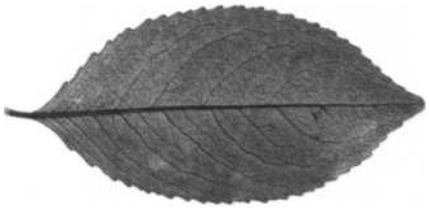

Figure 1
Full-grown Khat leaf (at about two-thirds natural size)

drugs. There is also a tradition of khat use by farmers and craftspeople, who chew it to enhance work performance and to stay alert.

Khat consumption has increased significantly during recent decades; it has been estimated that at present about 5 million portions per day are consumed. Although use is limited to the region where it grows, khat is now also exported by air to Europe and North America, where it is sold mainly to immigrants from Yemen and East Africa.

Effects. The pharmacology of khat has been reviewed and its effects are characterized by a moderate degree of central nervous system (CNS) stimulation, resulting in a state of mild euphoria and excitement, often accompanied by talkativeness to excess. High doses may induce restlessness and sometimes manic behavior. Excessive consumption may lead to toxic psychosis. Khat produces ANOREXIA (loss of appetite) and constipation; it has sympathomimetic effects on the cardiovascular system. Dilation of the pupil and staring are indicative of the acute effect of khat. Habitual chewing is usually revealed by a brownish staining of the teeth.

The effects are very similar to those of AMPHETAMINE, and the difference between the two drugs is quantitative rather than qualitative. Accordingly, habitual khat use may give rise to psychic dependence, which usually is moderate but often persistent. The withdrawal symptoms after prolonged use are slight trembling, lethargy, mild depression, and recurrent bad dreams. Khat use by the habitué is often compulsive, with the necessary supplies obtained at least once a day, even at the expense of vital needs; in the countries where khat use is widespread, the socioeconomic consequences of the habit are considerable.

Constituents. Khat contains the alkaloids norephedrine, cathine, and cathinone (see Figure 2). Norephedrine and cathine do not contribute significantly to the psychostimulant action, however, they are probably of importance for the sympathomimetic effects (on the autonomic nervous system). The constituent that is mainly responsible for the stimulant qualities and the dependence-producing effects of khat is cathinone. This ALKALOID must be considered a natural amphetamine, since the two substances have the same mechanism of action. However, cathinone has a half-life of only 1.5 hours, whereas that of amphetamine is much

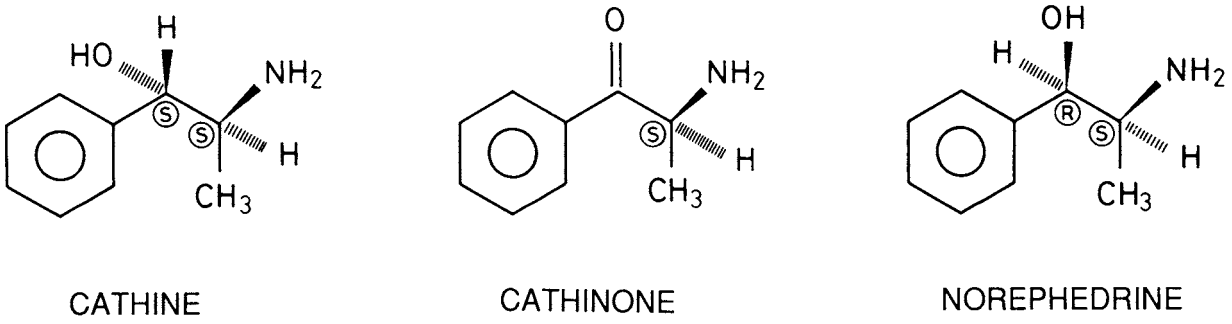

Figure 2
Structure of the Khatamines.
Cathine (S,S(+)phenylpropanolamine or (+)norpseudoephedrine, cathinone (S(−)alphaaminopropiophenone) and norephedrine (R,S(−)phenylpropanolamine). In an analysis of twenty-two khat samples of different origin, the average concentration of these alkaloids in 100 grams of fresh khat were found to be 120 milligrams, 36 milligrams, and 8 milligrams, respectively (Geisshüsler & Brenneisen, 1987).

longer. Since cathinone is absorbed gradually from the leaves during chewing and is inactivated in the body rather rapidly, the pharmacological effects of khat are usually limited.

(SEE ALSO: *Amphetamine*)

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PETER KALIX

KORSAKOFF'S SYNDROME *See* Alcoholism; Complications: Neurological

L

L-ALPHA-ACETYLMETHADOL (LAAM) Acetylmethadol (also referred to as *l*-alpha-acetylmethadol, methadyl acetate, LAAM or L-AAM) is structurally related to METHADONE. LAAM is a potent OPIOID agonist with properties similar to methadone, except for its prolonged half-life. This slow elimination can be useful clinically, since 50–80 milligram doses of LAAM given three times a week are equivalent to daily doses of 50–100 milligrams of methadone in preventing the symptoms of opioid WITHDRAWAL. Thus, addicts on maintenance treatment would need to come to a clinic only three times a week for LAAM instead of daily for methadone. Since the early 1970s, methadone has been the only agent approved for use in maintenance-treatment programs for HEROIN addicts, but research has shown that LAAM can be a useful alternative. In 1993, the U.S. Food and Drug Administration (FDA) initiated the legal changes needed to make LAAM available for clinical use.

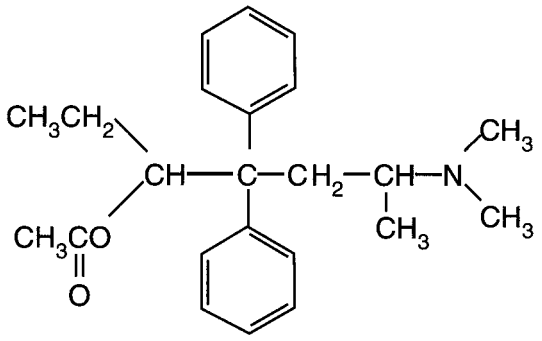

Figure 1
LAAM

(SEE ALSO: *Pharmacotherapy; Treatment*)

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GAVRIL W. PASTERNAK

LATIN AMERICA AS DRUG SOURCE *See* Bolivia; Colombia as Drug Source; International Drug Supply Systems; Mexico as Drug Source

LAUDANUM Laudanum refers to a tincture of OPIUM—an alcoholic extract (about 20%) of opium, which contains approximately 10 milligrams per milliliter of morphine. If used at all currently, it would be as an antidiarrheal. The solution is more concentrated than PAREGORIC, and smaller

Laudanum, Cistus ladanifer, *in flower.* (© Eric and David Hosking/CORBIS)

volumes are given; however, their actions are almost identical. At standard doses, they rapidly and effectively treat diarrhea without producing euphoria or analgesia. The solution does contain MORPHINE and other opioid alkaloids and, at higher doses, it can be abused—as it was during the late-nineteenth and early twentieth centuries, when it was sold widely as a tonic and cure-all, in shops, by mail order, and by traveling medicine shows. Laudanum use and abuse are often mentioned in novels and plays of and about the period.

(SEE ALSO: *Dover's Powder*)

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GAVRIL W. PASTERNAK

LAW ENFORCEMENT *See* Anslinger, Harry J., and U.S. Drug Policy; Coerced Treatment for Substance Offenders; Appendix, Volume 4

LD50 In preclinical studies, the LD50 is the median lethal dose—the dose of a drug that produces death in 50 percent of the experimental animals tested. The LD50 can be estimated from a dose-effect curve, where the concentration of the drug is plotted against the percentage of animals that die. The ratio of the LD50 to the ED50 (the median effective dose) indicates the therapeutic index of a drug for that effect and suggests how selective the drug is in producing its desired effects. In clinical studies, the concentration of the drug required to produce toxic effects can be compared to the concentration required for therapeutic effects in the population to esimate the clinical therapeutic index.

(SEE ALSO: *Research: Animal Model*)

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NICK E. GOEDERS

LEGALIZATION OF DRUGS *See* Policy Alternatives

LEGAL REGULATION OF DRUGS AND ALCOHOL Legal regulation can be used in four general ways to influence the incidence, prevalence, patterns, and circumstances of consumption of potentially harmful substances—including ALCOHOL, TOBACCO, and other DRUGS. The most direct mode of legal intervention is *to establish the conditions under which a potentially harmful substance is available.* In doing so, the law can employ either

(1) a "prohibitory" scheme that prohibits the production or distribution of the substance for nonmedical or self-defined uses, or
(2) a "regulatory" regime, which permits the substance to be lawfully available for nonmedical or self-defined uses but that may regulate the product, its price, and the conditions under which it is accessible.

A completely successful prohibition would prevent any nonmedical consumption of the proscribed substance; however, the more likely consequence of a prohibitory scheme is that an illicit distribution

system will arise to respond to whatever demand exists for the substance. In that case, the manner in which the prohibition is enforced can also influence the product, its price, and the conditions under which it is available.

A second mode of legal regulation is to *regulate the flow of information and messages regarding use of the particular substance.* The government may initiate its own informational efforts to influence attitudes, beliefs, and behavior. Government may also attempt to influence private communications, either by proscribing certain messages altogether or by regulating or restricting their content. Such restrictions have generally taken two forms—mandatory warnings and proscriptions of certain types of messages.

A third mode of legal control is the *direct regulation of consumption*, either by proscribing and imposing sanctions for undesired behavior or by withholding benefits or privileges to which the individual would otherwise be entitled. Thus, the law may proscribe use of a substance altogether, or it may prohibit such behavior in certain specified circumstances. Examples of total bans include unauthorized possession and consumption of controlled substances and consumption of alcohol by persons under the minimum age. Situational prohibitions include laws against consuming alcohol or smoking tobacco in public areas. Laws that require drug testing of workers and that permit job termination or discipline as a consequence of a positive test illustrate less coercive measures of deterrence.

A fourth use of the law emphasizes its declarative aspects. Whether or not a legal control has a direct impact on the marketplace or on the prevalence of the disapproved behavior, it may *symbolize and express the official government view of the behavior and may generate derivative effects on behavioral patterns by influencing attitudes and beliefs.* To the extent that citizens customarily defer to and respect the law or are influenced by messages of official approval or disapproval, a declaration of illegality may serve an educative, or didactic, role. Specification of a minimum drinking age, regulation of the availability of drug PARAPHERNALIA, and sanctions for possession of illicit drugs may all generate these symbolic effects, even if the direct effects tend to be modest.

AVAILABILITY

The National Commission on Marihuana and Drug Abuse identified four models of availability for psychoactive substances: The first involves no special controls at all; the substance is treated in the same way as other [unregulated] market commodities. Under the second approach, the substance is subject to special controls but remains lawfully available for self-defined [or nonmedical] purposes. The third model limits availability to specific purposes, generally to medical and research uses only. Under the fourth approach, the substance is not legally available at all except perhaps for narrowly circumscribed use in research. The first two models can be characterized as regulatory approaches (because the substance is legitimately available for nonmedical or self-defined purposes) and the second two as "prohibitory" approaches (because the substance is not available for self-defined or nonmedical purposes). Tobacco and alcohol are lawfully available for nonmedical uses, but they are subject to variable regulatory controls designed to affect the product, place, and conditions of consumption. (Only the solvents and INHALANTS—glue, lacquer, thinner, ether, gasoline, nitrous oxide—are essentially uncontrolled.) However, most psychoactive substances (legally denominated controlled substances) are subject to prohibitory controls; with the one minor exception of PEYOTE, which has been available to members of the Native American Church for sacramental uses—this means their availability is limited by law to medical and research uses.

Alcohol. The availability of alcohol is governed by alcoholic beverage controls (ABC) that vary from state to state. ABC agencies view their primary responsibilities as providing an orderly market for the distribution of alcoholic beverages, controlling criminal involvement in the market, and generating tax revenues. Since the 1960s, the trend has been to liberalize restrictions on access to, and availability of, alcohol in order to facilitate private choice, to protect commercial interests, and to raise revenue. Only since the late 1980s have some ABC agencies shown any inclination to use their regulatory authority to influence the prevalence pattern, and circumstances of consumption. Relevant aspects of ABC regulation include pricing and/or taxation policies, zoning, and rules regarding hours and days of sale.

Direct regulation, under the authority of ABC boards, is not the only method by which the law can influence the conditions under which alcohol is available. For example, one way to discourage retail sellers of alcohol from selling the substance to a person already intoxicated is to hold them legally liable for injuries subsequently caused by the intoxicated consumer, even after leaving the premises. Although the legal theory has changed over the years, the risk of liability for commercial suppliers under so-called DRAM SHOP LIABILITY LAWS is relatively well established. Moreover, the courts of several states have extended liability to the hosts of social events who served alcohol to "obviously intoxicated" guests who then cause injuries in their intoxicated condition.

Tobacco. For the most part, the public health dimensions of tobacco regulation have been reflected only in product, package, and advertising requirements designed to facilitate informed consumer choice. Only since the late 1980s has the federal government moved toward a policy that unequivocally establishes reduced consumption as its goal. Although a national prohibition is unlikely in the foreseeable future, several regulatory initiatives are being undertaken at all levels of government. For example, states will not receive federal money for mental health and substance-abuse services, unless they implement a plan for enforcing bans against the distribution of tobacco products to minors. Many localities have banned vending machines. In addition, several states have raised cigarette excise taxes with the aim of reducing consumption, and the federal excise tax has been increased by a substantial amount, with the dual aims of reducing smoking and raising revenue.

In 1996, the federal Food and Drug Administration (FDA) asserted jurisdiction over traditional tobacco products under the Food, Drug and Cosmetic Act, on the theory that tobacco products are intentionally marketed to satisfy consumers' addiction to nicotine. Based on this interpretation of the Act, the FDA adopted regulations prohibiting the distribution of tobacco products to minors and, as discussed below, restricting the marketing of tobacco products to youths. Although the U.S. Supreme Court ruled in 2000 that the FDA did not have jurisdiction over traditional tobacco products under existing law, it is only a matter of time before Congress confers such authority.

In addition, smokers or their survivors have sued tobacco companies, with mixed success, seeking damages for smoking-induced disease or death. In 1998 the major tobacco companies entered into a Master Settlement Agreement with the state attorney general, agreeing to pay $246 billion to the states over the duration of 25 years to settle lawsuits seeking to recover the states' costs of treating smoking-related diseases. Obviously, imposing liability on manufacturers for the adverse health consequences of smoking can have a major impact on the economics of the industry. In this instance, the indirect regulation of tobacco by the tort system has exerted a more potent influence on industry behavior than many direct regulatory alternatives, such as pricing policies, outlet limitations, or tar and nicotine limitations.

Controlled Substances. The manufacture and distribution of OPIATES, COCAINE, CANNABIS (MARIJUANA) stimulants, depressants, and hallucinogenic substances outside medical and scientific channels are unlawful under both federal and state "controlled substance" laws. The production and distribution of these substances within medical and scientific channels are subject to varied levels of restrictions based on their "potential for abuse" and their level of accepted medical use under the CONTROLLED SUBSTANCES ACT of 1970. The wisdom of these prohibitions, especially in relation to cannabis, has been questioned by some on the grounds that the suppression of nonmedical use is not a legitimate governmental objective, and if it is, that the costs of the prohibitions exceed the benefits of the reduced consumption they achieve.

A particularly controversial aspect of cannabis regulation has been its classification as a Schedule I drug under the Federal Controlled Substances Act and its state counterparts. Schedule I is the most restrictive classification, reserved for drugs without any accepted medical use. Critics of the law have argued that marijuana is medically useful to treat glaucoma, AIDS wasting syndromes, and other conditions, and several states have adopted laws that aim to legitimize bonafide medical uses under state law. These laws have created the unusual situation in which any effort to make marijuana available for medical uses could be prosecuted as a violation of federal law. The Institute of Medicine of the National Academy of Sciences has identified promising avenues of therapeutic use for the active

constituents of cannabis and has recommended further research.

INFORMATION REGULATION

A government aiming to discourage what it perceives as unhealthy or unsafe behavior is not likely to be satisfied with the influence of its own messages and may seek to regulate communication by others within the bounds of the First Amendment, which protects freedom of speech. This can be done in two ways. First, the government may require individuals or organizations to convey the government's desired message. Laws requiring product manufacturers to include information on or with their products have become a standard feature of health and safety regulation. In recent years, mandatory package warnings have been utilized as a means of informing consumers about the dangers of tobacco and, more recently, of alcohol use. Second, government may ban communication of messages that it regards as undesirable. For example, laws banning false or misleading advertising are common, but government may choose to go a step further—to suppress a message because it is thought to encourage unhealthy or socially disapproved drug, alcohol, or tobacco-using behaviors. Examples include the federal ban on broadcast advertising of cigarettes and state laws that ban alcohol advertising. Public-health advocates have urged the federal government to prohibit all forms of tobacco advertising. Whether such prohibitions actually affect the level of consumption (as opposed to product choice) remains controversial. The FDA's 1996 Tobacco Rule, which was invalidated by the Supreme Court in 2000, would have restricted the advertising of tobacco products to a text-only format, and would also have banned other forms of promotional activity that are thought to make use of tobacco products attractive to children and adolescents. The tobacco companies agreed to abide by some of these marketing restrictions in the Master Settlement Agreement executed in connection with the suit brought by the attorney generals of these states.

Proposals have also been made to move beyond advertising into the area of entertainment programming, eliminating messages that portray smoking and drinking in an attractive way. Clearly, such initiatives would raise serious constitutional questions concerning free speech.

Governments have also occasionally attempted to purge the environment of messages that are thought to encourage illicit drug use. For example, one provision of the Model Drug Paraphernalia Act (drafted by the federal drug enforcement agency as a model for states to enact) specifically bans paraphernalia advertising. In 1973, the Federal Communications Commission (FCC) threatened to revoke the licenses of radio stations whose lyrics were thought to encourage illicit drug use.

DIRECT REGULATION OF CONSUMER BEHAVIOR

A decision to discourage nonmedical drug use—and to proscribe transactions outside medical channels in order to restrict availability for such use—does not necessarily entail a decision to proscribe and punish unauthorized consumption. Values of individual freedom weigh very differently in the two contexts.

From the perspective of libertarian philosophy, it has been argued that the criminalization of private use (and possession for such use) of drugs is categorically illegitimate, and the criminal prohibition should be limited to behavior that endangers others. This, also leads to a discussion of the ways in which drugs might affect others. Even if criminalization is not categorically objectionable, the costs of it may exceed the benefits. The National Commission on Marihuana and Drug Abuse relied on such a cost-benefit assessment in 1972 when it recommended the decriminalization of possession of marijuana for personal use. A few states have decriminalized the possession of marijuana, although they have usually substituted a civil fine. Some of the states that took this action subsequently recriminalized the possession. Aside from marijuana, possession of all other controlled substances is a criminal offense in all states as well as under federal law. In addition, the possession of alcohol by underage consumers is an offense in most states. Even if the possession or use of a substance is not categorically proscribed, prohibitions can be utilized to deter and punish socially harmful behavior or to provide leverage to coerce individuals into treatment. Public smoking laws and laws prohibiting driving while intoxicated (or while having a certain level of blood alcohol content) provide the prime examples.

DECLARATION ASPECTS OF LEGAL REGULATION

Government sends messages by its actions as well as its words. By declaring conduct illegal or by using any of the other instruments of legal intervention described above, the government expresses and formalizes social norms. However, knowledge of the official preferences may actually encourage the disapproved behavior among disaffected, outsider groups. Measuring such symbolic effects is difficult because of the need to isolate these hypothesized effects from other influences on attitudes and beliefs.

Arguments drawing on the declarative aspects of legal regulation are routinely employed by proponents of restrictive controls over the availability and consumption of alcohol, tobacco, and other drugs. Criminal sanctions against the simple possession of controlled substances are frequently regarded as indispensable symbols of social disapproval. Such arguments have been prominent in debates concerning the decriminalization of possession of marijuana. Moreover, graded or stratified penalty schemes, which punish the possession of "more harmful" drugs more severely than that of "less harmful" drugs, may be favored because they denote the relative seriousness of these transgressions. Public SMOKING bans and antiparaphernalia laws seem to be particularly designed to reinforce attitudes unfavorable to smoking and recreational drug use.

Statements of legal rules can serve an educational role even if they do not penalize the undesired behavior. Minimum-drinking-age laws (which prohibit the distribution of alcohol to youth) provide a good example because they denote the norm even if the youthful drinker is not punished. Similarly, bans on alcohol or tobacco advertising might be enacted to erase a possible symbol of social approval even if the proponents did not believe that such bans would directly reduce consumption.

(SEE ALSO: *Advertising; Alcohol; Dram Shop Liability Laws; Minimum Drinking Age Laws; Opiods and Opiod Control; Policy Alternatives*)

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RICHARD J. BONNIE

LE PATRIARCHE Le Patriarche is both the name of an organization treating addicts and the nickname (the "patriarch") of its founder, Lucien J. Engelmajer. The program was begun in Toulouse, France, in 1972, and by 1989 it had spread to Spain, Belgium, Italy, Germany, Portugal, and Ireland.

Le Patriarche focused its work in residential treatment centers, located primarily in rural areas on large farming estates. In addition, the organization operated small intake and community-interaction units in urban centers throughout Mediterranean Europe. During the 1990s, it also opened several centers in large cities in the United States, but most of these closed after several years.

The program's philosophy is vague: remain drug-free and work hard in a semi-Utopian setting. It offers little in the form of organized therapies with measurable outcomes.

Almost from the start, Le Patriarche was embroiled in controversy. Addicts were made to work

on projects owned by Engelmajer without salary. Some individuals were relocated from one European country to another while their passports were withheld. Use of force was not uncommon. The organization used illegal immigrant labor for commercial activities.

After a finding of fraud by the French government, Le Patriarche tried to reorganize to gain credibility. However, the organization in Italy with which it began to associate—San Patrigiano—was surrounded by similar controversy. Eventually, Engelmajer was removed from visible leadership, and members of his family now direct the program, renamed Dianova. The headquarters are located in Switzerland, where the finances for all locations are controlled.

Dianova continues the practice of not paying wages for what in essence is forced labor, thereby perpetuating Le Patriarche's structure of creating dependence among the addicts who seek help there.

In the early 1990s, worldwide membership was about 10,000. There is no reliable information about the current number of participants in Dianova.

DAVID A. DEITCH

LIBRIUM *See* Benzodiazepines

LIFE SKILLS TRAINING *See* Prevention; Prevention Programs

LIMBIC SYSTEM The limbic system is a group of BRAIN STRUCTURES organized into a functional unit that is important in the expression of emotion and mood states. The term *limbic lobe* and associated terminology can be traced to the French neuroanatomist Paul Broca (1824–1880), who used it first to describe the forebrain structures that encircle the brain stem. The *limbic system* is a broader classification, composed of brain structures that form an integrated circuit surrounding the thalamus—an important relay station between higher brain centers and the hind brain and spinal cord.

The limbic system is thought to be important in emotional behaviors. This was hypothesized on the basis of neuropathological investigations of the brains of individuals displaying bizarre emotional disturbances. These initial clinical observations were followed by animal studies, in which the loss of these structures produced significant changes in emotional responsiveness. As research techniques and methodologies were refined, it became clear that limbic structures had an important and complex role in the expression of behavior. It is now believed that these structures are involved in a number of significant behavioral processes. In particular, the limbic system and related structures are thought to be important in the expression of emotion related to euphoria and feelings of well-being. For these reasons, the limbic system may have an important role in drug abuse.

LIMBIC SYSTEM COMPONENTS

The limbic system that surrounds the thalamus provides an interface between the midbrain and higher cortical structures. The general structure and components of the limbic system are shown in Figure 1. These include the AMYGDALA, the NUCLEUS ACCUMBENS, the olfactory tubercle, the septal nuclei, the hippocampus, the hypothalamus, the cingulate cortex, and the frontal cortex. As can be seen in the figure, these structures are positioned between the brain's major relay station—the thalamus—and higher cortical structures. The separate components of the limbic system are interconnected such that activity initiated in one structure affects other components. One of the hypotheses about the basis of emotion speculated that reverberating neuronal activity in this system was responsible for affective behaviors. Initial animal studies using either direct electrical stimulation or lesions (loss) of various components of the limbic system substantiated the important role of this system in behavior.

THE ROLE OF THE LIMBIC SYSTEM IN BEHAVIOR

Electrical stimulation or the destruction (lesions) of components of the limbic system alter behavioral processes. Lesions of the hippocampus disrupt memory processes, whereas lesions or stimulation of the amygdala affect emotional behavior and feeding in a manner similar to manipulations of the medial and lateral hypothalamus. Stimulation of the lateral hypothalamus produces aggres-

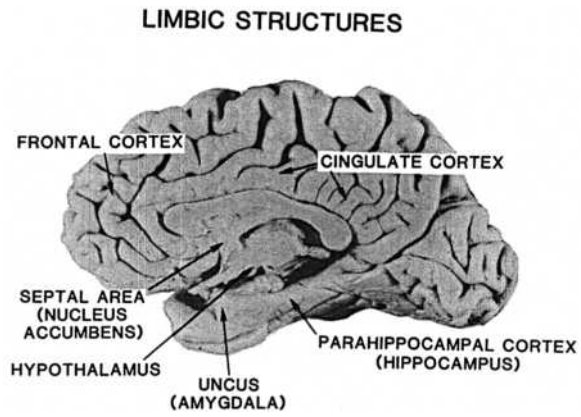

Figure 1
The Limbic System—composed of structures generally located between the brain stem and higher cortical structures. Some of these components are labeled in this sagittal section of the brain. The structures in parentheses lie behind the structures listed above them. The hypothalamus, hippocampus, septal nuclei, nucleus accumbens, amygdala, cingulate cortex, and frontal cortex are components of the limbic system that may have an important role in drug abuse.

sive responses, whereas lesions of this area produce a placid behavioral profile. In contrast, lesions of the medial hypothalamus produce a highly excitable and aggressive pattern of behavior, whereas lesions of the amygdala result in placid and nonaggressive behavior. Early studies found that lesions of the lateral hypothalamus can decrease feeding, whereas lesions of the ventromedial region produce excessive levels of feeding resulting in obesity. Recent experimental studies have demonstrated the complex nature of the involvement of hypothalamic cells in feeding and drinking; however, like most complex behaviors, the mechanisms that control hunger and satiety are not simply located in a single brain center.

Some structures of the limbic system are important in REINFORCEMENT processes. The term *reinforcement* applies to processes perceived as rewarding or good, which therefore are repeated, such as electrical self-stimulation. For example, animals will repeatedly emit a response that leads to the delivery of brief electrical stimulation of small electrodes that are implanted in selected brain structures. Humans will also choose to stimulate many of these same brain regions and report positive feelings of well-being and euphoria. The limbic system sites that produce these effects in animals include the lateral hypothalamus, nucleus accumbens, frontal cortex, cingulate cortex, and the brain-stem nuclei believed to be part of the limbic system—these include the substantia nigra and ventral tegmental area, which both contain DOPAMINE neurons that send inputs to many limbic-system components. Measures of brain-glucose metabolism, which directly reflect brain-cell activity, have been used to determine the involvement of specific brain regions in animals electrically self-stimulating three of these brain regions. The stimulation of each of these regions produced significant activation of several limbic-system structures that included the nucleus accumbens, amygdala, hippocampus, and the frontal and cingulate cortices. This area of investigation has led neuroscientists to propose that there are brain circuits dedicated to the behavioral processes related to reinforcement. Drugs of abuse likely produce their positive effects through the activation of these brain circuits.

THE ROLE OF THE LIMBIC SYSTEM IN DRUG ABUSE

A large number of experiments have focused on identifying the brain circuits that mediate the reinforcing effects of abused drugs, because the reinforcing effects are responsible for drug abuse. These experiments have included the use of drug self-administration techniques and sophisticated neurochemical procedures to measure the involvement of specific NEUROTRANSMITTER systems. As of the early 1990s, evidence indicates that limbic structures and brain cells that project to limbic structures play an important role in these processes. It is clear that dopamine-containing neurons that project from the ventral tegmental area to the nucleus accumbens have a critical role in the reinforcing actions of COCAINE and AMPHETAMINE. Removal of these inputs with toxic agents that selectively destroy dopamine-releasing brain cells disrupts intravenous self-administration of these drugs. Additional evidence of the importance of this region in drug abuse comes from glucose-utilization studies. The levels of glucose metabolism are significantly elevated in a number of limbic structures in animals self-administering co-

caine intravenously. Other experiments have directly shown dopamine levels in the nucleus accumbens to be increased in animals intravenously self-administering cocaine. Collectively, these data imply an important role for the limbic system in general and specifically for dopamine neurons in the limbic system tied to the brain processes involved in stimulant abuse.

The brain circuits involved in OPIATE reinforcement appear to be very similar to those mediating cocaine self-administration. Limbic structures are clearly implicated in opiate reinforcement, but a central role for dopamine is less obvious. Significant changes in the utilization of some chemicals (neurotransmitters) involved in transmission between brain cells have been shown in the nucleus accumbens, amygdala, and the frontal and cingulate cortices of animals intravenously self-administering morphine. However, loss of dopaminergic inputs to the nucleus accumbens does not affect drug intake, whereas a similar loss of serotonergic inputs does. Similarly, nucleus-accumbens dopamine does not appear to be elevated in animals self-administering heroin as it is in animals self-administering cocaine. However, evidence does indicate an important role for limbic structures and chemicals used to communicate between cells of the limbic system in opiate reinforcement.

Limbic structures also appear to be important for ethanol (drinking ALCOHOL) reinforcement. The levels of dopamine appear to be elevated in the nucleus accumbens of rats orally self-administering alcohol. Injections of drugs that antagonize dopamine directly into the nucleus accumbens decrease alcohol self-administration, whereas drugs that enhance dopamine action increase alcohol intake. In addition, animals will self-administer alcohol directly into the ventral tegmental area—an area that contains the cell bodies for the dopamine cells that send inputs to the nucleus accumbens. These data collectively indicate that the nucleus accumbens and dopamine-releasing inputs to the nucleus accumbens are important to alcohol reinforcement.

CONCLUSION

The limbic system plays an important role in behavior. These brain structures appear to be central to the processes that mediate the reinforcing effects of electrical-brain stimulation and of several highly abused drugs. The nucleus accumbens appears to be a structure central to the reinforcing properties of cocaine and amphetamine, but it appears less important to opiate and alcohol reinforcement. A more exact definition of specific neurochemicals and brain-cell pathways in the limbic system that are involved in drug abuse will become clearer as new methodologies are developed.

(SEE ALSO: *Neuron; Neurotransmission; Research.*)

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STEVEN I. DWORKIN

LIQUOR *See* Alcohol; Distillation; Distilled Spirits

LSD *See* Lysergic acid diethylamide and psychedelics

LUNG DAMAGE *See* Crack; Marijuana; Nicotine; Tobacco

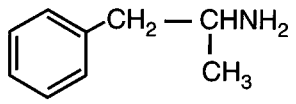
LYSERGIC ACID DIETHYLAMIDE (LSD) AND PSYCHEDELICS LSD is the abbreviation for lysergic acid diethylamide. It is the most potent member of a group of hallucinogenic substances called the indole-type HALLUCINOGENS. These drugs have structural similarities to another indole, the neurotransmitter SEROTONIN.

HISTORY

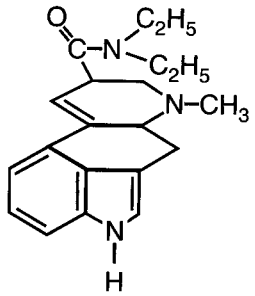
LSD was originally synthesized at the Sandoz Pharmaceutical Company, in Switzerland, as part of a long project begun in the 1930s. The aim was to develop useful medications that were derived from ergot, a fungus (*Claviceps purpurea*) that infects such grasses as rye. Some of these compounds were found to be useful in medicine—such as methysergide, for the treatment of migraine headaches, and ergotamine, which is widely used in obstetrics to induce contractions of the uterus and stop bleeding after the delivery of a baby. These medications do not have hallucinogenic properties.

The chemist in charge of this drug development project was Albert Hofmann. In 1943, he synthesized a compound he called LSD-25, since it was the twenty-fifth compound made in this series of ergot derivatives. He accidentally ingested some of it and within forty minutes had the first LSD "trip." He told his colleagues he was not feeling quite right and got on his bicycle to go home. Later, he carefully described the vividly clear flood of perceptions that are characteristic of the "mind manifesting" or psychedelic drug. This, then, was a complete surprise. Thereafter, the drug and various substitutions of different atoms on the basic molecule were extensively tested for medical uses in the late 1940s and in the 1950s. No specific medical use of LSD or its psychedelic variants has been found.

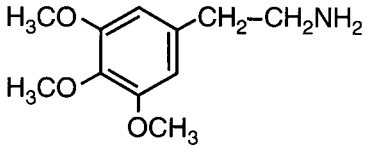
Because of its potency and the extensive reports of laboratory studies in animals and in the clinic, LSD has become the prototypical hallucinogen, or psychedelic drug. It also became the emblem of a social movement—which, in fact, was a confluence of various movements that had begun in the early 1960s; they peaked in the late 1960s. By 1973, the "acid culture" had subsided into a small but still active subculture of various psychedelic drug devotees seeking meaning and profound insights. The feeling of a "great discovery" about such drugs and the human mind had occurred as early as the nineteenth century; artists and writers, such as Baudelaire and Rimbaud in Paris, had discovered HASHISH and the altered, somewhat dreamy, states of consciousness and euphoria produced by this potent form of MARIJUANA—the active ingredient of which is TETRAHYDROCANNABINOL (THC). For a period, they became absorbed with hashish and wrote about its alluring effects. The drug scene



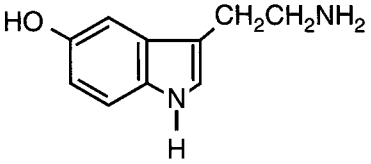
AMPHETAMINE
(psychostimulant)



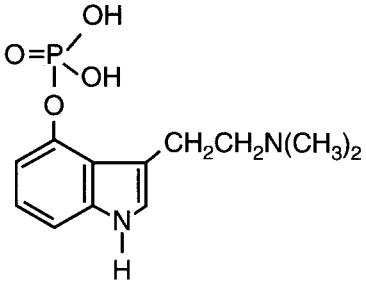
LSD



MESCALINE



SEROTONIN
(neurotransmitter)



PSILOCYBIN

evoked the promise that the human mind must contain remarkable powers. Toward the beginning of the twentieth century, MESCALINE, the active hallucinogenic compound in the PEYOTE cactus, similarly was tried by a few explorers in medicine and in the arts. In New York City, during the early part of World War I, many influential people and intellectuals took either peyote "buttons" (the dried tops of the peyote cactus) or mescaline (the synthesized active ingredient of the buttons) and called it a "dry drunk." Similarly, after World War II, LSD caused a flurry of excitement among some professionals, and its medical value was tested in psychiatric patients. Writers such as Aldous Huxley wrote exciting books about the effects of mescaline and, later, LSD—yet there was still no widely popular movement until 1960.

Then Timothy Leary, a young psychology instructor at Harvard, explored the Mexican or "magic" mushroom, *Psilocybe mexicana*, and its active ingredient, PSILOCYBIN—and later LSD—claiming criminals became loving and peaceful and others more creative. He popularized this on campus and, when he was not reappointed to the faculty, proclaimed himself to be a martyr to his cause. Between 1960 and 1966, the media repeatedly "discovered" LSD—in effect, advertising it. As publicity increased, subcultures experimenting with mushrooms and LSD grew up in the East and West Coast cities. Musicians, rock music, the hippie lifestyle, "flower children," and many in the various protest movements against the Establishment and the VIETNAM War were loosely joined to Leary's attempt to lead affluent and middle-class youth. Well-publicized festivals celebrated LSD and marijuana, such as the Summer of Love in the Haight-Ashbury section of San Francisco. Leary's challenge was for youth to "turn on, tune in and drop out" with acid. As more and more youth were curious to try experiences their parents had never dreamed of, rebellion led not only to acid experiments but to extensive POLYDRUG ABUSE—the extensive use of marijuana and various street substances. Either LSD or some variant and even heroin were tried. In addition, the search for new drugs with different and improved characteristics (more or less euphoria, hallucinogenic activity, or stimulant properties), literally hundreds of so-called DESIGNER DRUGS were synthesized (DOM, MDMA, DMT, etc.). Because any drug can have bad effects, the unsupervised use of all of these compounds led to frequent "bad trips" (which fundamentally were panic reactions) that brought people to emergency rooms. This generated widespread concern that all American youth (and, later, those in Europe) would become dreamy and "way-out acid heads." In 1966, the Sandoz Laboratories ceased distribution of the drug because of the often-exaggerated bad reactions and the public concern. As the claims for enduring LSD insights proved transient, research with LSD in humans essentially stopped.

Thus, one of the ways people use the effects of drugs that seem to enhance the clarity of mentation (mental activity) and perception (while not producing confusion, dreamy-euphoria, or oversedation) is to become absorbed in periods of intense exploration with a few others "in the know." Those with such inside information form a kind of cult and then advertise, but they eventually see some bad effects (the wrong people taking the drug in the wrong circumstance with unfortunate consequences) and sooner or later see little real use for the drugs. The minor or major epidemics then die down, only to recur as later generations rediscover the compounds.

EFFECTS

LSD is one of the most potent hallucinogens known; one-billionth of a gram of LSD per gram of brain produces profound mental changes. Although subjective effects occur in some individuals after doses as low as 50 micrograms, typical street doses range from 10 to 300 micrograms—street dosages vary widely. Misrepresentation also frequently occurs; someone will try to purchase synthetic TETRAHYDROCANNABINOL (THC), the active ingredient of marijuana, and receive LSD. Thus, the intake of LSD can be accidental as well as intentional, and the lack of quality control in illicit supplies is a hazard. Because of its high potency, LSD can be applied to paper blotters or the backs of postage stamps from which it is dissolved for consumption. Unsubstantiated reports of LSD added to stick-on tattoos for young children have caused alarm, even though absorption through skin would be far too slow to deliver enough drug to the brain to produce and sustain a trip.

The absorption of LSD from the gastrointestinal tract and other mucous membranes occurs rapidly, with drug diffusion to all tissues, including brain.

The onset of psychological and behavioral effects occurs approximately 30 minutes after oral administration, peaks in the next 2 to 4 hours, depending on the dose, with gradual return to normal by 10 to 12 hours. The first 4 hours after a 200-microgram dose are called a trip. In the next 4 to 8 hours, when over half the drug has left the brain, the "TV show in the head" has stopped. At this point subjects think the drug is no longer active, but later they recognize that they, in fact, had paranoid thoughts and "ideas of reference" in the last 4 to 8 hours of the trip. This simply means that there is the feeling of being at the center of things, being hyperalert, and having a conviction that everything going on refers to oneself. This is a regular but little publicized aftereffect, which finally dissipates 10 to 12 hours after the dose.

From 12 to 24 hours after the trip, there may be some slight letdown or feeling of fatigue—as if one had been on a long, steep roller coaster ride. After these intense and even frightening moments, the ordinary world might for a time seem drab. There is no craving to take more LSD to relieve this boredom; one trip usually produces satiation for a time, although some may want to repeat the experience. Memory for the events during the trip is quite clear. Those who revisit the experience sooner or later decide they have learned what they can and go on with the practical, daily affairs of living. In one experiment on CREATIVITY, subjects received either LSD or the stimulant amphetamine during a period of pleasant surroundings and music. The only difference between the two groups six months later was a slight tendency for those who had received LSD to buy more recordings! So the promise of lasting insight or creativity was not kept.

Drugs that make one feel different—alcohol being typical—can signal a "holiday from daily reality." The way the effects of such drugs are interpreted is critical. BEER at the Super Bowl means "loudly letting go" and champagne at the White House means a time for graceful speech and feelings. Thus personal and social expectations (called *set*—or how one is set to go) and the surroundings (called *setting*) have much to do with the ultimate effects of drugs. This is distinctively and especially the case with psychedelics. Thus when the chemist Albert Hofmann first ingested the active ingredient of the Mexican mushroom, psilocybin, the perceptions capturing his attention were related to Aztec symbols and art! For some, therefore, the trip may simply be funny and odd—for others it will have special meanings. Set and setting partially determine the character of such trips.

Fundamentally, LSD produces a heightened clarity and awareness of sensory signals—of sights, sounds, touch, lights, and colors. Similarly there is special significance given to thoughts, memories, or verbal interchanges. For example, gestures or inflections of speech or many cues that are normally in the background are felt to be more important than what is being said or usually meant—and in looking at a picture, the central figures may take on a life of their own, the small background details that are normally ignored emerging, capturing attention.

While awareness is strikingly increased, control over what is being attended to is weakened. For all these reasons, unstable surroundings or confused motives at the time of drug ingestion may lead to a less-controlled trip or even a panic-generating trip. Many are aware that the trip is not quite real and fundamentally feel as if they are "spectators" of what they are so intensely experiencing. Many rely on guides, a group, or the rhythm of music to carry them through this period of altered perceptions in which control is diminished. Thus, personal intent and reliable surroundings are major factors affecting the different kinds of experiences that people will have.

While every trip has an individual characteristic, there are regularities in the trips. This has been called a "march of effects" following drug ingestion. Thus, observers note, the first sign of feeling different is like "butterflies in the stomach" or a slight nausea and feeling of "whoops, here we go" as if on a roller coaster. Parts of the body simultaneously feel strange or different. At about the same time (30–40 minutes after drug ingestion), the cheeks are slightly flushed and pupil size begins to increase, maximizing within an hour or two. These changes are due to the effects of LSD on the sympathetic and parasympathetic nervous systems. The pupils react normally but are enlarged. After 4 hours they slowly begin to return to normal size, which finally is achieved at 10 to 12 hours after taking LSD. At the beginning of the trip, all soon note that what is at the periphery of their vision suddenly seems as clear as what is normally at the center of vision. Over the next 90 minutes, there is a feeling that tension is welling up. Laughing or crying will relieve the tension. Often subjects say they

Dr. Timothy Leary (center) in the custody of U.S. customs officials in New York City, October 11, 1966. Leary had been arrested under a section of federal law prohibiting users of narcotics or convicted narcotics violators from leaving or re-entering the U.S. without permission. (© Bettmann/CORBIS)

are laughing because of what they see or crying because of their feelings. But this is simply based on a need to relieve the fluctuating rise of tension. The trip moves on into the second and third hours when perceptual fluctuations and intensities are mainly noted. People also report perceiving several feelings simultaneously. A common observation is, "I don't know if I'm anxious, thrilled, or terrified." Just as perceptions are in flux, so are feelings, and these feelings and emotions may capture center stage in the second and third hours. Throughout the trip, people feel as if they are on the brink of an exhilarating but also dangerous experience. This intensity dies down about 4 hours after the usual dosage. If very large doses of LSD (500–1,000 micrograms) are taken, there is less capacity to be a spectator and far more intense self-absorption and fear. Some call this "dying of the ego" and relate the experience to mystical versions of death and rebirth.

Since the familiar seems novel and is seen in a different way, specialists in perception have been interested in what is called the "breakdown of constancies" that occurs with the drug. Normally we correct for what the retina sees by putting the world into order. We usually suppress the nonessential and focus on what we need to do to get about during the day. Just as with a camera, the retina sees the hand placed 6 to 8 inches in front of the eye as large. But the brain corrects for it and keeps size constant. Under LSD, corrections for constancy do not seem to happen. Many sensations that are normally dampened can thus have free play under the drug and the world will seem far less regular than it does in daily life.

One of the aftereffects in some—clearly not all—people is called "flashbacks." Days, months, or years after tripping, with no particular trigger or with an intense sensation, there may be a sudden few minutes in which subjects feel like they are back under the drug. They also may see flashing lights and other optical illusions. These flashbacks may be very disturbing. Flashbacks can occur after only a single drug experience and unpredictably. There has been no explanation as to why or how flashbacks occur. Scientists cannot predict (by observing a trip) if flashbacks will later occur or who is vulnerable. While these aftereffects are upsetting to some, most people do not experience them or those that do are not bothered. Others simply observe that their dreams may be more intense for a time after the drug experiences. One scientist noted that riding on a train to work, he was distracted from focusing on his newspaper for several months by the telephone poles whizzing by. These were normally at the periphery of his attention as he was reading, but after LSD, he could no longer suppress this irrelevant detail. There were more reports of such phenomena after publicity about them; given the millions of trips with LSD, these aftereffects are certainly infrequent but not rare.

Perhaps the most alarming bad effects of the drug have been the panic states occurring during a trip. Native Americans note that if one is in conflict, the effects of mescaline during religious ceremonies are unpleasant and can evoke terror. They then pray with the panicked person and "talk him down." One cannot predict whether a panic experience will occur. "One good trip does not predict a second one" is the general wisdom concerning this risk. Higher doses lead to less control and more intense effects, but panic states can occur at doses as low as 75 to 100 micrograms. For those who might be at risk for other mental disorders, hallucinogenic experiences may often destabilize them and precipitate some form of mental illness. For others, the experience may lead to a subsequent absorption with the unreal ("dropping out"), rather than coping with the challenges that the

tasks of the ordinary world present. Occasional suicides or rare impulsive acting out of odd ideas arising during a trip have led some to loss of control and tragedy.

For most, the experiences have few negative or positive aftereffects. Although it has often been suspected, no permanent change to the cells of the brain (brain damage) has ever been scientifically established. There is no generally accepted evidence that the drug produces chromosomal abnormalities or damage to a developing fetus (although no nonprescription drugs during pregnancy is the only safe rule to follow). The bad effects of a period of diminished control are unpredictable, and in that fact lies the real risk. Thus, it is the intensity of the experience and how well or poorly it can be managed, the unpredictable flashbacks, and how this "TV show in the head" or this "waking dream" gets woven into one's subsequent life that are at issue when hazards are considered.

TOLERANCE

One striking feature of LSD, mescaline, and related psychedelic drugs is tolerance, which is a loss of typical drug effects after repeated doses. In brief, with daily doses the duration and intensity of effects rapidly diminish to the point where no subjective effects are perceived. After 200 micrograms per day of LSD, there is simply no detectable drug effect on the third or fourth day. After three or four days without LSD, the full initial effects can be triggered by the same dose that has been "tolerated." Thus tolerance develops and dissipates rapidly. When subjects are tolerant to LSD, the usual dose of mescaline required for a trip is also no longer effective. This is called cross-tolerance. It is readily seen with similar dosage schedules of psilocybin, LSD, and mescaline. There is no cross-tolerance with the nonhallucinogenic stimulant drug amphetamine. Thus, there must be some common mechanism of action among the psychedelic drugs beyond their structure and similar array of mental effects.

Tolerance is seen both in humans and laboratory animals. The lack of pupil enlargement is a common sign of tolerance. In animals, some drug effects show tolerance and some do not. For example, a heightened sensitivity of rats to mild electric shock persists after daily doses and does not show tolerance. Such persisting drug effects during periods of tolerance have not been studied in humans. How and why a psychedelic drug loses and regains its potency in this fashion is not yet understood, but there is no withdrawal discomfort after stopping a psychedelic drug when it has been taken over several days. This differs from the classic effects described for opioid drugs, where an uncomfortable withdrawal with drug cessation requires more drug for relief. Such physical drug withdrawal phenomena are not found with psychedelics.

LSD AND SEROTONIN

LSD is known to affect many places in the brain where the body's neurotransmitter serotonin naturally has actions and effects, and the biochemical effects of LSD in the brain are mostly linked to those sites related to serotonin. LSD acts as a kind of impostor at receptors that recognize serotonin. LSD is like serotonin but different. Thus with LSD, the receptor signals other parts of the brain that there is too much serotonin, and these parts of the brain respond by tuning down cells that make serotonin. Yet, in fact, the chief effect of LSD is to cause *less* serotonin to be released in the neighborhood of the receptor—rather than too much, there is too little. This is one example of how LSD miscues the systems governing the flow of information between various brain neurons. In fact, overloading the brain with serotonin can reduce the LSD effect, and diminishing brain supplies of serotonin will increase LSD effects. Yet serotonin itself does not cause the scrambled perceptions that LSD does. How this miscue by LSD leads to the vivid effects is still unknown.

LSD, other indole-type psychedelics, and many hallucinogens related to mescaline (but surprisingly not mescaline itself) are known to act especially at a subtype of the serotonin receptor called the $5HT_2$ receptor. In laboratory animals, daily doses of LSD or psilocybin lead to fewer of these receptors, an effect that would be expected to produce tolerance; however, with 3 or 4 days off the drug, the number of $5HT_2$ receptors returns to normal. Both LSD and mescaline act at certain brain neurons, such as the locus coeruleus, and make it more responsive to inputs from the environment—such as a pinch. Researchers speak of such effects as lowering the gates to sensory input. We know the ways by which LSD affects certain

brain systems but still far less than we need to know to explain the full panoply of effects.

Although many of the psychedelic drugs are known to interact with serotonergic $5HT_2$ receptors, and this interaction appears to be of critical importance in producing their hallucinogenic effects, the hallucinogenic drugs can bind to a subtype of serotonin receptors that is located on serotonin nerve-cell bodies and on their terminals (which release serotonin that goes to the adjacent nerves with $5HT_2$ receptors). Interactions with these various receptors can lead to changes in the firing rate of such cells. The designer drugs MDMA and MDA cause the release of both dopamine and serotonin, effects that might contribute to their psychostimulant properties. The differential interactions of the various hallucinogens with multiple sites and systems may underlie the qualitative differences in the experience they produce.

(SEE ALSO: *Cults and Drug Use; Hallucinogenic Plants; High School Senior Survey; Plants, Drugs from; Yippies*)

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MADD *See* Mothers Against Drunk Driving

MAFIA *See* International Drug Supply Systems

MAGIC MUSHROOM *See* Psilocybin

MANDATORY SENTENCING Mandatory sentencing laws provide that people convicted of particular crimes receive particular sentences. Examples include laws specifying that people convicted of selling HEROIN or COCAINE within 1,000 yards of a school receive at least a three-year prison term, or that people convicted of selling more than four ounces of heroin or cocaine receive at least a five-year prison term. The latter are referred to as mandatory minimum sentences. Some mandatory sentencing laws require life sentences. A Michigan law, for example, which the U.S. Supreme Court upheld against a claim that mandatory life sentences constitute "cruel and unusual punishment" in violation of the Eighth Amendment to the U.S. Constitution, required life sentences without possibility of parole for people convicted of possessing more than 650 grams of cocaine (*Harmelin v. Michigan*, 49 Cr.L. 2350 [6/27/91]). An Alabama law required life sentences for people who, having previously been twice convicted of felonies, are convicted of a third felony. Laws like Alabama's are sometimes called "habitual offender" or "predicate felony" laws.

ENACTMENT OF MANDATORY SENTENCING LAWS

A historically unprecedented number of mandatory sentencing laws were enacted during the 1970s and 1980s. Most involve drugs, firearms, or both. Between 1978 and 1981, forty-nine states enacted mandatory sentencing laws. Every state and the federal government enacted mandatory sentencing laws during the 1980s. In 2000, over a hundred separate mandatory minimum penalty provisions were contained in federal criminal statutes.

Apart from specific offenses that carry mandatory sentences, state and federal sentencing guidelines mandate that judges impose minimum sentences based on the crime committed, aggravating factors, and the criminal history of the defendant. These guidelines increased punishment for criminal offenses and limited judicial discretion in sentencing by identifying the punishment required upon conviction for a particular offense. Many of these statutes eliminated or greatly restricted parole for prison inmates. Congress passed the Sentencing Reform Act of 1984 (SRA). The SRA eliminated parole for federal prisoners and reduced the amount of time off granted for good behavior. The SRA also established the U.S. Sentencing Guidelines Commission and directed it to create a new

sentencing system. In 1987, the commission's guidelines became effective.

The popularity of sentencing guidelines in the United States marked a rejection of *indeterminate sentencing*. Under indeterminate sentencing, judges set maximum lengths of prison sentences, and sometime minimums, but parole boards decide when a prisoner will be released. In contrast, the Federal Sentencing Guidelines shift the focus in sentencing from the offender to the offense. The guidelines categorize offenses and identify the sentence required upon conviction. Judges are allowed to increase or decrease sentences, which are called departures, only if they have good reasons and cite these reasons into the trial record. Upward departures are easy to achieve, as judges are allowed to consider all relevant conduct. This conduct can include the circumstances surrounding the crime, offenses that were committed at the same time as the charged offense but were not charged, prior convictions, and acts for which the defendant was previously tried but acquitted. Federal judges have a more difficult time decreasing a sentence. A downward departure is acceptable if the defendant accepts responsibility for the crime, or committed the crime to avoid a more serious offense. Prosecutors often successfully challenge decreases sentences on appeal.

Mandatory sentencing laws have long been controversial. The American Law Institute, an association of lawyers, judges, and law professors that created the *Model Penal Code*, a model law on which the criminal laws of nearly half the states are patterned, opposes enactment of mandatory sentencing laws. So does the American Bar Association. In 1991, a survey of U.S. federal judges showed that 62 percent favored repeal of federal laws calling for mandatory sentences in drug cases. Federal and state judges have continued to chafe under these statutory mandates.

OBJECTIONS TO MANDATORY SENTENCING LAWS

Opponents of mandatory sentencing laws oppose them for a variety of reasons. Many judges and lawyers believe that mandatory sentencing laws are arbitrary and sometimes require judges to impose sentences that are unduly harsh. They think that justice requires that sentences be individualized to fit the circumstances of the offender and of the crime. They also think that sentences should vary depending on considerations such as whether the offender was a ringleader or a follower; whether the offender played a major role or a minor one; whether he or she was motivated by greed or poverty; whether a seller of drugs was an addict raising money to support a drug habit or a professional drug dealer; and whether the quantity involved was large or small. A law requiring that anyone convicted of selling more than a small amount of heroin receive a five-year prison sentence ignores all such distinctions.

Opponents also complain that mandatory sentencing laws adversely affect court operations. Because prosecuting attorneys decide what charges to file in each case, mandatory sentencing laws shift power from the judge to the prosecutor. Most crimes are not covered by mandatory sentencing laws. Typically, for example, trafficking in drugs is subject to mandatory penalties, but possession of drugs is not. Since nearly every drug trafficker also possesses drugs, prosecutors can decide which charge to file; a trafficking charge ties the judge's hands; a possession charge gives the judge discretion.

Another objection is that mandatory penalties remove much of the defendant's incentive to plead guilty and thus increase the frequency of trials and lengthen the time required to resolve cases. In most courts, 85 to 95 percent of convictions result from guilty pleas. Many result from plea bargains, in which the prosecutor agrees either to dismiss some charges or to approve a particular sentence if the defendant pleads guilty. If mandatory penalties remove incentives from plea bargains, then trials, backlogs, and delays increase.

Yet another objection is that mandatory sentencing laws sometimes result in deceptive practices on the part of judges. To avoid imposing sentences that they believe are too severe, judges sometimes ignore the mandatory sentence law and impose some other sentence, or acquit defendants of crimes that bear mandatory penalties.

In the context of drug laws, the controversy over disparate mandatory minimum sentences for dealers of crack and powder cocaine has raged since the late 1980s. Under a 1986 federal law, one gram of crack is equivalent to one hundred grams of powder cocaine. The U.S. Sentencing Guideline Commission adopted this ratio when it revised its guidelines that year. However, in 1988 Congress

amended the law to establish mandatory minimum sentences for cocaine dealing. Thus, selling five grams of crack cocaine is punishable by a mandatory minimum sentence of five years. To receive the same sentence for trafficking in powder cocaine, a defendant would have to sell five hundred grams. This has resulted in longer prison sentences for small-time crack dealers than for cocaine wholesalers. The federal law and similar state laws have been challenged as violations of equal protection, as African Americans have been charged with more crack cocaine offenses than whites. Similarly, whites have been charged with selling powder cocaine more often than African Americans. These legal arguments have met with little success. By the mid-1990s, the U.S. Sentencing Guideline Commission sought to reduce the disparity in sentencing. As of late 2000, however, it had been unsuccessful in its efforts.

ARGUMENTS FOR MANDATORY SENTENCING LAWS

Supporters of mandatory sentences are not troubled by the harshness of the laws or the fact that they shift power from the judge to the prosecutor. One of the goals of such laws is to assure that the mandated sentence will be imposed whether the judge agrees with the sentence or not. Supporters are troubled by deceptive efforts of judges (and sometimes of prosecutors) to avoid applying them. They argue that judges are wrong to try to circumvent mandatories, that if legislatures pass laws, judges should enforce them whether or not they agree with them. Finally, supporters say they are sorry if mandatory sentencing affects guilty pleas, trial rates, and court delays, but they regard those problems as a price worth paying.

Proponents of mandatory sentencing laws make four arguments. First, that the laws allow legislators to assure citizens their concerns are being taken seriously. Second, that harsh mandatory sentencing laws deter offenders from committing crimes. Third, that certain crimes are so serious that people who commit them should be severely punished and that legislators should insist judges impose severe penalties in such cases. Fourth, that mandatory sentencing laws are a device for assuring that offenders who commit the same crime will receive the same penalty.

RESEARCH ON MANDATORY SENTENCING LAWS

Evaluations of mandatory sentencing laws offer greater support to their opponents than to their supporters. The Panel on Sentencing Research of the National Research Council, the research wing of the National Academy of Sciences, examined all research on mandatory penalties through 1983. Studies on the deterrent effect of mandatory sentencing laws conclude either that passage of such laws has no deterrent effect or that they have a modest deterrent effect that soon disappears. Research on how mandatory sentencing laws affect court operations shows that such laws do shift power from judges to prosecutors, do sometimes result in lower guilty plea rates and higher trial rates, often cause case processing delays, and frequently result in imposition of sentences that the judges and lawyers involved believe are harsher than the defendant deserves. All of these conclusions were reached by the evaluators of the ROCKEFELLER DRUG LAWS in New York State in the mid-1970s.

The conclusions of earlier research were confirmed by the most ambitious and sophisticated study of mandatory penalties ever completed—a report on mandatory penalties in the U.S. federal courts by the U.S. Sentencing Commission. That study concluded that people convicted of crimes subject to mandatory penalties were two and one-half times more likely to be convicted after trials (30% of convictions) than are other federal defendants (12.5%). The study found that "mandatory minimums transfer sentencing power from the court to the prosecutor," that "honesty and truth in sentencing" are compromised by prosecutors' and judges' efforts to work around mandatory sentences, and that "lack of uniform application [of mandatories] creates unwarranted disparity in sentencing."

Thus, on the major empirical issues about which opponents and supporters of mandatory penalties disagree, the great weight of the evidence supports opponents' views. Empirical evidence, however, cannot refute supporters' normative claims that mandatory penalties should be enacted to assure citizens that their concerns about crime are taken seriously or that certain crimes deserve severe punishment and that mandatory sentencing laws should be enacted to increase the likelihood

that such punishments will be imposed. Opponents of mandatory penalties do not necessarily disagree that lawmakers should try to respond to citizens' concerns, or that some crimes deserve harsh penalties; they do believe that mandatory penalties are an ineffective way to achieve those goals.

(SEE ALSO: *Civil Commitment; Drug Laws: Prosecution of; Legal Regulation of Drugs and Alcohol; Treatment Alternatives to Street Crime*)

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MARATHON HOUSE *See* Treatment Programs/Centers Organizations: An Historial Perspective

MARIHUANA COMMISSION: RECOMMENDATIONS ON DECRIMINALIZATION

Before 1960, use of MARIJUANA in the United States was generally confined to drug-using subcultures in the inner cities or in rural areas. Sale and use of the drug were prohibited both by federal law and by the laws of every state. Because marijuana was classified in 1937 as a "narcotic drug," along with COCAINE and OPIATES, penalties were severe; simple possession for personal use was a felony in most states. During the 1960s, marijuana smoking suddenly became prevalent on college campuses—for the first time among white middle-class youth of the baby-boom generation. Marijuana use also became associated, as a protest behavior, with dissenters (both adults and youth) against the war in VIETNAM, and by the U.S. MILITARY serving in Vietnam, especially from 1963 to 1973. As use of the drug increased, so did the number of arrests and so did the surrounding controversy. Questions were raised about the actual effects of marijuana on the health and behavior of those who used it and about the wisdom of prevailing social policy.

In response to swirling controversy, many proposals were introduced in Congress for a commission to undertake an authoritative study of the marijuana issue. Eventually, in the Comprehensive Drug Abuse Prevention and Control Act of 1970, Congress established the NATIONAL COMMISSION ON MARIHUANA AND DRUG ABUSE to undertake a two-year study—the first year on marijuana and the second year on the causes of drug abuse in general.

The commission had thirteen members—four from Congress (two each from the House and the Senate) and nine appointed by the president. President Richard M. Nixon appointed Raymond P. Shafer, formerly governor of Pennsylvania, as chairman of the commission, and he appointed Dana L. Farnsworth, M.D., director of Student Health Services at Harvard University, to be vice-chairman. The executive director was Michael R. Sonnenreich, formerly the deputy chief counsel of the Bureau of Narcotics and Dangerous Drugs of the Justice Department.

The commission assimilated the available literature on marijuana use and its effects and also sponsored its own research, including a national survey of use patterns and public attitudes, and a study of enforcement of the marijuana laws in six jurisdictions. In March 1972, the commission issued its first report, *Marihuana: A Signal of Misunderstanding*.

PRINCIPAL FINDINGS

The commission estimated that although 24 million Americans had used marijuana at least once, about 50 percent had simply experimented with the drug out of curiosity and given it up. Among the 50 percent who had continued to use marijuana, most used it only occasionally, once a week or less, for recreational purposes. A small percentage of the more frequent users (about 2% of the total ever-using population—or 4% of the continuing users) used the drug more than once daily. Marijuana use was clearly age-related: about half of the ever-users were 16 to 25 years of age, and 44 percent of those who were currently in college or graduate school had used marijuana at least once.

The commission concluded that there was "little proven danger of physical or psychological harm from the experimental or intermittent use" of marijuana. "The risk of harm," it continued, "lies instead in the heavy, long-term use of the drug, particularly of its more potent preparations." Even this risk was of uncertain dimensions, the commission noted, because the psychological consequences of long-term heavy use were unknown. In light of the fact that 90 percent of marijuana users used the drug only experimentally or intermittently, the commission judged that "its use at the present level does not constitute a major threat to public health." The commission also specifically found that marijuana did not induce physical dependence; did not lead, by virtue of its pharmacology, to use of other drugs; and did not cause criminal behavior.

POLICY RECOMMENDATIONS

The commission's principal policy recommendation was that possession of one ounce or less of marijuana for personal use be "decriminalized." At the same time, the commission rejected outright legalization of the drug and recommended perpetuation of prohibitions against cultivation and distribution for commercial purposes. The commission stipulated that social policy should aim to discourage use of the drug, but it emphasized that the costs of a criminal prohibition against possession far exceeded its benefits in suppressing use.

Although President Nixon disavowed the commission's principal recommendation on marijuana, it won widespread support. In 1973, the National Conference of Commissioners on Uniform State Laws promulgated amendments to the Uniform Controlled Substances Act that codified the commission's recommendation. Some form of decriminalization was endorsed the same year by a variety of national organizations, including the American Bar Association and numerous state and local bar associations, the National Education Association, the Consumers' Union, the National Council of Churches, the American Public Health Association, and the governing board of the American Medical Association.

In 1973, Oregon became the first state to decriminalize possession of small amounts of marijuana. Within the next five years, ten additional states eliminated incarceration as a penalty for simple possession, usually substituting a $100.00 fine. Five of these states made possession a "civil offense"; in others, it remained a criminal offense although the law typically contained a provision for expunction of criminal records after a specified period of time. Decriminalization of marijuana use was endorsed by President Jimmy Carter in 1977.

Political and legislative support for decriminalization began to wane, however, even during the Carter Administration. The more permissive stance on marijuana use implicit in decriminalization efforts led to mounting public resistance. Some of the strongest opposition came from groups of parents who organized to lobby for more focus on PREVENTION efforts. Although these parent groups were generally conservative politically, they gained a receptive ear in the Carter White House. Their arguments against decriminalization were bolstered by findings from the National High School Senior Survey showing that, starting in 1975, daily marijuana use had been increasing progressively among high school students. During the Reagan and Bush administrations the parents' movement and their concerns about marijuana use came to have a major influence on national drug policy. In the early 1990s, possession of the drug remained a criminal offense in most states, as well as under federal law.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy; High School Senior Survey; Legal Regulation of Drugs and Alcohol; Prevention*)

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MARIJUANA In the United States, this is the most common term for the HEMP plant Cannabis sativa and its mind-altering (PSYCHOACTIVE) products. The term derives from the Mexican Spanish *mariguana/marihuana* (sometimes explained as Mary's leaf or Mary's plant, or from María y Juan, that is, Mary and John, the source of the English slang Mary Jane or maryjane.) It came into recorded English about 1890 and has become the mainstream term in American publications, law, and general usage. The term cannabis is sometimes used in medical literature and by the British; it means *hemp* in Latin and is derived from the Greek, *kannabis*, itself borrowed into Greek from an unknown source. In ASIA, where the plant originated, it is grown legally and commercially both for its fiber content (it is used to make strong rope) and for its drug content; there it is called BHANG (from Sanskrit bhang) or bang, GANJA or churganja, and HASHISH.

BOTANY

Hemp grows easily throughout the tropics, subtropics, and temperate regions, varying from a few feet to 15 feet (4.6 m) in height. Once established, it reseeds itself and spreads to neighboring areas; when birds eat the seeds, the defecated seeds may be scattered over considerable distances and produce new plants.

Two genetic strains of hemp are recognized: one produces plants excellent for fiber with very little drug material; the other produces plants with weak fibers but much drug content (TETRAHYDROCANNABINOL, THC). To harvest the drug-laden plant, it is simply cut down and usually chopped into small pieces with all parts included. These clippings resemble lawn cuttings, so one of the slang terms is "grass." The major use of this form in the United States is for illegal marijuana cigarettes, often called reefers.

Since the early 1900s, marijuana has been considered the one drug that might introduce the susceptible to hard drugs, drug abuse, and drug dealing. In the United States until 1937, Cannabis had been used in medical practice for a number of conditions but marijuana use for its euphoric effect was relatively uncommon. By 1937, forty-six of the then forty-eight states had laws against the use of marijuana, and its use had already been made a criminal offense under federal law. Until the 1960s, it was smoked largely by African Americans and Hispanics in the United States but was generally shunned by the white majority. During the social and political protests of the 1960s, a change in attitudes allowed widespread but illegal marijuana use into all levels of society, along with an increase in the use of several other illegal drugs and a boom in the drug trade that continued into the 1990s.

HISTORY

Various historical allusions to medicinal plants suggest that Cannabis was known and used for several thousand years. The earliest references to the plant are in ancient Chinese and Indian writings. From India, the use of Cannabis spread to Persia, Assyria, and the rest of the Near East. The Arabs adopted and spread it through North Africa as they conquered those lands for Islam from the seventh to the fifteenth centuries. Islam forbids the use of ALCOHOL, but not explicitly Cannabis (since it was adopted after the laws established by the Prophet Muhammad, who lived from about 570 to about 632 A.D.). In Arabic, it is called HASHISH, meaning grass. After the Arabs crossed the Strait of Gibraltar into the Iberian peninsula in 711, they ruled there until 1492. Portugal and Spain did not generally adopt its use. The Spanish conquistadors, however, introduced Cannabis into the New World, where it was readily adopted by African slaves, who were already familiar with it because of Arab trade and the spread of Islam into their continent.

CHEMISTRY

Like most plants, Cannabis contains many substances, perhaps two hundred or more. Those that relate most to the drug effects are a group of chemically similar compounds called cannabinoids. Of these, the most important and plentiful are cannabidiol (CBD), tetrahydrocannabinol (THC), and cannabinol (CBN). The biosynthetic pathway in the plant (that is, the step-by-step sequence in which the plant produces substances) goes from CBD to THC to CBN. Thus it is possible to identify the maturity of the plant by the relative content of these three cannabinoids. Immature plants show a preponderance of CBD; old plants may contain solely CBN; plants that are at their peak contain all cannabinoids, but mostly THC, which is the agent that produces the mind-altering effect. Some strains of plants contain variants on the THC structure, which usually have somewhat less drug effect than those with THC. Although some users contend that marijuana has different effects from those of isolated THC, most evidence indicates that virtually all of the mind-altering effects of marijuana are attributable to the THC content.

The THC content may vary greatly, depending on the genetic strain of the plant, the part of the plant involved (for example, the leaves or the flowers), and the maturity of the plant. The THC content of plants used for hemp production, such as those that grow wild in the U.S. Midwest, may be negligible to zero; marijuana produced from plants known for high drug content, such as sensemilla, may contain 2 to 3 percent THC. Manicured plants, from which the leaves are carefully separated and only the new leaves used for drug effect, may contain 3 to 4 percent THC. Hashish, which represents the ultimate in manicuring, generally contains 4 to 8 percent THC.

THC is sensitive to exposure to air and light. Thus, marijuana that is not protected from such exposure undergoes gradual degradation until the drug content is gone. When protected from air and light, marijuana may retain its activity for many months.

EPIDEMIOLOGY

Marijuana may rank behind only CAFFEINE, alcohol, and NICOTINE as the most widely used drug in the world. It is estimated that between 200 and 300 million people use this material in one way or another. In the United States alone, probably some 20 to 30 million people have used the drug, although the number of regular users is probably far less, but still a few million.

A tobacco-like substance produced by drying the leaves and flowering tops of the cannabis plant, marijuana varies significantly in its potency, depending on the source and selection of plant materials used. (Drug Enforcement Administration)

In the United States, marijuana is a drug preferred by young people; the rate of marijuana use is therefore followed among schoolchildren to estimate changing trends. Survey responses of highschool students, concerning marijuana, show very wide variations. Overall, 3 to 17 percent (median 12%) reported at least a single use of marijuana during the preceding thirty days. Such use is relatively low compared with that of smoking at least one cigarette, 9 to 37 percent (median 31%), or having at least one drink of alcohol, 28 to 64 percent (median 54%). Thus, it would appear that marijuana is not nearly as widely used as two of our three national drugs. Although this data indicates a trend toward decreased use of and greater concern about marijuana compared with nicotine and alco-

hol, this pattern has not held long enough to establish a true trend; it may be simply a minor blip.

A number of factors seem to contribute to use of marijuana among young people. Being male, using cigarettes and alcohol, and becoming delinquent are predisposing factors. Coming from a broken home and performing poorly in school are also predictive factors. Among adolescents in Australia and New Zealand, use of stimulants, HALLUCINOGENS, NARCOTICS, and SEDATIVES was virtually limited to those young people who used marijuana. Overall, it appears that school factors are less predictive of Cannabis use than are other social factors.

PSYCHOPHARMACOLOGY

Marijuana has a wide range of pharmacologic effects that suggest actions like those of stimulants such as the AMPHETAMINES, hallucinogens such as LSD, and depressants such as alcohol, SEDATIVES, atropine, or MORPHINE. Thus, marijuana does not fit any single traditional pharmacologic classification, and, hence, must be considered as a separate class.

The experienced smoker of marijuana is usually aware of a drug effect after two or three inhalations. As smoking continues, the effects increase, reaching a maximum about twenty minutes after the smoke has been finished. Most effects of the drug have usually vanished after three hours, by which time tests show that concentrations of THC in the body's plasma are low. Peak effects after eating marijuana may be delayed for three to four hours, but may then last for six to eight hours.

The early stage is one of being high, characterized by euphoria, uncontrollable laughter, alteration of one's sense of time, depersonalization, and sharpened vision. Later, the user becomes relaxed and experiences introspective and dreamlike states, if not actual sleep. Thinking or concentrating becomes difficult, although by force of will the person can concentrate to some extent.

Two characteristic signs of Cannabis intoxication are increased pulse rate and reddening of the conjunctiva (the whites of the eyes). The latter correlates well with the presence of detectable concentrations of THC in the plasma. Pupil size is not changed. The blood pressure may fall, especially in the upright position (orthostatic hypotension). An antiemetic (decrease in sense of nausea) effect may be present, and muscle weakness, tremors, unsteadiness, and increased deep-tendon reflexes (such as the knee jerk) may also be noted.

Virtually any performance test shows impairment if the doses are large enough and the test is difficult enough, although no distinctive biochemical changes have been found in human beings.

TOLERANCE to Cannabis has been demonstrated in virtually every animal species that has been tested. It is apparent in human beings only among heavy long-term users. Different degrees of tolerance develop for different effects of the drug, with tolerance for the tachycardiac effect (increased pulse rate) developing fairly rapidly. A mild WITHDRAWAL syndrome has been noted following very high doses.

HEALTH CONSEQUENCES

The ambiguity surrounding the health hazards of Cannabis may be attributed to a number of factors besides those that ordinarily prevail. First, from animal studies, it has been difficult to prove or disprove health hazards in human beings. Second, Cannabis is still used mainly by young persons in the best of health. Third, Cannabis is often used in combination with tobacco and alcohol, among licit drugs, as well as with a variety of other illicit drugs. Finally, the whole issue of Cannabis use is so laden with emotion that serious investigations of the health hazards of the drug have been colored by the prejudices of the experimenter, either for or against the drug as a potential hazard or benefit to health.

Psychiatric Consequences. Cannabis may directly produce an acute panic reaction, a toxic delirium, an acute paranoid state, or acute mania. Whether it can directly evoke depressive or schizophrenic states, or whether it can lead to sociopathy or even to the so-called AMOTIVATIONAL SYNDROME is much less certain.

That Cannabis use may make schizophrenia already present even worse is beyond any question. Such worsening followed acutely after use of Cannabis by schizophrenics, despite continued maintenance of antipsychotic drugs, and other adverse reactions were encountered among seventy patients in Sweden—anxiety reactions, flashbacks, dysphoric reactions, and abstinence syndromes.

Whether chronic use of Cannabis changes the basic personality of users so that they become less impelled to work and to strive for success has been

a vexing question. As with other questions concerning Cannabis use, it is difficult to separate consequences from possible causes.

Automobile Driving. If marijuana were to become an accepted social drug, it would be important to know its effects on driving ability. Fully 50 percent of the fatal auto accidents in the United States are associated with alcohol, another social drug. Neither experimental nor epidemiological approaches to the marijuana question have yet provided definitive answers.

Cardiovascular Problems. For persons with heart disease caused by hardening of the coronary arteries or by congestive heart failure, the effects of Cannabis smoking would be harmful: tachycardia, orthostatic hypotension, and increased concentrations of carbon monoxide in the blood.

Clearly, smoking of any kind is bad for patients with angina, but the greater effect of Cannabis as compared with tobacco in increasing heart rate makes this drug especially bad for such patients. Fortunately, thus far, few angina patients have been devotees of Cannabis.

Lung Problems. Virtually all users of Cannabis in North America take the drug by smoking. As inhaling any foreign material into the lung may have adverse consequences, well proven in the case of tobacco, this mode of administration of Cannabis might also be suspect. A formal study has shown that very heavy marijuana smoking for six to eight weeks caused mild but significant airway obstruction.

The issue of damage to lungs from Cannabis is somewhat unclear because many Cannabis users also use tobacco. As yet, it is far easier to find pulmonary cripples from the abuse of tobacco than it is to find any evidence of clinically important weakness of the lungs caused by smoking Cannabis.

Endocrine and Metabolic Effects. A review of literature on this subject concluded that sperm production was decreased, but without evidence of infertility. Ovulation was inhibited as luteinizing hormone, which stimulates ovulation, was decreased.

Immunity. A number of test-tube studies, using both human and animal material, suggest that cell-mediated immunity (the capacity of white blood cells to fight invading bacteria, viruses, or cancer cells) may be decreased after exposure to Cannabis. Clinically, one might assume that sustained impairment of cell-mediated immunity might lead to increased opportunistic infections or to increased prevalence of cancer, as seen in the current epidemic of ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS). No such clinical evidence has been discovered.

THERAPEUTIC USES

For many centuries, Cannabis was used as a treatment, but only during the nineteenth century did a particularly lively interest develop for exploiting its healing powers. Cannabis was then reported to be effective in treating tetanus, convulsive disorders, neuralgia, migraine, menstrual problems, psychoses following childbirth, insomnia in the aged, depression, and gonorrhea, as well as in helping cure addiction to opium or to chloral hydrate. In addition, it was used to stimulate appetite and to relieve the pain and anxiety of patients terminally ill with cancer. Few of these claims have even been properly tested in clinical studies.

Antiemetic for Patients in Cancer Chemotherapy. An antiemetic is a substance that suppresses vomiting. CANCER chemotherapy, especially with the agent cisplatin, produces severe nausea and vomiting, which is extremely difficult to treat with ordinary antiemetic drugs, such as prochlorperazine. This complication is so severe that many patients forgo effective cancer chemotherapy. The antiemetic effects of Cannabis had been suggested as early as 1972. In that year, a synthetic drug similar to THC, nabilone, was developed. It has been tested extensively for antiemetic activity. A crossover study comparing nabilone with prochlorperazine revealed significantly better results (that is, less nausea and vomiting) following nabilone therapy, although side effects from nabilone were also common.

The potential role of THC as an antiemetic may have become irrelevant because of recent developments. Metoclopramide, a newly developed antiemetic unrelated to the cannabinoids, has been found to be effective when given in high intravenous doses. Lorazepam, dexamethasone, and ondansetron are also useful as antiemetic agents when given by injection. These drugs are often used in various combinations, which meet most requirements. Thus, THC may be superseded even before it has had widespread clinical trial.

Glaucoma. The disease glaucoma causes pressure in the eyeball to increase greatly. If untreated, it can lead to blindness. Discovery of the ability of Cannabis to lower intraocular (inside the eyeball) pressure was more or less a matter of chance. This pressure was measured as part of a multifaceted study of the effects of chronic smoking of large amounts of Cannabis: it decreased as much as 45 percent in nine of eleven subjects, thirty minutes after smoking.

This exploitation of cannabinoids for treatment of glaucoma will require much further developmental work to ascertain which cannabinoid will be lastingly effective and well tolerated topically.

Miscellaneous Uses. Cannabinoids have been found to have analgesic (pain-relieving) activity, and efforts are being made to synthesize new compounds that separate this action from the others. They have also been used for relaxing muscles, for treating bronchial asthma, and for stopping convulsions. Thus far, none of these additional potential therapeutic uses has been fully established.

TREATMENT OF MARIJUANA USE

In general, marijuana users, even those whose use is heavy, do not feel compelled to seek treatment unless such use is complicated by other drugs, such as COCAINE or alcohol. In this case, treatment efforts are usually directed toward the complicating drug. Thus, treatment programs directed specifically at marijuana use are rare. A TWELVE-STEP approach, similar to that for alcohol, has been proposed, but its feasibility and its efficacy have not been tested.

GATEWAY EFFECT

Since about 1950 (but not much prior to that time) in the United States, smoking of marijuana has been linked statistically to the use of other illegal drugs, such as heroin and cocaine. Most observers have concluded that the link is sociological rather than biological, and that the use of marijuana is a marker for individuals who are more prone to seek new experiences even when these violate social norms and local laws. Further, the process of obtaining illegal marijuana increases the likelihood of contact with dealers and other individuals who have access to drugs such as HEROIN. Consequently, marijuana has been referred to as a "gateway" drug, one whose use often leads to the use of other illegal drugs. Some programs are aimed at preventing even experimentation with marijuana—not only for whatever inherent benefits this approach may have, but also in the hope that in doing so the movement to other more potentially lethal drugs will be prevented.

LEGAL STATUS

Despite its widespread use, marijuana has not yet been admitted to the company of accepted social drugs such as alcohol and nicotine. Laws remain that prescribe penalties for its possession, use, and sale. In some jurisdictions, possession and use of small amounts of the drug is a civil crime punishable only by a small fine. Despite the liberalization of the law in these areas, they have not been overrun with eager marijuana users. Perhaps the reason is that in most other jurisdictions, laws against its use are rarely enforced. Enforcement can be capricious, however, when employed in situations in which more serious crimes cannot be adequately documented.

A new drug application was approved for THC (Marinol) to be used therapeutically for control of the nausea and vomiting associated with cancer chemotherapy. Thus, THC was moved from Schedule 1 of controlled substances (no medical use) to Schedule 2 (medical use despite potential for abuse). Nabilone, the synthetic drug similar to THC, used for the same purpose, also has this status.

Thus far, no attempt has been made to establish legal limits on the amounts of THC in the blood that might be construed as impairing automobile driving. No doubt the issue has not yet appeared to be of enough gravity, since marijuana contributes little to the danger of driving as compared with alcohol.

(SEE ALSO: *Adolescents and Drug Use; Cannabis Sativa; Complications; Controls; Driving, Alcohol, and Drugs; High School Senior Survey; Marihuana Commission; Yippies*)

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MARIJUANA EPIDEMICS *See* Epidemics of Drug Use; Yippies

MAST *See* Michigan Alcoholism Screening Test

MDMA This drug is popularly known as "ecstasy," XTC, and ADAM. It is a synthesized compound and a member of the family of HALLUCINOGENS known as the substituted phenethylamines, which also includes methylenedioxyamphetamine (MDA) and 2,5-dimethoxy-4-methylamphetamine (DOM) (see Figure 1). These hallucinogens are structurally related to the phenethylamine-type NEUROTRANSMITTERS dopamine, norepinephrine, and epinephrine. Many analogs of these compounds have been synthesized and are sometimes found on the street—the so-called DESIGNER DRUGS.

Controversy exists as to whether MDMA and MDA should be classified with the other hallucinogens. Both MDMA and MDA have structural similarities to the PSYCHOSTIMULANT AMPHETAMINE, and they have amphetamine-like psychostimulant properties. Yet, these designer drugs also have properties in common with LYSERGIC ACID DIETHYLAMIDE (LSD) and MESCALINE; with lower doses, however, they produce fewer perceptual phenomena and less emotional liability, or "keyed-up" feelings and disturbances of thought, than other hallucinogens, and there tends to be a tranquil state with a feeling that tender emotions are meaningful. As doses are increased, the illusions and other LSD-like phenomena are seen. Because of their mixed effects, MDMA and MDA are sometimes referred to as STIMULANT-hallucinogens.

Unlike LSD, users of MDMA have reported nausea, jaw clenching and teeth grinding, increased muscle tension, and blurred vision, as well as panic attacks. MDMA also causes amphetamine-like stimulation of the autonomic nervous system, producing increases in blood pressure, heart rate, and body temperature. A type of hangover the day after taking MDMA has been described, involving headache, insomnia, fatigue, drowsiness, sore jaw muscles, and loss of balance.

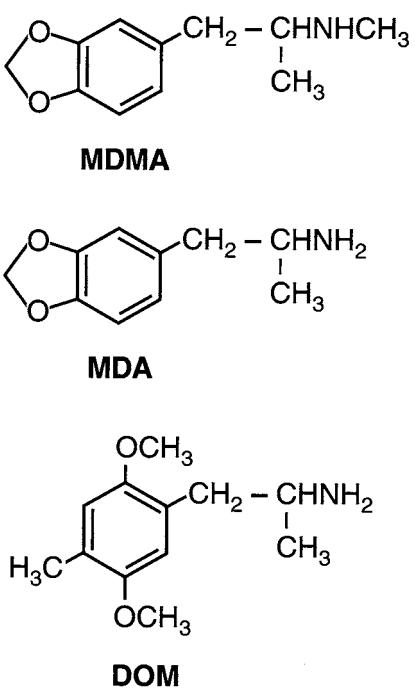

Figure 1
Phenethylamine Hallucinogens

Like the other hallucinogens, the exact mechanisms of action of MDMA are not known. MDMA, like the indole- and phenethylamine-type hallucinogens, binds to receptors for the neurotransmitter serotonin. Thus, many effects might be due to interactions with brain serotonergic systems. MDMA, however, also causes the release of both dopamine and serotonin, so some effects may be related to their stimulant properties.

By the early 1990s, some evidence indicated that MDMA might damage nerve cells. In laboratory experiments, MDMA can produce long-lasting changes in the function of neurons that use serotonin as the neurotransmitter, sometimes causing the death of these cells. Even though LSD also interacts with serotonergic nerve cells, the administration of massive doses of LSD does not damage these cells. In contrast, in experimental animals, a single dose

MDMA (Ecstasy) packaged for bulk distribution. (Drug Enforcement Administration)

of MDMA approximately three times higher than the typical street dose has been shown to affect brain serotonergic systems for several weeks. In some studies, neurochemical markers did not return to normal until one year after drug administration. Moreover, it is not clear whether there was actual regeneration of neurons or only compensatory changes in the remaining undamaged neurons. In these experiments, the neurotoxic effects of MDMA appear to depend on total exposure. Both the dose taken and the number of times the drug is consumed may be related to brain-cell changes. The exact mechanism of MDMA-induced neurotoxicity is unknown at this time and may be due to MDMA itself, or it could involve the formation of a neurotoxic metabolite.

Although there is controversy whether studies utilizing laboratory animals can be extrapolated to human MDMA users, some evidence suggests that brain function can be altered in humans exposed to MDMA. Although the consequences to behavior and thinking caused by damage to the serotonergic nerve cells in young users are unknown, some effects of MDMA-induced toxicity may become apparent as the users age. Cells die as part of the aging process, and if exposure to MDMA kills or weakens a certain proportion of cells, the effects of normal cell loss due to aging might be exacerbated. Serotonergic systems have been implicated in the control of sleep, food intake, sexual behavior, anxiety, and mood. Thus, serotonergic cell loss could have major consequences.

(SEE ALSO: *Complications: Mental Disorders; Dopamine; Methamphetamine; Serotonin*)

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MEDELLIN CARTEL *See* Colombia as Drug Source

MEDICATIONS APPROACHES FOR TREATING SUBSTANCE ABUSE *See* Pharmacotherapy

MEGA VITAMIN THERAPY *See* Treatment, History of; Vitamins

MEMORY AND DRUGS: STATE DEPENDENT LEARNING The term *state dependent learning* (SDL) refers to the fact that memories acquired while a person is drugged may be forgotten when the drug wears off and not remembered until the person again takes the drug. Conversely, material learned in the undrugged state may be forgotten when a drug is taken; and material learned under one drug may be forgotten when another drug is used. SDL is sometimes called drug

dissociation of learning, referring to the fact that material learned while drugged is dissociated from normal consciousness and not able to be retrieved.

Throughout the nineteenth century, there was a high level of public interest in multiple personality, fugue states, and other types of episodic amnesia; SDL was first reported in 1835 by George Combe, an English phrenologist, who viewed it as an analogous phenomenon, perhaps based on similar properties of the brain. SDL became an accepted property of mind during the latter half of the nineteenth century, and was a central theme in the plot of *The Moonstone*, (1868), a well-known mystery novel written by Wilkie Collins. Then, at the beginning of the twentieth century, interest in these dissociative phenomena waned and was replaced by an interest in the amnesias caused by repression, which Freud described. SDL was essentially forgotten.

SDL was rediscovered in the 1960s, this time in experiments using animals, and since then has been a popular topic of research and clinical speculation. Two types of mechanisms are postulated as possibly producing SDL. According to one theory, drugs produce sensory stimuli, subjective sensations—and one's ability to retrieve memories is aided by reinstatement of the stimuli that were present when learning occurred. A second theory suggests that some other property of brain results in memories being most easily retrieved when the conditions of brain excitability that were present during learning are reestablished. Sensory stimuli are not involved in producing SDL, according to this second theory. Thus far it has not been possible to confirm either of these proposed mechanisms experimentally, although the sensory model is more widely accepted.

SDL is produced only by drugs that act on the brain. There are marked differences in the strength of the SDL effects produced by the different centrally acting drugs. For example, BARBITURATES and ALCOHOL produce strong SDL effects, whereas chlorpromazine (Thorazine) produces almost no such effects. SDL is more likely to occur with high doses of drugs, and research on SDL has been severely hampered by the fact that these doses also produce other effects on memorization and retrieval that are difficult to distinguish from SDL effects. Some research suggests that the relative ability of different drugs to produce SDL may differ depending on the type of task that is employed, but this conclusion is not yet well substantiated.

Many consider SDL to be closely related to drug discriminations, believing that the discriminative control exercised by drug conditions is produced by the same drug effects that produce SDL amnesias at higher doses.

After SDL was rediscovered in the 1960s, clinicians feared that the lessons of psychotherapy carried out while a patient was drugged might be forgotten when drug treatment was discontinued. Subsequent studies showed that strong SDL effects typically did not occur except at doses higher than those normally employed during chronic treatment with psychotropic drugs. Some evidence, however, suggests that the stimulant drugs used to treat hyperactive children may produce SDL in those children. There is increasing evidence that some types of learning may take place under general anesthesia, although patients report they remember nothing after the anesthesia wears off. A considerable amount of research is currently focused on the possibility that SDL may block explicit recall of learning under general anesthesia, even though such learning occurs.

Many centrally acting drugs alter moods. A currently active area of research deals with the possibility that emotions act as memory cues and that memories learned in one emotional state may be recalled best when that emotion reoccurs; they may be recalled less easily at other times. Finally, there has been a dramatic increase in the number of reported cases of multiple personality disorder during the past decade. One of the theories used to explain this disorder holds that the process underlying it is similar, at a mechanistic level, to that which produces drug-induced SDL.

(SEE ALSO: *Memory*, *Effects of Drugs on; Research; Animal Model*)

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MEMORY, EFFECTS OF DRUGS ON

Research investigating the effects on memory of ALCOHOL (ethanol) and drugs of abuse is disproportionally small in relation to the widespread use of these substances worldwide. The available evidence clearly indicates that ethanol and abused drugs significantly affect memory processes. Much of current knowledge of the effects of such commonly used substances on memory is based on experiments using laboratory animals. In typical experiments, the animals are trained in a learning task and given a memory retention test after a delay of one day or longer. In experiments on commonly used learning tasks, the animals are trained to acquire responses that provide escape from, or avoidance of, aversive (negative) stimulation. Appetitive motivation (food or water reward) is also used to train animals in mazes and other types of spatial learning.

When investigating acute (single treatment) influences on learning and memory, drugs can be administered before the training, shortly after the training, or before the memory test. When drugs are administered before training, it is difficult to distinguish effects on memory from other influences on sensory, motivational, and motor processes. When administered within a few minutes after training, but not after a delay of several hours, drugs of many classes can enhance or impair memory. Such findings are interpreted as indicating that the drugs can modulate memory-consolidation processes occurring after a training session. The drug effects are typically dose-dependent. For example, drugs that enhance memory when administered in low doses may impair memory when administered in higher doses. Experiments examining the effects of a drug administered prior to memory testing are difficult to interpret, since drugs can affect many processes affecting behavior other than memory. For the same reasons, the alterations in memory performance that are produced by the chronic (long-term) administration of drugs are also difficult to interpret.

ALCOHOL (ETHANOL)

In rats and mice, an acute (a large) dose of alcohol prior to learning usually impairs memory of the training. The effect is heightened by the drug clonazepam, a BENZODIAZEPINE RECEPTOR AGONIST; it is lessened by bicuculline and picrotoxin, drugs that block receptors for the inhibitory NEUROTRANSMITTER GABA (GABA-A receptors). Such findings suggest that ethanol-induced amnesia is mediated by the benzodiazepine/GABA-A receptor complex. These findings are consistent with extensive evidence that benzodiazepines (see section below) induce amnesia in humans as well as in laboratory animals. Memory impairment induced by a large dose of alcohol is also lessened by physostigmine, the acetylcholinesterase inhibitor, suggesting that ethanol influences on memory involve cholinergic mechanisms.

Chronic administration of a high dose of ethanol to rats or mice over time induces memory impairment, accompanied by a decreased function of cholinergic systems in specific brain regions, including the hippocampus and neocortex. The syndrome can be reversed by an implant, into either BRAIN STRUCTURE, of fetal brain tissue that has high numbers of cholinergic cells or by giving oxotremorine, the cholinergic muscarinic agonist, prior to memory testing. Such findings suggest that the memory impairment resulting from chronic ethanol ingestion is associated with a deficit of brain cholinergic function.

Acute or chronic ethanol ingestion produces memory problems in humans. Large amounts of ethanol taken over a short period (hours or days) may cause a severe amnesia—a "blackout" for events occurring during and/or shortly before the period of intoxication. Some alcoholic blackouts may be caused partially by state-dependency—that is, during a later intoxication, individuals may sometimes remember experiences that occurred during a previous blackout. This phenomenon was illustrated in Charles Chaplin's 1931 film *City Lights*, in which the hard-drinking millionaire remembered Charlie only when under the influence of alcohol.

Paradoxically, experiments with human subjects indicate that low doses of ethanol administered

immediately after learning enhance retention. Similar results have been obtained in studies using laboratory animals; however, it is not clear that effects seen in animals are due primarily to ethanol effects on brain processes underlying memory. They may reflect, at least in part, the aversive aftereffects of ethanol.

Clinical research shows that chronic ingestion of alcohol can produce three general categories of brain impairment that are associated with memory deficits: the Wernicke-Korsakoff syndrome, alcoholic dementia, and "nonamnesiac" or "non-Korsakoff" disorders. Wernicke-Korsakoff syndrome, the best known, is due to Vitamin B_1 (thiamine) deficiency, resulting from poor food intake during sustained periods of alcohol consumption. It involves an acute phase, with mental confusion and difficulty with eye movements and walking. Most people who recover from this acute phase after treatment with thiamine will have Korsakoff's syndrome, in which impairment of the ability to learn and remember new information (anterograde amnesia) as well as retention of recently acquired information (retrograde amnesia) occur, although apparently normal intellectual function and the ability to acquire and retain skill-based information, such as purely visual/motor tasks, appear to be relatively unaffected. Some improvement in the memory deficits may occur with prolonged abstinence from alcohol.

Alcoholic dementia differs from Korsakoff's syndrome in that it is characterized by severe memory impairment as well as major intellectual deterioration that can be difficult to distinguish from Alzheimer's Disease by clinical examination. Improvements are, however, often seen if patients abstain from alcohol.

It is not known whether the deficits seen in early alcoholic dementia and in Korsakoff's syndrome are accompanied by alterations in GABAergic or cholinergic functioning. The changes seen in late alcoholic dementia, like those of Alzheimer's Disease, involve multiple focal brain lesions, primarily in the temporal lobe but also in other brain regions, and involve deficits in glutaminergic, GABAergic, and cholinergic systems.

The third type of memory problem linked to alcohol ingestion has been variously referred to as "neurologically intact" or "neurologically asymptomatic" and is characterized by subtle impairments in dealing with abstractions, problem solving, and memory. Significant recovery with abstinence is typical.

BENZODIAZEPINES

BENZODIAZEPINES, which are used clinically in the treatment of ANXIETY and the induction of sleep, are among the most widely used (and abused) drugs. It has been known for several decades that benzodiazepines, including diazepam (Valium), triazolam (Halcion), and CHLORDIAZEPOXIDE (Librium) induce anterograde amnesia in humans. Studies using laboratory animals indicate that benzodiazepines impair memory when administered before training, but they generally do not impair memory when administered posttraining. The lack of posttraining effects may be due, at least in part, to the fact that benzodiazepines are absorbed slowly and are slow to reach peak concentrations in the brain following peripheral injections. The anterograde amnesia induced by benzodiazepines is not due either to alterations in sensory or motivational processes affecting learning or to state-dependency.

Benzodiazepines are known to act by modulating GABA-A neurotransmitter receptors on the benzodiazepine/GABA receptor complex. Their effects on memory appear to be mediated primarily by the brain structures designated as the amygdaloid complex and hippocampus. When administered acutely, either systemically or directly into specific brain regions, including the amygdaloid complex and the hippocampus immediately posttraining, retention is enhanced by flumazenil, the benzodiazepine-receptor antagonist, and by the GABA-A-receptor antagonists bicuculline and picrotoxin. Findings indicating that the amnesia induced by peripherally administered benzodiazepines is blocked by GABAergic antagonists administered directly into the amygdaloid complex, as well as by lesions of the amygdaloid complex, provide additional evidence that this brain region is involved in benzodiazepine effects on memory. Although benzodiazepine-like substances are found in the brain, it is not yet known whether they are synthesized in brain cells or derived from food. Evidence that training experiences release these naturally occurring brain substances from synaptic vesicles in neurons suggests that they may play a role in modulating memory-storage processes.

MARIJUANA

In laboratory animals, both acute and chronic administration of marijuana extracts or of their active principles, the TETRAHYDROCANNABINOLS (THC), have been reported to impair the acquisition and retention of a very wide variety of tasks. It is not known whether these effects are due to influences on memory or simply to the sedative influences of the drug. There is some evidence suggesting that acute or chronic use of MARIJUANA impairs human memory. It is not known, however, whether such effects are due specifically to influences on brain processes underlying memory or to other influences on behavior. Cessation of marijuana use typically results in rapid recovery from the drug effects. Little is known about brain influences mediating marijuana effects on learning and memory.

OPIATES AND OPIOID PEPTIDES

The OPIATE drugs MORPHINE and HEROIN, administered posttraining, impair retention in laboratory animals. The memory impairment is not state-dependent: Administration of opiates prior to retention testing does not decrease the impairment. Opiate-receptor ANTAGONISTS, including NALOXONE and NALTREXONE, enhance memory and block the memory impairment produced by opiates. Endogenous opioid peptides (brain peptides that mimic the effect of morphine, heroin, and other opiates) also affect memory. The opioid beta-endorphin is released in the brain when animals are exposed to novel training situations. Memory impairment is induced by posttraining injections of beta-ENDORPHIN as well as by injections into several brain regions, including the amygdaloid complex and medial septum. Opiate antagonists administered into these brain regions enhance memory. Unlike the effects of opiate drugs, the memory impairment induced by beta-endorphin may be due, at least in part, to the induction of state-dependency: Under some conditions beta-endorphin administered (or endogenously released) prior to memory testing may lessen the memory impairment induced by a posttraining injection of the peptide.

Despite the widespread and long-standing use of opiate drugs by humans, there have been no systematic studies on the effect of morphine, heroin, or other opiates on human memory. Chronic opiate users do show memory deficits, but these may result from general deterioration rather than from any specific effect of the opiates. Acute administration of opiates (as in preanesthetic medication, for example) may induce a temporary amnesia. The failure of patients to remember experiences immediately prior to surgery may be due, at least in part, to an amnestic effect of the opiates used for ANALGESIA (PAIN suppression). The effect of opiate antagonists has been explored clinically in the treatment of dementias, but with limited success.

AMPHETAMINE

In laboratory animals, chronic administration of AMPHETAMINE prior to training impairs performance in many types of learning tasks. Such effects are typically obtained in experiments using high doses of amphetamine and complex learning tasks. In contrast, extensive evidence, from studies using a variety of types of training tasks, indicates that acute posttraining injections of amphetamine produce dose-dependent enhancement of memory. Retention is also enhanced by direct administration of amphetamine into several brain regions, including the amygdaloid complex, hippocampus, and caudate nucleus. Amphetamine is known to act by releasing the catecholamines epinephrine, norepinephrine, and dopamine from cells and block their reuptake. Amphetamine effects on memory appear to result primarily from influences on brain dopaminergic systems as well as influences on the release of peripheral catecholamines.

Amphetamine users often report that their "learning capacity" is enhanced by single doses of the substance. Since there are few systematic and well-controlled studies of the effects of amphetamine on memory in humans, however, it is not known whether such reports reflect subjective changes in perception and mood or effects on memory. Chronic amphetamine use is usually accompanied by a deterioration of memory function, an effect that subsides with cessation of use.

COCAINE

Despite the extensive use and abuse of COCAINE, little is known about cocaine effects on memory. Results of studies using rats and mice indicate that acute posttraining administration induces dose-de-

pendent effects comparable to those of amphetamine: Memory is enhanced by low doses and impaired by higher doses. The brain processes mediating cocaine influences on memory have not been extensively investigated. The effects appear to be mediated by influences on adrenergic and dopaminergic systems. Also, as with amphetamine, users of cocaine report that memory is enhanced by acute doses and impaired by chronic use. Systematic, well-controlled studies of the effect of cocaine on human memory are lacking. The effects on memory and intellectual functioning of other drugs—such as PHENCYCLIDINE (PCP), BARBITURATES, NICOTINE, and INHALANTS—are considered in connection with these agents and in separate articles.

(SEE ALSO: *Memory and Drugs; Research: Learning, Conditioning, and Drug Effects; Wikler's Pharmacologic Theory of Drug Addiction*)

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IVAN IZQUIERDO
JAMES L. MCGAUGH

MEPERIDINE Meperidine is a totally synthetic OPIOID analgesic (painkiller) with a structure quite distinct from MORPHINE, a natural OPIATE. Unlike morphine's rigid fused ring structures, the structure of meperidine is flexible; it is a phenylpiperidine and bends so that the key portions of the molecule can assume positions similar to those of morphine. A number of other compounds with similar structures are widely used in medicine, including loperamide (used primarily for treating diarrhea) and the extraordinarily potent ANALGESIC agents fentanyl, sufentanil, lofentanil, and alfentanil (for treating PAIN).

Meperidine is a compound with strong analgesic effects similar to morphine's, although greater amounts are needed to produce the same level of analgesia. It is one of the more commonly prescribed opioid analgesics and is better known under one of its brand names, Demerol. Given by injection, 100 milligrams of meperidine equals 10 milligrams of morphine. Meperidine can be administered orally as well as by injection but its potency it not as great following oral administration, so the dose must be increased proportionally. Like morphine, continued use of meperidine is associated with decreased analgesia—TOLERANCE—as well as PHYSICAL DEPENDENCE. As with the other opioids, ADDICTION (defined as a drug-seeking behavior) is not commonly observed with this drug when used for medicinal purposes, but meperidine is highly

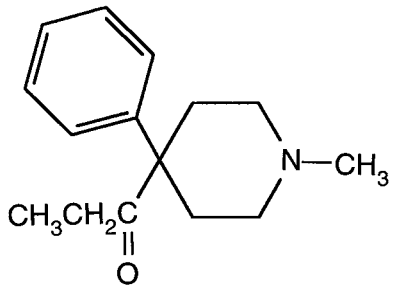

Figure 1
Meperidine

valued on the street and is widely abused, particularly in its injectable forms.

Medically, meperidine is a significant problem in patients with kidney conditions, where drug-removal from the body is impaired. Metabolized to normeperidine, a closely related compound, it is eliminated by the kidneys. In patients with kidney problems, this metabolite can accumulate to high levels, which can cloud mental processes and even produce convulsions. Since ELDERLY patients often have impaired kidney function, special care must be taken when using meperidine with them.

(SEE ALSO: *Addiction: Concepts and Definitions; Opioid Complications and Withdrawal*)

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GAVRIL W. PASTERNAK

MEPROBAMATE This is a SEDATIVE-HYPNOTIC drug that is now typically used to treat muscle spasms. Meprobamate is prescribed and sold as Deprol, Equagesic, Equanil, Meprospan, and Miltown. Because of its abuse potential, it is included in Schedule IV of the CONTROLLED SUBSTANCES ACT. It was first introduced into clinical medicine in 1955 for the treatment of ANXIETY. At the time it was thought to have specific antianxiety effects and to be quite different from other sedative-hypnotics. Also introduced at about the same time were chlorpromazine (Thorazine), which had remarkable ANTIPSYCHOTIC effects, and reserpine, which had tranquilizing as well as blood pressure-lowering effects. These three agents were considered the harbingers of the new era of PSYCHOPHARMACOLOGY and helped popularize the new term *tranquilizer*.

Within a year or two after its introduction, meprobamate had become one of the most widely prescribed drugs in the United States. It was not long however, before its distinction from other sedative-hypnotic agents was reassessed, and within a decade it was recognized that meprobamate shared many of the properties of other central nervous

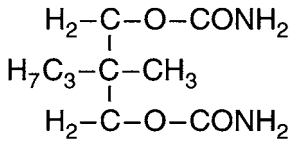

Figure 1
Meprobamate

system depressants, such as the BARBITURATES. By the early 1960s, its use for the treatment of anxiety was eclipsed by the BENZODIAZEPINES. Although it is prescribed as a muscle relaxant, the only use currently approved in the United States by the Food and Drug Administration is as a sedative-hypnotic.

Meprobamate has a number of side effects, including tremors, nausea, depression, and various allergic reactions. Continued use of high doses can result in TOLERANCE AND PHYSICAL DEPENDENCE. Convulsions and other signs of withdrawal are reported upon termination of high-dose treatment or inappropriate use.

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SCOTT E. LUKAS

MESCALINE This is a naturally occurring HALLUCINOGEN, one of the oldest PSYCHEDELIC substances known. It was first obtained from the PEYOTE cactus (*Lophophra williamsii* or *Anhalonium lewinii*), which grows in the southwestern United States and northern Mexico. Peyote buttons, the dried tops of the peyote cactus, were originally used by pre-Columbian Native Americans in those regions as an antispasmodic as well as for highly structured religious rituals; the button was eaten or

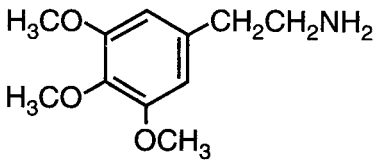

Figure 1
Mescaline

was steeped to make a drink. It continues to be used in ritual by the Native American Church.

Mescaline is a member of the phenethylamine-type family of hallucinogens, which includes DOM, MDA, and MDMA. The overall behavior effects of mescaline are very similar to those produced by LYSERGIC ACID DIETHYLAMIDE (LSD); however, it is approximately 100 to 1,000 times less potent than LSD, although the effects of mescaline last from 10 to 12 hours.

(SEE ALSO: *Psilocybin; Religion and Drug Use*)

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METHADONE Methadone (Dolophine) is a synthesized molecule with pharmacological actions very similar to those of the OPIOD drug, MORPHINE. Methadone serves an important place in the history of opioid ANALGESICS, since it is one of the first synthesized agents (1939). The ability to synthesize opioid analgesics from simple chemicals diminishes our reliance on natural products (such as morphine, CODEINE, and thebaine) to provide the base for many of the currently used opioid analgesics. Structurally, the drug does not look like morphine. Unlike the rigid fused ring structures of morphine, the structure of methadone is extremely flexible. It bends so that the key portions of the molecule can assume positions similar to those of morphine. The structure of methadone is very similar to that of propoxyphene (Darvon), a weaker opiate widely used to treat mild to moderate pain. It has two stereoisomers, but the (−)isomer is far more active than the (+)isomer.

Methadone can be administered orally, intramuscularly, or intravenously. It is well absorbed from the gastrointestinal track making it very useful orally. Its oral/parenteral ratio of potency is approximately two. Methadone is threefold more potent than morphine orally, but about equipotent when given by injection. It is metabolized by the liver to a variety of inactive compounds, which then are eliminated by the kidneys.

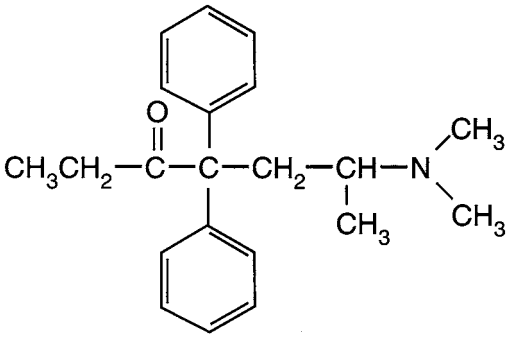

Figure 1
Methadone

Pharmacologically, methadone is used in the form of its hydrochloride salt. It has actions quite similar to morphine and works predominantly through *mu* opiate RECEPTORS. As an analgesic, methadone is similar in actions and in potency to morphine. It produces analgesia, as well as many of the side effects associated with morphine use, including respiratory depression and constipation. A major difference between methadone and morphine is methadone's long duration of action. Typically, the drug is given to patients every six to eight hours. This long duration of action can be very advantageous, particularly in patients who require the drug for long periods of time, such as cancer patients. However, there are some disadvantages. With a half-life ranging from twenty to thirty hours, it may take many days of continued dosing to reach constant (or steady-state) levels of the drug in the body. Thus, the full effect of a change in drug dose may not be seen for three or four days. This may make it difficult to adjust the dose for an individual patient. Increasing the dose too rapidly may even lead to delayed increases in its concentration in the body, far beyond those anticipated and, in some situations, may actually lead to an overdose. Continued administration of methadone will produce TOLERANCE AND PHYSICAL DEPENDENCE. The actions of methadone, like those of morphine, are readily reversed by ANTAGONISTS such as NALOXONE or NALTREXONE; however, these antagonists will also produce an immediate WITHDRAWAL syndrome in physically dependent people.

Despite its clear utility in the control of PAIN, the major use of methadone in the United States is in the treatment of HEROIN addicts. Although metha-

done must be administered approximately every six to eight hours to maintain analgesia, its slow rate of elimination prevents the appearance of withdrawal symptoms for over twenty-four hours. This slow appearance of withdrawal signs has made this agent very useful in maintenance programs, since it permits once-a-day dosing. With chronic administration of high doses of methadone, addicts become very tolerant, markedly limiting the euphoria an addict might obtain from illicit use of other opiates such as heroin. Thus, methadone minimizes occasional opiate use, is readily tolerated by the addicts, and can be administered once a day, which makes it easily dispensed. Methadone has been used clinically in maintenance programs and is one of the most effective treatment modalities available for opiate addicts.

(SEE ALSO: *Addiction: Concepts and Definitions; Methadone Maintenance Programs; Pain, Drugs Used in Treatment of; Treatment Types: Pharmacotherapy*)

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GAVRIL W. PASTERNAK

METHADONE MAINTENANCE PROGRAMS The history of methadone treatment offers a striking example of the benefits and limits of research findings on public attitudes and policies for methadone maintenance treatment. To understand methadone maintenance treatment, it is necessary to appreciate the profound stigma attached to both patients and treatment providers. This establishes the context for understanding how a modality with the most extensive research base in the addiction treatment field nonetheless can engender passionate dispute.

Methadone maintenance as a treatment modality was developed in the mid-1960s by Vincent Dole and Marie Nyswander in response to prevailing concerns about epidemic levels of heroin addiction and related health problems, mortality (especially among young people 15 to 35 years old) and high relapse rates. Methadone itself had been synthesized in Germany in World War II as a synthetic analgesic and was studied at the U.S. Public Health Service Hospital in Lexington, Kentucky, after the war. It was approved by the U.S. Food & Drug Administration in August 1947 for use in the treatment of pain. Its initial use in the treatment of addiction was to ease withdrawal in addicts being treated for heroin addiction; it was subsequently determined to be well suited to long-term maintenance treatment. As a treatment tool, methadone provides a safe and effective way to eliminate drug craving, withdrawal, and drug-seeking behavior, and free patients to lead productive lives. In conjunction with educational, medical, and counseling services, it has been thoroughly documented as enabling patients to discontinue or reduce illicit drug use and associated criminal activity, improve physical and mental well being, become responsible family members, further their education, obtain and maintain stable employment, and resume or establish a productive lifestyle. Despite three decades of research confirming its value, methadone maintenance treatment remains a source of contention among treatment providers, the public in general, and officials and policymakers in particular. Unlike controversies based on a difference of opinion between informed parties, debate about methadone usually involves several common misunderstandings about the drug and its uses.

COMMON MISUNDERSTANDINGS

Much of the uneasiness about methadone stems from the idea that it is "just substituting one addicting drug for another." Indeed, this is technically correct; methadone treatment is drug-replacement therapy in which a long-acting, orally administered preparation is substituted for a short-acting opioid that is used intravenously. The long-acting (24 to 36 hours) effect of preventing withdrawal allows most patients to receive a dose and function in a stable manner, without the four-hour cycles of euphoria and withdrawal that characterize heroin use. The objection that methadone is "addicting" reflects the recognition that the medication is dependence-producing. Addiction treat-

ment professionals increasingly distinguish between physical dependence and addiction, the latter being characterized by behavior that is compulsive, out of control, and persists despite adverse consequences. Chronic-pain patients will develop physical dependence though their overall functioning is improved. Appropriate prescribing of benzodiazepines for patients with anxiety disorders is another example of another dependence-producing drug used beneficially for thousands of patients. Although physical dependence is a factor to be considered, addiction specialists increasingly assess the extent to which the person's functioning and quality of life are improved or impaired in order to determine whether physical dependence is an acceptable consequence of medication use.

Another point of discord is the belief that "methadone keeps you high," a notion that reflects misunderstanding about the effects of a properly adjusted dose. Once stabilized, most patients experience little or no subjective effects; heroin addicts will readily state that they seek methadone to avoid becoming sick (prevent withdrawal effects), not to get high. When the patient's dose is being stabilized, he or she may experience some subjective effects, but the wide therapeutic window allows for dose adjustment between the points of craving and somnolence. Dose adjustment may take some weeks and may be disrupted by a variety of medical and lifestyle factors, but once achieved the patient should function normally. There is ample scientific evidence that the long-term administration of methadone results in no physical or psychological impairment of any kind that can be perceived by the patient, observed by a physician, or detected by a scientist. More specifically, there is no impairment of balance, coordination, mental abilities, eyehand coordination, depth perception, or psychomotor functioning. Recently, advocacy efforts have been successful on behalf of patients identified through workplace drug testing and threatened with negative consequences. It is anticipated that the Americans with Disabilities Act will further protect patients against such forms of discrimination.

A third point of resistance, objection to long-term or even life-long maintenance, is better addressed following the presentation of some basic information about opioid addiction and the nature of treatment.

HOW DOES METHADONE TREATMENT WORK?

Most addiction specialists agree that addictive disorders are complex phenomena involving the interaction of biologic, psychosocial, and cultural variables, all of which need to be considered to make treatment effective. Dole and Nyswander, who pioneered the use of methadone, held the view that there was something unique about opioid addiction that made it difficult for patients to remain drug-free. Although originally intended as a long-term treatment for a metabolic defect, many initially hoped that methadone could be used to transition heroin addicts to a drug-free lifestyle and then be discontinued. Research in the subsequent 30 years indicates that less than 20 percent will be able to discontinue methadone and remain drug-free. As his thinking evolved, Dole (1988) postulated that a receptor system dysfunction resulting from chronic use leads to permanent alterations which we do not currently know how to reverse. New brain imaging technology holds the promise of better understanding and, eventually, improved intervention, but in the interim it appears that methadone is corrective although not curative for the severely addicted person. Two important questions for future research are whether a preexisting condition enhances the vulnerability of some patients more than others, and whether long-term addicts can ever recover normal functioning without maintenance therapy.

For now, studies indicate that methadone is a benign drug which exhibits stability of receptor occupation and thus permits interacting systems to function normally. One example of this is the normalization of hormone cycles and the return of regular menstrual cycles in women. This distinguishes it from heroin, a short-acting narcotic producing rapid changes that make a stable state of adaptation impossible. Although tolerance develops to most effects, it is fortunate that even long-term use (30 years or more) does not produce tolerance to the reduced craving, or to the narcotic withdrawal prevention effect.

The desired response to methadone depends on maintenance of a stable blood level at all times. Appropriate doses usually keep the patient in the therapeutic range of 150 to 600 ng per mL in the blood and produce the stable state so important for rehabilitation. What is referred to as a "rush" or

"high" is the result of rapidly changing blood levels; thus, once therapeutic levels are achieved and maintained, the patient experiences little subjective effect.

Unfortunately, negative attitudes toward methadone have historically played a significant role in dosing practices, manifested in dose ceilings imposed by state or local regulations without regard to medical criteria. Such policies placed value on giving as little of the drug as possible (versus the therapeutic level needed to accomplish the goal), influenced in part by the belief (unsubstantiated) that lower doses would make it easier to discontinue methadone. It was common to have dose ceilings of 40 mg per day. It is now well established that this is inadequate to maintain the necessary plasma concentrations to be effective; the effective range is between 60 and 120 mg per day for most patients, with some needing less than 60 and others considerably more than 120 mg. The higher and more adequate doses are strikingly well correlated with reductions in illicit drug use and improved retention in treatment (GAO, 1990; Caplehorn & Bell, 1991). How painfully ironic to recall that patients on low doses who complained that "my dose isn't holding me" were often dismissed with the assertion that they were "merely engaging in drug-seeking behavior." And when the distressed patient then supplemented the methadone dose with heroin, it was concluded either that the patient was poorly motivated, or the treatment was ineffective. Studies by D'Aunno and Vaughan (1992) show that more than 50 percent of patients nationwide receive doses that are inadequate to prevent continued illicit narcotic use, indicating both poor physician training and inappropriate involvement by regulatory agencies and legislative policies.

Initial hopes to use methadone as a drug to transition patients to a medication-free life style have proven unrealistic. Studies indicate that although short-term abstinence is common, relapse is the norm for 80 percent or more (McLellan et al. 1983; Ball & Ross, 1991). Clinicians who have worked with this population over the long term believe that although lifestyle changes are essential to successfully discontinuing methadone, such changes in conjunction with high motivation will still be insufficient for most; neurobiological factors remain a deciding factor. Because the current treatment system, overburdened by regulations and inappropriate expectations, is dehumanizing for many, programs usually make efforts to assist the patient who wishes to taper off methadone. However, many of these programs attempt to create an environment in which the patient is encouraged to succeed, but also to resume methadone treatment promptly once relapse or the likelihood of it occurs.

Vincent Tobin, director of this methadone treatment center in Greenfield, Massachusetts, and registered nurse Mary Ann Gendreau await clinic clients, April 13, 2000. (AP Photo/Craig Line)

METHADONE AND OTHER DRUG USE

Methadone patients may engage in alcohol, cocaine, and other drug use prevalent in their communities. It is important to remember that methadone is opioid-specific and does not in itself increase or prevent other kinds of drug use. It does, however, offer the enormous advantage of making the patient accessible to other kinds of intervention. Rules governing take-home medication are intended to reduce the diversion of methadone onto the illicit market. At minimum, they mandate that the patient come to the clinic at least once weekly and, in most cases, even more frequently. Thus, the patient can be exposed to educational presentations and materials, and to counseling interventions as indicated by an individualized treatment plan, which is required as part of the treatment effort. Cocaine use has received particular attention, as it has been identified as increasing dropout, slowing progress, and undermining the gains of previously stable patients. Research and training efforts have been brought to bear on this problem. Alcohol use

remains a problem, particularly since many patients define their difficulty in terms of illicit drug use and are resistant to the notion of giving up drinking. With the blending of the "cultures" of alcohol and drug treatment providers, counselors are increasingly sophisticated about addressing problem drinking, although it is uncommon for programs to define goals for everyone in terms of abstinence from all intoxicants, as other parts of the treatment system do. Nonetheless, there is increasing sophistication in interventions, and programs have the added advantage of being able to dispense disulfiram with methadone when appropriate.

TREATING OPIOID-ADDICTED PREGNANT WOMEN

Methadone maintenance has been viewed as an effective treatment for opioid addiction in pregnant women since the early 1970s. In addition to the benefits of psychosocial interventions provided by the program, methadone maintenance treatment prevents erratic maternal opioid drug levels, thus protecting the fetus from repeated episodes of withdrawal. Programs either provide prenatal care onsite, or monitor the patient to see that prenatal care is obtained elsewhere, thus reducing the incidence of obstetrical and fetal complications, in utero growth retardation, and neonatal morbidity and mortality (Finnegan, 1991). Exposure to HIV infection through ongoing needle use is also reduced. Programs typically provide interventions around nutrition, parenting skills, exercise, and other related topics.

Methadone-maintained mothers produce offspring more similar to drug-free controls, in contrast to the poorer health status of offspring born to women using street drugs. It is clear that the most damaging consequences of opioid use during pregnancy occur with repeated episodes of intoxication and withdrawal (Jarvis & Schnoll, 1994). Although expectant mothers can be stabilized on methadone, body changes specific to pregnancy cause them to frequently develop increasing signs and symptoms of withdrawal as the pregnancy progresses, and they may need dose increases in order to maintain therapeutic plasma level and remain comfortable. Splitting the dose so that it can be ingested twice daily often produces better results, both reducing fetal stress and increasing the comfort of the pregnant woman, but local regulatory obstacles, not allowing patients to take half their daily dose out side the clinic, make this impractical for many programs.

There is inconsistent evidence to support the commonly held belief that the severity of the neonatal abstinence syndrome is proportional to the methadone dose, but many programs urge the expectant mothers to reduce their dose so the "baby won't be born addicted." In fact, the management of neonatal abstinence syndrome is relatively straightforward; fetal discomfort can usually be eliminated within hours and withdrawal can be accomplished within 14 to 28 days. No lasting impairment from these experiences has been demonstrated.

ADDRESSING PSYCHOSOCIAL ISSUES

Many existing methadone programs fall far short of the resources needed to do an effective job, but extensive research over a long period of time has clarified many of the treatment tasks. The stigma against heroin addicts in general and methadone patients in particular has created a treatment climate in which both patients and treatment providers may become demoralized about the value of the treatment endeavor. Often isolated from the mainstream, providers may not be able to obtain access to resources for patients on methadone. For example, methadone patients are often excluded from housing support or residential treatment. Nonetheless, there exists growing documentation that minimal intervention using methadone does reduce illicit drug use and hence needle sharing, and enhanced treatment accomplishes a great deal more (McLellan et al., 1993).

Historically, drug counseling has been provided in clinics by counselors who often have no credentials but are provided some training onsite. This counseling focuses on managing the patient's personal problems: problems specific to drug use, physical health, interpersonal relationships, family interactions, and vocational and educational goals. The counselor also performs the role of the case manager and is a liaison between physicians and medical institutions, courts and social services. Counselors also help the patient to develop coping strategies for current problems, perform initial screening for medication and other program services, and attend to issues concerning program

rules, privileges, and policies. The regulations governing methadone treatment are more complex, detailed, and restrictive than others in medicine or psychology, and maintaining a therapeutic alliance while meeting these obligations is a daunting task for clinical staff.

Studies of the drug-dependent population indicate that over 50 percent have a comorbid psychiatric disorder (Regier et al., 1990), and among the opioid-dependent population, depression is particularly common. Treatment outcome is improved by adding supplemental psychotherapy with professionally trained staff (Woody et al., 1983) for patients who meet the criteria for high psychiatric severity. It is important that such staff be well integrated into the treatment team. Medication may also be given concurrent with methadone, and methadone patient's use of antidepressants is increasingly common. Possible interaction effects are manageable with consistent monitoring and good staff teamwork. Psychotic conditions are relatively less frequent, but clinics are likely to have some highly disturbed individuals as part of their population and hence should be able to recognize and manage these patients appropriately. The patients benefit from the structure of frequent clinic attendance combined with the low psychological intrusiveness possible within the program.

It is also desirable for vocational interventions to be integrated into the program's mission, although the economic conditions in many urban areas also necessitate the development of alternatives to bring structure to daily life. Parenting classes that provide information and skill training and the opportunity to explore related issues are often well received by parents who feel the absence of good role models and skills.

Since twelve step programs actively promote abstinence from all potentially addictive drugs, this has been a barrier to methadone patients participating in them. Coupled with this are negative attitudes toward methadone and its users. Medication interventions such as methadone were not compatible with twelve step program participation in the minds of the Alcoholic's Anonymous' founders (Zweben, 1991), but meeting participants nonetheless may not always be open to methadone patients. In recent years, this climate has begun to change and methadone patients have started to increasingly attend twelve step meetings. Methadone maintenance programs are developing their own special meetings onsite, which in turn encourage patients' utilization of twelve step meetings in the larger community.

HIV/AIDS AND HEPATITIS C

A positive reexamination of methadone treatment has been greatly stimulated by documentation of its role in reducing the spread of HIV. Seroprevalence is much lower among those who have been on long-term maintenance, particularly those who entered treatment prior to the onset of the rapid spread of HIV in the local population (Hartel et al., 1988; Batki, 1988). Clinics provide accessibility to large numbers of intravenous drug users, making them an excellent site for prevention and education, screening, testing, and counseling. Because methadone patients have a continuing forum to discuss their life issues, counselors may be able to facilitate behavior change around the issues of safer sex practices and other high-risk behaviors. Further gains accrue as the patient progresses in treatment, as an abstinent person is in a better position to exercise good judgment than an intoxicated one. Currently, efforts are being made to integrate HIV/AIDS-related activities as fully as possible into methadone treatment programs.

The hepatitis C virus (HCV) has emerged as a problem of major significance, with many clinics reporting a prevalence upwards of 80 percent. Among those with HIV, coinfection with HCV is high. Inasmuch as 50 to 80 percent of new injectors become infected with HCV within 6 to 12 months, methadone maintenance will not reduce its spread as effectively as has occured with HIV. However, it does provide a structured system in which the patient can be monitored for good medical care, informed of emerging treatments, and educated about health practices to reduce the burden on the liver while more promising treatments are being developed.

WHAT THE FUTURE HOLDS

Methadone maintenance has demonstrated its effectiveness in reducing illicit drug use and facilitating the transition to a productive lifestyle. In the mid to late 1990s, two major scientific bodies reviewed the evidence on methadone maintenance and concluded it was an effective modality whose usefulness was greatly reduced by stigma and over

regulation (National Consensus Development Panel on Effective Medical Treatment of Opiate Addiction, 1998; Rettig & Yarmolinsky, 1995). The documents produced by these groups have been instrumental in efforts around the country to reduce barriers and make the delivery system more flexible and responsive to patient needs.

Research including long-term followup indicates that stabilized and socially responsible methadone patients can be safely given a month of take-home medication by physicians in an office-based practice (Novick & Joseph, 1991; Novick et al., 1994). The federal government is in the process of formulating guidelines and regulations to permit treatment to occur in the office of a physician affiliated with a methadone clinic. For the patient, this represents a significant opportunity to shift from the traditional treatment system, segregated from the rest of medical practice since the 1960s, to the mainstream medical system. Although these changes are likely to be implemented most easily with stabilized methadone patients, pilot programs are underway to admit new patients (such as those in rural areas) to an office-based practice. Concurrently, the development of an accreditation mechanism is intended to simplify regulations and emphasize clinical practice guidelines that are more easily modified in response to emerging research findings. These activities will likely reduce barriers to treatment and allow for the development of less restrictive treatment settings.

Other maintenance pharmacotherapies, particularly LAAM and buprenorphine, have been developed and will broaden the options and possibilities for effective intervention. Federally sponsored training efforts have improved the quality of care and will continue to be essential to disseminating current information and providing opportunities for skill development. Slowly, patients have emerged as visible examples of success and to serve as role models for others. Barriers to participation in residential treatment are beginning to be removed. It is hoped that developments will engender future gains and allow this modality to gain the acceptance it so greatly deserves.

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METHAMPHETAMINE Methamphetamine (also called METHEDRINE) is a potent PSYCHOMOTOR STIMULANT with a chemical structure similar to AMPHETAMINE. Methamphetamine's stimulant effects on the central nervous system are more pronounced than those of amphetamine, while its peripheral effects (e.g., cardiovascular and gastrointestinal) are less marked. Like amphetamine, it causes increased activity, increased talkativeness, more energy and less fatigue, decreased food intake, and a general sense of well-being. Injecting the drug intravenously results in the production of a "rush," described by some as the best part of the drug effect. Methamphetamine is more soluble than DEXTROAMPHETAMINE, and, when available, of this group is generally the illicit user's drug of choice for intravenous injection—although dextroamphetamine dissolves sufficiently to permit intravenous use.

Japan was the first nation to experience a major epidemic of methamphetamine use. Immediately following World War II, large quantities of methamphetamine, which had been produced to keep combat troops alert, were released for sale to the Japanese public. Within a short time there was widespread use and abuse of the drug, much of it

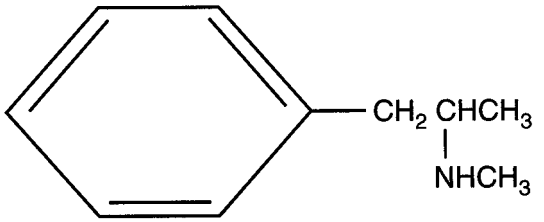

Figure 1
Methamphetamine

intravenously. At the peak of the epidemic, more than a million users were involved. Despite the experience of the Japanese, the belief persisted in the United States that amphetamines did not lead to serious compulsive use, and these drugs were not subject to any special regulatory controls like the ones governing the availability of the opioid drugs until 1964.

The first methamphetamine ("speed") epidemic in the United States began in the 1960s in the San Francisco area. A number of physicians there were prescribing the drug to HEROIN abusers for self-injection—to treat their heroin dependence by substituting methamphetamine. The drug achieved widespread popularity, with increasing numbers of people claiming heroin abuse and requesting prescriptions for methamphetamine. When the sale of intravenous methamphetamine to retail pharmacies was curtailed in the mid-1960s, illicitly synthesized methamphetamine began to appear. By the late 1960s a substantial number of users throughout the United States were injecting high doses of this illicit methamphetamine in cyclical use patterns—resulting in toxic syndromes that included the development of a paranoid psychosis (i.e., amphetamine psychosis).

Although illicit methamphetamine never completely disappeared from street use, its availability was considerably reduced by the 1970s. This trend began to reverse during the 1980s, with pockets of methamphetamine abuse occurring in the United States. Hawaii was the first area of the United States to experience the most recent methamphetamine outbreak, mostly in the form of smokable methamphetamine. Initial reports of smoking methamphetamine occurred in late 1986, with increases occurring about a year later, and a more sustained increase occurring in 1988 and 1989. Called "ice" or "crystal," this is the same sub-

stance as "speed," which was abused several decades earlier.

Methamphetamine, sold as "ice," is a large, usually clear crystal of high purity (greater than 90%) that is generally smoked using a glass pipe with two openings, much like a CRACK-cocaine pipe. Because it is a large crystal, it is difficult to adulterate with inert substances, a property that makes it extremely desirable to purchasers of illicit products. The smoke is odorless and, unlike crack, the residue of the drug stays in the pipe and can be resmoked. The effect is long-lasting, reported by users to be as long as twelve hours, although it is likely that this prolonged effect is due to the use of several doses.

Like COCAINE, methamphetamine abuse occurs in binges, with users taking the drug repeatedly for several hours to several days. During this time the user generally neither eats nor sleeps. Ending a methamphetamine binge is accompanied by fatigue, depression, and other "crash"-related effects. One of the most profound of the toxic effects of repeated methamphetamine use is the development of a paranoid psychosis, often indistinguishable from schizophrenia. With time off the drug, this psychosis generally resolves, although it can reappear if the user returns to methamphetamine abuse. Some Japanese psychiatrists have reported that methamphetamine psychosis may persist for many months.

(SEE ALSO: *Amphetamine Epidemics; Designer Drugs; Epidemics of Drug Abuse*)

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MARIAN W. FISCHMAN

METHANOL Methanol is the simplest alcohol, containing only one carbon atom, four hydrogen atoms, and one oxygen atom (CH_3OH). It is also called methyl alcohol, WOOD ALCOHOL, carbinol, wood naphtha, wood spirit, pyroxylic spirit, and pyroligneous alcohol or spirit. It is a flammable, potentially toxic, mobile liquid, used as an industrial solvent, in antifreeze, and in chemical manufacture. Ingestion may result in severe acidosis, visual impairment, and other effects on the central nervous system. Methanol does not produce significant inebriation unless a very large amount is consumed.

Methanol itself is not toxic, but it is metabolized by enzymes in the body to create formaldehyde and formic acid—both of which are very toxic substances. The formic acid can cause blindness. Ethanol (ethyl alcohol—drinking alcohol) can be used as an antidote for methanol poisoning, because it competes with the methanol for the enzyme. As a result, there is a delay of formaldehyde and formic acid production, and these toxic substances do not rise to such high levels. Although methanol is frequently added to ethanol-based cleaning solutions, its addition denatures the solution and makes it unsafe to drink. Only desperate alcoholics will drink methanol, but it is sometimes drunk by accident by people experimenting with various alcohol substitutes.

(SEE ALSO: *Alcohol*)

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S. E. LUKAS

METHAQUALONE This is a nonbarbiturate, short-acting SEDATIVE-HYPNOTIC drug that has been used to treat insomnia. It was originally introduced in 1951 as a treatment for malaria. In the 1960s and 1970s, it became a popular drug of abuse among college students. Frequently called Quaaludes or "Ludes," the drug, like the short-

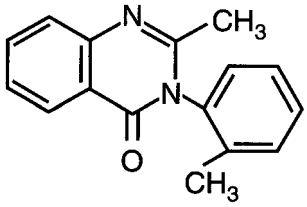

Figure 1
Methaqualone

acting BARBITURATES, produced euphoric effects; some users claimed it had APHRODISIAC effects.

It is usually taken in pill form, and depending on the dose, the effects last a few hours. The body eliminates about half of the ingested dose in about ten to forty hours, so that even forty-eight hours after ingestion, some drug may still be present. Prolonged use of methaqualone in high doses can lead to TOLERANCE AND PHYSICAL DEPENDENCE, and abrupt cessation of daily ingestion can result in WITHDRAWAL symptoms that are quite similar to those seen in barbiturate withdrawal. Fatal convulsions have resulted from sudden withdrawal. Fatal overdoses can occur when the drug is used alone, but especially when it is mixed with ethanol (ALCOHOL) and/or barbiturates. Because it was so commonly abused in the United States, the drug was shifted to Schedule I of the CONTROLLED SUBSTANCES ACT in the 1980s. Thus, it can no longer be prescribed and its nonmedical use is subject to severe criminal penalties. Although it is rarely used illicitly in the United States, it is still available in other countries and is a drug of abuse in some.

(SEE ALSO: *Addiction: Concepts and Definitions*)

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SCOTT E. LUKAS

METHEDRINE Methedrine was the proprietary name given METHAMPHETAMINE hydrochloride by the pharmaceutical company Burroughs Wellcome. It was sold in ampules and until 1963–1964 was readily available by prescription. Methedrine (or "meth") became one of the street names of

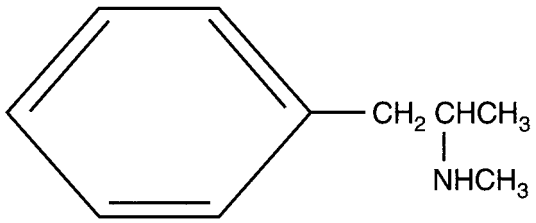

Figure 1
Methedrine

methamphetamine during the 1960s and early 1970s when high-dose methamphetamine ("speed") was a major drug of abuse. It was a particular problem in northern California where, after the manufacturer withdrew commercially made Methedrine from the market in 1963, large quantities of black-market, illicitly synthesized methamphetamine became available for sale.

(SEE ALSO: *Amphetamine Epidemics; Designer Drugs; Epidemics of Drug Abuse*)

MARIAN W. FISCHMAN

METHYLPHENIDATE This is a central nervous system STIMULANT, structurally related and with similar effects to AMPHETAMINE. It is used by prescription as Ritalin. It was initially marketed as a mood enhancer in the mid-1950s and described as having less abuse potential than amphetamine; however, within a few years a number of dramatic reports of its abuse and toxicity were published. Methylphenidate is commercially available (by prescription) in pill form, reaching peak effect in one to two hours. Like the amphetamines and other stimulant drugs, methylphenidate is a controlled substance, placed in Schedule II of the CONTROLLED SUBSTANCES ACT to indicate that although it has medical utility it also has substantial ABUSE LIABILITY.

In most people, methylphenidate increases general activity, decreases food intake, produces positive subjective effects (an elevated mood), and can interfere with sleep. With continued use, tolerance can develop to these effects and users will often escalate their doses to achieve the desired effects of their initial doses of methylphenidate. Continued high-dose methylphenidate use can result in toxic consequences similar to those seen after amphetamine use—with ANXIETY, sleeplessness, and even-

tually a toxic paranoid psychosis. High-dose users often begin with oral methylphenidate use but switch to injecting the drug in order to maximize the effect and achieve the initial "rush" that is typical of intravenous drug abuse. Commercially manufactured methylphenidate pills (the only form available) contain talcum, an insoluble substance, which can cause toxic effects (such as abcesses) when the pills are dissolved in water and injected intravenously or under the skin.

Laboratory animals tested with methylphenidate show increases in locomotor activity after single doses, increased sensitivity to this effect after repeated doses, and the development of stereotyped repetitive behavior patterns after chronic dosing. In addition, these animals remain more responsive to methylphenidate even after the drug treatment has been discontinued. It has been suggested that the continuous repetition of behavior that characterizes the response to chronic methylphenidate treatment is a good model for the human stimulant psychosis and, as in animals, humans who use high doses become increasingly sensitive to stimulants such as methylphenidate, with psychosis increasingly likely at lower doses after its initial appearance. There are, however, no data to support this hypothesis.

In addition to its action as an appetite suppressant, methlyphenidate has been found to have other therapeutic utility. Like *d*-amphetamine, it has been used successfully in the treatment of ATTENTION-DEFICIT HYPERACTIVITY DISORDER (ADHD), a syndrome that first becomes evident during childhood and is characterized by excessive activity and difficulty in maintaining attention. Because of its relatively short half-life, two or three doses of methylphenidate are required each day, although recently a slow-release form of the medication has become available, promising more stable blood levels with only a single daily dosing. Methylphenidate has been shown to alleviate or moderate many of the symptoms of this disorder, although it is not effective in all cases and its long-term efficacy is not well understood.

Side effects of treatment can include insomnia, loss of appetite, and weight loss, all effects of stimulant drugs in general. In addition, concern about the longer lasting effects on learning and cognition in youngsters maintained on this drug for many years has made practitioners cautious and often unwilling to prescribe it. Recent research and practice, however, has supported methylphenidate as the stimulant of choice for treating this disorder. As with the amphetamines, methylphenidate is also effective in the treatment of narcolepsy, in which sudden attacks of sleep can occur unexpectedly.

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MARIAN W. FISCHMAN

MEXICO AS DRUG SOURCE Drug control in Mexico is unique—the reason both for Mexico's paradoxical success as well as for its ongoing difficulty in managing the issue. Believing that destruction at their agricultural source is the most effective way to reduce supplies and halt trafficking, Mexico began to spray the OPIUM poppy (PAPAVER SOMNIFERUM) and MARIJUANA plant (CANNABIS SATIVA) in late 1975 with the herbicides paraquat and 2,4-D. Plants, not people, became the target in the 1970s and 1980s. Until the early 1990s, the drug-eradication program was the centerpiece of Mexico's program. With the 1990s increase in Colombian cocaine transiting Mexico, the Mexican government increased its efforts to work with the United States in halting COCAINE smuggled through Mexico, sharing intelligence, extraditing non-Mexican nationals, and reducing drug-related corruption. However, by 2000 the government's efforts remained hampered by corruption in the police and military. Tensions between the United States and Mexico increased as U.S. officials and legislators questioned the ability of Mexico to curb drug trafficking, which had grown dramatically as more enforcement efforts were placed on other South American countries, including Columbia. Several prominent officials were found to have worked with drug traffickers to subvert reform efforts. Finally, the election of Vicente Fox as Mexico's president in 2000 signaled the possibility of political change, as Fox became the first president not elected from the Institutional Revolutionary Party (PRI) in the modern era.

Mexico's principal agency for drug control is the attorney general's office, but the Mexican military has also been involved with manual crop eradication and operational support for herbicidal spraying. The Mexican military has also become involved in tactical reconnaissance, interdiction, and destruction of secret landing strips. In the late 1990s, the Mexican government established the Federal Preventative Police (FPP) to integrate the law enforcement responsibilities of several existing federal agencies and to focus on crime prevention and public security. Historically, the government of Mexico has increased its effectiveness in the drug-control field as a positive response to U.S. diplomatic and enforcement pressures. During the 1990s, under increased U.S. pressure, the two countries agreed to a Binational Drug Strategy.

UNIQUENESS OF MEXICO

At least four factors set Mexico apart from its drug-producing neighbors to the south, creating an environment for drug control. First, Mexico is the only country in the Western Hemisphere that produces significant amounts of opium poppy and HEROIN with little use by its people. Although large numbers of its people abuse marijuana and INHALANTS, Mexico may be the only opium-producing country in the world with almost no domestic market. Yet, Mexico shares a 1,900-mile (3,057-km) border with the U.S.—a country that has one of the world's largest and most lucrative markets for heroin.

Second, powerful drug rings have bought power and influence in several Latin American countries, yet, unlike their Peruvian and Colombian counterparts, Mexican drug traffickers have no symbiotic relationship with ideological, terrorist-oriented, political factions—whose goal is to change the prevailing political order. Nevertheless, during the 1990s drug-influenced political corruption became a very public problem, one which contributed to the election of President Fox in 2000.

Third, growing opium in Mexico is relatively recent; it has always been illegal and involves only a small number of citizens. Illicit opium and marijuana are grown not on privately owned plots but on open unowned (hence, government) land, largely as extra-cash crops, not as subsistence crops. If these illicit crops were all destroyed, growers would not starve. Unlike coca—which is a legal crop (that can provide the raw material for an illegal commodity) and has been cultivated for centuries in Bolivia and Peru—Mexico's opium crop has never become the center of a social, cultural, or agricultural economy.

Fourth, Mexico is a relatively wealthy country with vast natural and human resources. Mexico has shown its ability to build an infrastructure to implement an ongoing drug-control campaign. Beginning in the mid-1970s, Mexico started the world's first successful herbicidal opium-eradication program, which continues today. However, these strengths have been severely tested, as an economic downturn in the 1990s and increased drug trafficking has strained the nation's ability to control drug crime.

CAMARENA MURDER

Drug control has been an important issue between the United States and Mexico since the 1960s. The abduction and murder of U.S. DEA agent Enrique Camarena in Mexico in February 1985 elevated the drug issue on the bilateral diplomatic agenda of the two countries. The murder focused public attention on the perhaps decreasing effectiveness of Mexican drug-control efforts and represented a turning point in U.S.-Mexican relations. After Camarena's murder, drugs became a confrontational issue at uncharacteristically high levels of the two governments. Both the U.S. secretary of state and Mexico's foreign minister discussed the murder and subsequent government response as a paramount diplomatic issue; drug control was no longer treated only as a law-enforcement issue between the two countries. In response to continuing U.S. pressure, the Mexican government took a series of actions that resulted in the apprehension and incarceration of the several drug traffickers responsible. Nevertheless, tensions between the two governments remained high throughout the 1990s, as trafficking and corruption increased.

HISTORICAL ROOTS

Mexico's international drug-control efforts have their roots in the SHANGHAI Convention of 1909 and the Hague Opium Convention of 1911–1912. In 1923, Mexico's President Alvaro Obregon prohibited the production of opium and condemned

Officials from the Mexican Attorney General's office display to the media one ton of confiscated marijuana and three suspects in Mexico City, March 1, 1996. (AP Photo/Jose Luis Magana)

what was then widespread and increasing drug-induced violence. In 1934, President Cardenas del Rio created the first centralized narcotics administrative unit in the government.

After the United States entered World War II in 1941, Mexico was asked to provide opium for the war effort, since it was processed into MORPHINE, a medication used extensively for war-related wounds. In both Mexico and the United States, HEMP was grown to fill U.S. military need for rope and cordage; hemp is processed from *Cannabis sativa*, which is also used as marijuana. By mid-1943, opium constituted the most profitable cash crop in Mexico's northwestern state of Sinaloa. Despite Mexico's efforts to control the production of these crops after the war, drugs were grown, processed, and smuggled into the United States from Mexico.

In the late 1960s and early 1970s, Mexico soon became the major supplier for the illicit U.S. heroin market when Turkey prohibited opium cultivation and the French Connection had been ended. Consequently, in the fall of 1969, the U.S. BUREAU OF NARCOTICS AND DANGEROUS DRUGS (the predecessor organization of DEA) and the U.S. Customs Service initiated Operation Intercept—a three-week operation that subjected every person crossing the border in the San Ysidro, California, area to intensive baggage and body searches. The economic losses and dismay on both sides of the border prompted termination of the operation—but not before focusing attention on the volume of drugs entering the United States from Mexico. The Mexican government then began to locate and manually destroy the poppy fields—the source, at that time, of all the heroin produced in the Western Hemisphere. Originally, the search for poppy fields was made in small planes that flew over mountain zones where crops were suspected to be growing on remote plots of government land.

Prior to 1975, once the poppy had been spotted and the approximate location registered in official correspondence, military squads were sent to destroy the plants by cutting them down. In 1975, the Mexicans began to use the most modern technology—a system called Multi-Opium Poppy Sensing (MOPS), which used multispectral sensing cameras on board low-flying aircraft to read and print images from the electromagnetic spectrum. In nature, every substance emits its own unique electromagnetic waves that can be read on the color spectrum using special cameras. The fields were then destroyed by aerial application of the contact herbicides 2,4-D and paraquat. By the 1990s, a fleet of nearly 120 aircraft were being used.

MEXICAN GOVERNMENT ORGANIZATIONAL STRUCTURE

Organizationally, the Mexican attorney general's office plans and implements the drug-eradication campaign. Nearly 700 civilian pilots, mechanics, communications experts, and technical personnel make the campaign as effective as possible—working specific zones and sectors, with a coordinating office in each zone. Forward operating bases connect all the zones to Mexico City via a sophisticated communications system. Mexico's military is also used to stop the illicit cocaine that transits Mexico from South America to the United States, exchanging intelligence and training, and destroying clandestine trafficker landing strips.

ERADICATION RESULTS

Between 1982 and 1989, Mexico's rapidly deteriorating economy, bureaucratic inertia, technical inefficiency, poor management, low morale, complacency, and corruption led to the decreased effectiveness of the eradication program. Countermeasures by growers who planted smaller fields, at higher altitudes, under cover of foliage, during more than the two traditional growing seasons, fur-

ther decreased program success. In the mid-1970s, the eradication campaign was managed in large part by specialized organizations of both governments (Mexico's attorney general and U.S. law-enforcement units). In the mid-1980s, the Camarena murder took the campaign out of the strict purview of the specialist law-enforcement agencies and into the diplomatic arena. In the 1990s, the drug-control efforts increased to include interdiction of South American cocaine traveling through Mexico but destined for the United States. In 1991, the Mexican government increased its eradication of opium by 40 percent over 1990; and its eradication of marijuana by 60 percent. The drug eradication program has had dramatic results during the 1990s. Marijuana production dropped steadily during the 1990s, while opium production dropped to its second lowest level in the 1990s. Nevertheless, the U.S. government has found that most of the cocaine and much of the marijuana, heroin and methamphetamine consumed in the U.S. comes through Mexico. Mexican drug networks control a substantial part of the illicit drugs distributed in the United States.

(SEE ALSO: *Bolivia; Colombia; Drug Interdiction; International Drug Supply Systems*)

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MICHIGAN ALCOHOLISM SCREENING TEST (MAST) This is a brief self-report questionnaire designed to detect ALCOHOLISM (Selzer, 1971). It is widely used in clinical and research settings. The twenty-four scored items assess symptoms and consequences of ALCOHOL abuse, such as guilt about drinking; blackouts; DELIRIUM TREMENS; loss of control; family, social, employment, and legal problems following drinking bouts; and help-seeking behaviors, such as attending ALCOHOLICS ANONYMOUS meetings or entering a hospital because of drinking. Several shorter versions of the MAST have also been developed including the thirteen-item Short-MAST (Selzer, Vinokur, & van Rooijen, 1975) and the ten-item Brief-MAST (Pokorny, Miller, & Kaplan, 1972).

To complete the MAS, individuals answer yes or no to each item. The items are weighted on a scale of 1 to 5, with items concerning prior alcohol-related treatment experiences and help-seeking behaviors receiving higher weights. The total MAST score (range: 0–53) is derived by adding the weighted scores from all items that are endorsed. Studies indicate that the long version of the MAST possesses good internal-consistency reliability, as indicated by Cronbach's alpha coefficients of .83 to .93 (Gibbs, 1983). Therefore, the scale does appear to measure a unitary construct.

Selzer (1971) originally recommended adopting a cutting score of 5 or higher for a diagnosis of alcoholism with the MAST. However, since this cutting score was shown to produce a relatively high percentage of false positives (Gibbs, 1983), Selzer, Vinokur, and van Rooijen (1975) suggested the following cut points: 0 to 4, not alcoholic; 5 to 6, maybe alcoholic; 7 or more, alcoholic. Skinner (1982) recommended that scores of 7 to 24 be regarded as clear evidence of alcohol problems, and

that scores of over 25 be considered evidence of substantial alcohol problems. In a recent study, Ross, Gavin, and Skinner (1990) compared scores on the MAST to diagnoses of alcoholism obtained from the National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS) (Robins, Helzer, Croughan, & Ratcliff, 1981). In this study, the MAST cutting score that yielded the highest overall accuracy was 13 or greater.

The validity of the MAST has been examined in a number of studies in which MAST scores, or scores from the shorter versions of the instrument, were compared to other measures of drinking status, including diagnostic interviews, physicians' diagnoses, and other self-report instruments. In reviewing twelve of these studies, Gibbs (1983) concluded that MAST diagnoses agreed with diagnoses of alcoholism reached through other assessment procedures in about 75 percent of cases. Where inconsistencies between results were found, it was found that the MAST tended to overdiagnose alcoholism. This probably reflects the fact that a cutting score of 5 or higher on the MAST was used in these studies. By adopting a cutting score of 13, Ross et al. (1990) were able to achieve a greater degree of agreement when comparing MAST scores to DIS-derived diagnoses.

As with any instrument that relies on the veracity of self-report information, the reliability and validity of the MAST is dependent on the willingness of the interviewee to answer the items truthfully. All the items possess high face validity, which means it is relatively easy to answer them so as to appear non-alcoholic. The MAST may therefore not be a useful screening tool with individuals who are motivated to conceal their alcohol problems.

(SEE ALSO: *Addiction Severity Index; Diagnosis of Drug Abuse: Diagnostic Criteria; Diagnostic and Statistical Manual; Disease Concept of Alcoholism and Drug Abuse; Minnesota Multiphasic Personality Inventory*)

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A. THOMAS MCLELLAN

MIDDLE EAST AS DRUG SOURCE *See* International Drug Supply Systems

MILITARY, DRUG AND ALCOHOL ABUSE IN THE UNITED STATES Drug and alcohol use have historically been common among military personnel. Drugs have been used by soldiers to reduce pain, lessen fatigue, and increase alertness, or to help them cope with boredom or panic that accompany battle. During the U.S. Civil War, medical use of opium resulted in addiction among some soldiers. In the modern U.S. military, drug use became a recognized problem during the Vietnam War in the late 1960s and early 70s. Approximately 20 percent of Vietnam War veterans reported having used narcotics (e.g., heroin, opium) on a weekly basis, and 20 percent also were considered to be addicted based on reported symptoms of dependence (Robins, Helzer, & Davis, 1975). Although few personnel continued using heroin when they returned home, there were concerns about addiction.

Similar to drug use, heavy drinking in the military has been an accepted custom and tradition (Bryant, 1979; Schuckit, 1977). In the past, alcohol was thought to be a necessary item for subsistence and morale and, as such, was provided as a

daily ration to sailors and soldiers. Within the predominantly male U.S. military population, heavy drinking and being able to "hold one's liquor" have served as tests "of suitability for the demanding masculine military role" (Bryant, 1974). A common stereotype has been to characterize hard-fighting soldiers as hard-drinking soldiers. Alcoholic beverages have been available to military personnel at reduced prices at military outlets and until recently during "happy hours" at clubs on military installations (Bryant, 1974; Wertsch, 1991). In addition, alcohol has been used in the military to reward hard work, to ease interpersonal tensions, and to promote unit cohesion and camaraderie (Ingraham, 1984).

Drug and alcohol abuse are strongly opposed within the U.S. armed forces because of their negative effects on the health and well-being of military personnel and because of their detrimental effects on military readiness and the maintenance of high standards of performance and military discipline (Department of Defense, 1997). In the U.S. military, drug abuse is defined as the wrongful use, possession, distribution, or introduction onto a military installation of a controlled substance (e.g., marijuana, heroin, cocaine), prescription medication, over-the-counter medication, or intoxicating substance (other than alcohol). Alcohol abuse is defined as alcohol use that has adverse effects on the user's health or behavior, family, community, or the Department of Defense (DoD) or that leads to unacceptable behavior.

DEVELOPMENT OF MILITARY POLICY

The DoD convened a task force in 1967 to investigate drug and alcohol abuse in the military and in 1970 formulated a drug and alcohol abuse policy based on task force recommendations. The policy emphasized the prevention of drug and alcohol abuse through education and law enforcement procedures focusing on detection and early intervention (DoD, 1970, 1972). However, treatment was provided for problem users with an emphasis on returning them to service.

In response to continuing public concern about reports of serious drug addiction among U.S. forces in Southeast Asia, President Nixon in 1971 directed the DoD to take additional measures to address the drug problem. The result was the establishment of a urinalysis testing program that initially consisted of mandatory testing for service members leaving Southeast Asia and grew to include mandatory, random urinalysis for all U.S. forces worldwide. The program was discontinued for a period because of difficulties implementing it on a large scale, its high costs, and a court challenge that the Fifth Amendment protection against self-incrimination was being violated (U.S. v. Ruiz 1974).

The reaction to the crash of a jet on the aircraft carrier *Nimitz* in 1981 again focused public attention on the military's drug abuse problem, particularly marijuana use. Autopsies of fourteen Navy personnel killed in the crash showed evidence of marijuana use among six of the thirteen sailors and nonprescription antihistamine use by the pilot. The armed forces reinstituted urine testing for drugs in 1981 as a result of this incident and other concerns about drug use in the military. New breakthroughs in drug- testing confirmation procedures and more rigorous procedures for tracking urine samples overcame earlier legal objections. Urine tests, which are conducted either randomly or when a person is suspected of using drugs, are a major tool for the detection and deterrence of illicit drug use (DoD, 1997).

U.S. military substance use policy has been updated periodically since the early 1970s and currently is one of zero tolerance that includes an emphasis on preventing and detecting abuse and either discharging abusers from the military (the approach generally followed for drug abuse) or providing treatment and rehabilitation (the approach generally followed for alcohol abuse) (see Bray et al., 1993, 1999a for more detailed discussions of the development of military policy).

WORLDWIDE SURVEY SERIES

To help monitor the extent of drug and alcohol abuse, the DoD initiated a series of worldwide surveys among active-duty military personnel in the Army, Navy, Marine Corps, and Air Force. The first survey was conducted by Marvin Burt Associates in 1980 (Burt et al., 1980) and the others by Robert Bray and his colleagues at Research Triangle Institute in 1982, 1985, 1988, 1992, 1995, and 1998 (Bray et al., 1983, 1986, 1988, 1992, 1995, 1999b). The goals of the surveys have been to provide data to help assess the prevalence, correlates, and consequences of substance abuse and health in the military.

The surveys have all been conducted using similar methods. Civilian researchers first randomly selected a sample of about sixty military installations to represent the armed forces throughout the world. At these designated installations, they randomly selected men and women of all ranks to represent all active-duty personnel. Civilian research teams administered printed questionnaires anonymously to selected personnel in classroom settings on military bases. The few personnel (about 10%) who were unable to attend the group sessions (e.g., were on leave, sick, or temporarily away from the base) were mailed questionnaires and asked to complete and return them. Participants answered questions about their use of illegal drugs (e.g., marijuana, cocaine, heroin), the misuse of prescription drugs (e.g., stimulants, tranquilizers), about the frequency and amount of alcohol use, and problems resulting from drug or alcohol use. These data collection procedures yielded from over 15,000 to nearly 22,000 completed questionnaires for the various surveys. From 59 percent to 84 percent of those eligible to take part actually did so.

TRENDS IN DRUG AND ALCOHOL USE

Figure 1 presents trends over the seven worldwide surveys on the percentage of the active-duty military force who engaged in any illicit drug use or heavy alcohol use during the thirty days prior to the survey. Any illicit drug use was defined as use one or more times during the past thirty days of marijuana/hashish, cocaine, inhalants, hallucinogens, heroin, and nonmedical use of prescription-type drugs, including stimulants, sedatives, tranquilizers, or analgesics. Heavy alcohol use was defined as five or more drinks per typical drinking occasion at least once a week. As shown in Figure 1, use of any illicit drug declined sharply from just under 28 percent in 1980 to about 3 percent in 1998; heavy drinking declined significantly from approximately 21 percent in 1980 to just above 15 percent in 1998, although the decrease was less dramatic than for drug use. Heavy drinking by itself does not constitute alcohol abuse, but it does indicate drinking levels that are likely to result in negative consequences.

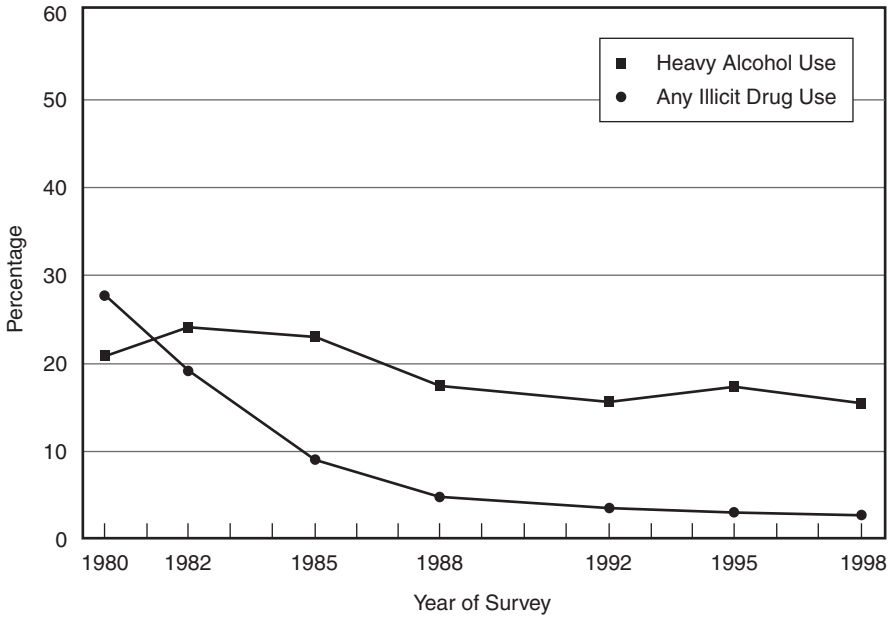

Figure 1
Trends in Any Illicit Drug Use and Heavy Alcohol Use, Past 60 Days, Total Department of Defense, 1980–1998

EFFECTS OF DEMOGRAPHIC CHANGES

Despite the significant downward trends in illicit drug use and heavy drinking noted in Figure 1, the question arises whether these declines are due to military programs and policies or to some alternative explanation. One possible explanation for the changes could be shifts in the demographic composition of the armed forces between 1980 and 1998. Members of the military in 1998, for example, were more likely to be older, to be officers, to be married, and to have more education than in 1980. These characteristics are also associated with less substance use. For example, 60 percent of personnel in 1998 were married compared to 53 percent in 1980; 61 percent were aged twenty-six or older in 1998 compared to 43 percent in 1980.

Analyses that adjusted for demographic differences across survey years from 1980 to 1998 showed that illicit drug use had the same significant decline as found before the adjustment, whereas heavy alcohol use did not. This suggests that the decline in illicit drug use shown in Figure 1 *was not* explained by shifts in the demographic composition of the military population, whereas the decline in heavy drinking *was* largely explained by demographic changes. Stated another way, if the demographic composition of the military in 1998 was like the composition in 1980, rates of illicit drug use in 1998 would still be notably lower, but rates of heavy drinking between these two survey years would have been about the same.

MILITARY AND CIVILIAN COMPARISONS

Another possible explanation for the trends in drug and alcohol use observed in Figure 1 is that the military may simply mirror similar trends occurring among civilians. Drug use among civilians has declined substantially in recent years (Office of Applied Studies [OAS], 1999), whereas declines in alcohol use among civilians have been more moderate (Clark & Hilton, 1991). To address this issue, data were compared for illicit drug use and heavy alcohol use among military personnel and civilians. Military data were drawn from the 1998 DoD survey, and civilian data from the 1997 National Household Survey on Drug Abuse (NHSDA), a nationwide survey of drug abuse. Military and civilian datasets were equated for age and geographic location of respondents, and civilian substance use rates were standardized (adjusted) to reflect the demographic distribution of the military.

Standardized comparisons showed that military personnel (about 3 percent) were significantly *less*

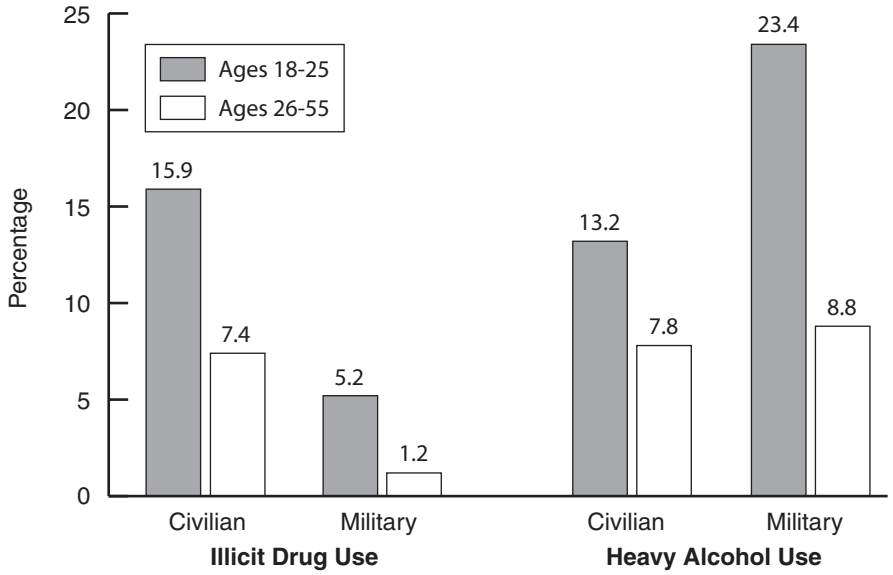

Figure 2
Civilian data have been standardized to the U.S.-based military data by gender, age, education, race/ethnicity, and marital status.

likely than civilians (about 11 percent) to have used any illicit drugs during the past 30 days, but they were significantly *more* likely to have been heavy drinkers (14 percent vs. 10 percent). For illicit drug use, the findings held across both younger (18 to 25) and older (26 to 55) age groups. For alcohol, heavy use was nearly twice as high among younger military personnel compared to younger civilians, but it was about the same among the older age groups. These findings are illustrated in Figure 2. A related analysis using data from the 1985 worldwide survey and civilian data from the 1985 NHSDA showed the same pattern of results (Bray et al., 1991). In the latter study, however, the rates of heavy drinking among military personnel were higher than among civilians for both age groups, which suggests that the rate of heavy drinking among older personnel declined between 1985 and 1998.

The findings indicate that substance-use trends in the military do not simply mirror similar changes among civilians. The lower rates of drug use among military personnel than civilians suggest either that military policies and practices deter drug use in the military or that military personnel hold attitudes and values that discourage substance use. Because of the military's stringent policy about no drug use and the urinalysis testing program to enforce it, it seems likely that the difference between military personnel and civilians results from military policies and practices. In contrast, the higher rates of heavy drinking among military personnel suggest that certain aspects of military life may foster heavy drinking and/or that military policies and programs directed toward reducing heavy alcohol use have not been as effective as similar efforts among civilians.

SUMMARY

Overall, these findings indicate that the military has made steady and notable progress in combating illicit drug use, particularly during the 1980s and 1990s. In 1998, illicit drug use was at minimal levels and rates were substantially lower than among civilians. In contrast, the military has made less progress in reducing heavy drinking. In 1998, heavy drinking affected nearly one in six active-duty personnel and was significantly higher than among civilians. Declines in heavy drinking between 1980 and 1998 were largely explained by changes in the demographic composition of the military. The military appears to have developed an effective formula to reduce illicit drug use and now needs to develop a comparable plan to reduce heavy drinking. Such an effort is currently in the initial stages. The DoD has begun a new prevention initiative that will target alcohol abuse as one of its key components.

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ROBERT M. BRAY

MINIMUM DRINKING AGE LAWS

Before the twentieth century, there were few legal restrictions on the consumption of alcoholic beverages by youth. Early in the twentieth century, laws prohibiting alcohol sales to minors began to be implemented, as part of a broader trend of increasing legal controls on adolescent behavior. The temperance movement succeeded in establishing national Prohibition in 1919 but when it was repealed in 1933, all states implemented legal minimum ages for alcohol purchase or consumption, with most states setting the age at 21.

From the 1930s through the 1960s, the issue received little public attention. In 1970, the 26th Amendment to the U.S. Constitution lowered the voting age in federal elections from 21 to 18. By 1974, all fifty states had lowered their voting ages for state elections to 18. As part of this trend of lowering the "age of majority," twenty-nine states lowered their minimum drinking ages between 1970 and 1975, most setting the age at 18 or 19. In the mid-1970s, studies began to emerge that showed significant increases in the rate of young drivers' involvement in traffic accidents following

the reductions in the legal drinking age. The trend toward lower drinking ages was reversed, with Maine being the first state to raise its legal drinking age from 18 to 20 in October 1977. Several other states soon followed, and research studies completed by the early 1980s found significant declines in youth traffic-crash involvement when states raised their legal drinking age. With the support of organized efforts by citizen-action groups such as REMOVE INTOXICATED DRIVERS and MOTHERS AGAINST DRUNK DRIVING, federal legislation was passed in 1984 that called for the withholding of a portion of federal highway-construction funds from any state that did not have a legal drinking age of 21 by October 1986. As a result, all the remaining states with a legal drinking age of below 21 raised their age to 21 by 1988. Thus, all states now have a uniform legal drinking age of 21, although details in regard to the purchase, possession, consumption, sales, and furnishing of alcohol to underage youth vary from state to state.

The legal drinking age became a major issue because of the serious consequences of young people's consumption of alcohol. Most teenagers drink; in addition, almost a third regularly become intoxicated. Damage resulting from the drinking of youth is extensive. Car crashes are the leading cause of death for teenagers (Baker et al., 1992), and one third to one half of the crashes involve alcohol (National Highway Traffic Safety Administration, 1990). Other leading causes of disability and death among youth, such as suicide, homicide, assault, drowning, and recreational injury, involve alcohol in one quarter to three quarters of the cases (Wagenaar, 1992). Injuries are only part of the problem. Early use of alcohol appears to affect multiple dimensions of physical, social, and cognitive development (Semlitz & Gold, 1986). Alcohol use increases the odds of having unprotected sex (i.e., failure to use a condom), which increases the chance of pregnancy and catching sexually transmitted diseases, including the human immunodeficiency virus (HIV), which causes AIDS (Plant, 1990; Strunin & Hingson, 1992). Many "date rape" situations involve individuals who have been drinking (Wagenaar et al., 1993a). Early use of alcohol increases the odds one will move on to using other drugs, such as MARIJUANA, COCAINE, or HEROIN (Kandel, 1989). Finally, the earlier one starts a pattern of regular drinking, the higher the chance of later serious problems with alcohol, including dependence (i.e., getting "hooked" so that it is very hard to quit). Despite the many problems associated with young people's drinking, the most obvious one, and the one that received the most attention in debates on the legal drinking age, is traffic-crash involvement.

EFFECTS OF THE DRINKING AGE ON CAR CRASHES

Seventeen studies of the effects of lowering the legal age for drinking on traffic crashes appeared between 1974 and 1982 (Wagenaar, 1983). Although results varied across studies and across states, most studies found significant increases in traffic crashes among youth after the drinking age had been lowered (usually from 21 to 18). Typically, lowering the drinking age resulted in 5 percent to 20 percent increases in fatal and injury-producing crashes likely to involve alcohol, such as single-vehicle crashes occurring at night.

Thirty-nine studies of the effects on traffic crashes of raising the legal age for drinking have appeared between 1979 and 1992 (Wagenaar, 1993). Twenty-eight of these studies found significant reductions in the involvement of youth in traffic crashes following increases in the legal drinking age. Typically, raising the drinking age resulted in 5 percent to 20 percent declines in fatal and injury-producing crashes likely to involve alcohol. With the aid of the better-designed studies with longer follow-up periods, it could be estimated that the long-term effects of raising the drinking age to 21 would be a 13 percent decline in single-vehicle nighttime crashes among those whose legal access to alcohol was removed (i.e., 18 to 20-year-olds).

The legal drinking age is probably the most extensively researched policy that is designed to reduce traffic crashes and other alcohol problems. Scientists and professionals in the field agree that lowering the legal age for drinking increased car crashes among youth, and that, subsequently, raising the legal age reversed the effect: It lowered car crashes among youth (United States General Accounting Office, 1987). The National Highway Traffic Safety Administration estimates that, even when counting only those states that raised the legal age after 1982, the U.S. age-21 policy now saves over one thousand lives per year in reduced car crashes alone (Arnold, 1985).

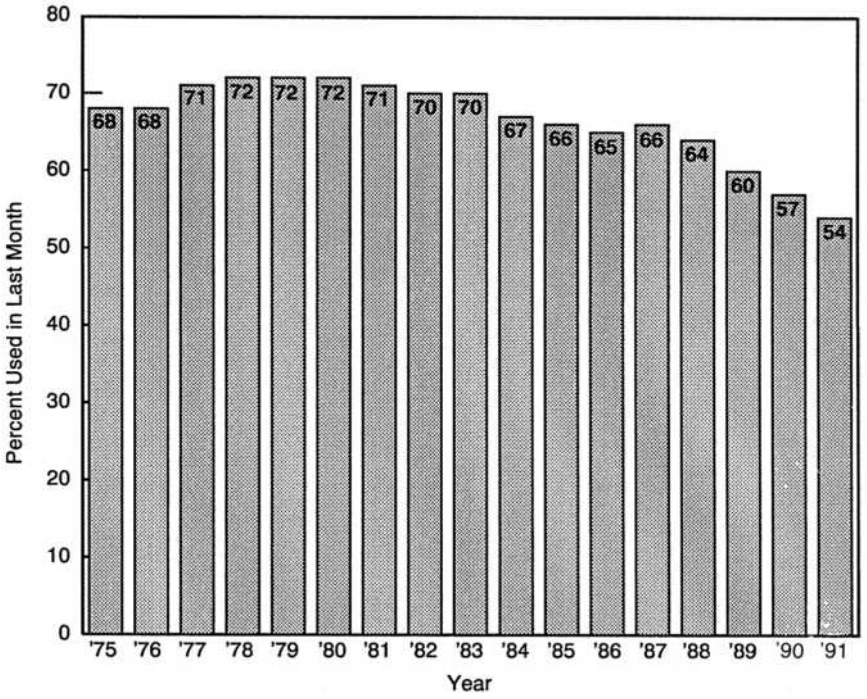

Figure 1
Alcohol Use, U.S. High School Seniors

EFFECTS OF THE DRINKING AGE ON OTHER PROBLEMS

Four studies have appeared on the effects on problems other than car crashes of raising the legal age to 21 (Wagenaar, 1993). One study found that vandalism was down 16 percent in four states that raised the drinking age, and another found that significant reductions in suicides, pedestrian injuries, and other unintentional injuries were associated with higher legal drinking ages. A study of two Australian states that lowered the legal drinking age found 22 percent to 40 percent increases in trauma-hospital admissions for causes other than car crashes, although another study did not confirm these findings. A Massachusetts study found no reductions in nontraffic trauma, suicide, and homicide deaths after the drinking age had been raised, perhaps because many of Massachusetts' residents lived close to bordering states that had lower drinking ages at the time of the study.

EFFECTS OF THE DRINKING AGE ON ALCOHOL USE

Seven studies examined the effect of the legal drinking age on aggregate alcoholic-beverage sales. Effects were mixed—some studies found that alcohol sales were related to the legal age, but others did not find such a relationship. These studies were difficult to interpret because alcohol sales to young drinkers could not be distinguished from sales to older drinkers.

Surveys of the effects on alcohol use among youth of lowering or raising the drinking age have produced conflicting results. Some have found that there was little effect of the legal drinking age on young people's drinking, whereas others have found that lower rates of youth drinking resulted when the legal drinking age was higher (see Wagenaar, 1993, for a review of the fourteen survey studies to date). A major limitation of many of these studies was their use of nonrandom samples of youth from particular high schools, colleges, and local communities rather than samples that were broadly representative of the youth in a state. Surveys of college students, which are usually limited to students in introductory social sciences courses, frequently report finding little effect of the legal drinking age on drinking patterns. In contrast, surveys of random samples of high school seniors and 18- to 20-year-olds across many states, including those entering college and those in the work force, report finding significant reductions in drinking that are associated with higher legal drinking ages

(Maisto & Rachal, 1980; O'Malley & Wagenaar, 1991). It appears, on the basis of the best-designed studies, that raising the legal drinking age results in reductions in young people's drinking. The age-21 policy, however, by no means eliminates this drinking by youth.

ENFORCEMENT OF THE MINIMUM DRINKING AGE

Although drinking among youth is now significantly down from its peak in 1980, when questioned, 54 percent of high school seniors still reported drinking in the last month, and 30 percent reported having had five or more drinks at a time at least once in the previous two weeks (Figures 1 and 2; data from Johnston, O'Malley, & Bachman, 1991). Among the many reasons that youth continue to drink, one important reason is that alcohol remains easily available to them, despite the minimum drinking age law. A recent study by Wagenaar and associates (1993b) indicated that only two of every one thousand episodes of underage drinking resulted in an arrest of the youth involved. More important, only five of every hundred thousand episodes of drinking by underage youth resulted in any action being taken against a store, restaurant, or bar for selling or serving alcohol to a minor. Because the chance of getting caught was so low, half or more of all alcohol outlets tested sold alcohol to youth without requesting any age identification (Preusser & Williams, 1991; Forster et al., 1993).

CONCLUSIONS

Evidence that showed that raising the drinking age to 21 reduced deaths and injuries in car crashes was a major factor in the debate about the drinking age. Other arguments were also heard, such as: Is it unconstitutional to discriminate solely on the basis of age? Federal courts have ruled that the drinking age is not discriminatory, because (1) drinking is not a fundamental right, (2) age is not an inherently suspect criterion for discrimination, (3) and the higher drinking age has a "rational basis" and is "reasonably related" to a legitimate goal of the state to reduce death and injury from traffic crashes (Guy, 1978). In a democracy, laws should have the support of the governed. Repeated polls have shown that the majority of the public clearly supports a legal drinking age of 21. Even among youth under the age of 21, some polls have shown majority support for the minimum drinking age of 21.

Is it logical to set the legal age of drinking at 21, when other rights and privileges of adulthood (e.g.,

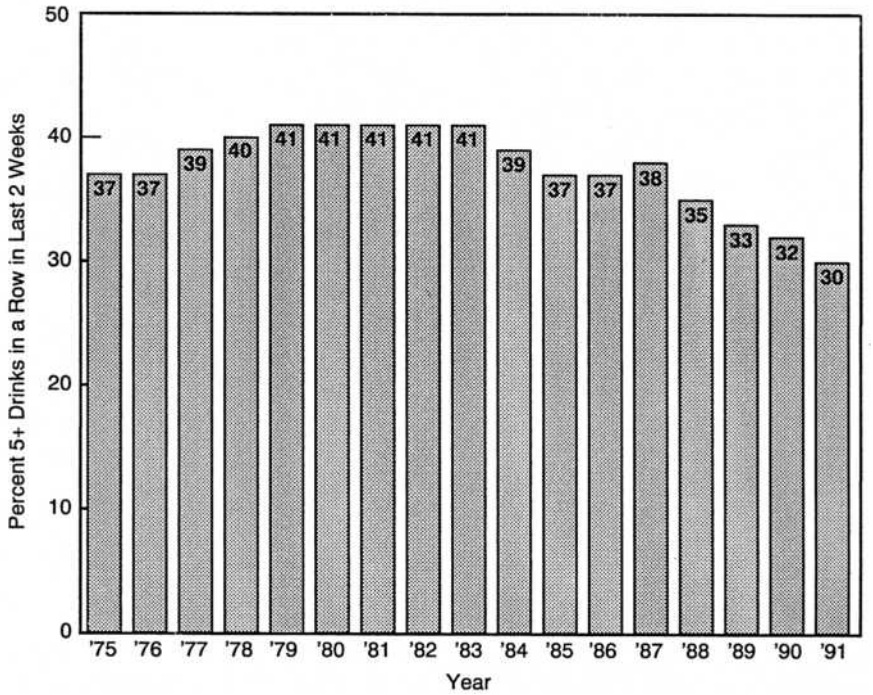

Figure 2
Binge Drinking, U.S. High School Seniors

voting, signing legally binding contracts) begin at age 18? The answer is yes, because we have many different legal ages, varying from 12 to 21, for voting, driving, sale and use of tobacco, legal consent for sexual intercourse, marriage, access to contraception without parental consent, compulsory school attendance, and so forth. Minimum ages are not set uniformly; they depend on the specific behavior involved, and they are arrived at by balancing the dangers and benefits of establishing the particular age.

Some have argued that a minimum drinking age of 21 will make things worse when young people finally get legal access to alcohol. This is the "rubber band" theory whereby it is claimed that prohibiting teenagers from drinking will cause a pent-up demand for the forbidden fruit. At 21, they will break loose and drink at significantly higher rates than they would have if they had been introduced to alcohol earlier. This theory is clearly not supported by research. For example, O'Malley and Wagenaar (1991) found just the opposite results in their nationwide study—that is, persons aged 21 to 24 drank at lower rates if they had to wait until 21 to have legal access to alcohol. A frequently heard related argument is that a minimum drinking age of 21 may reduce car crashes among teenagers, but this will only be a temporary effect if it simply delays those problems until the teenagers reach age 21. This argument is also false. The minimum age of 21 significantly, reduces car crashes among 18- to 20-year-olds, and those injuries and deaths are permanently saved. There is, furthermore, no rebound effect at age 21; in fact, the higher legal age appears to produce benefits, in terms of reduced drinking, that continue into a person's early twenties.

The debate surrounding the legal age for drinking appears settled in the United States. However, other countries (particularly in Europe where drinking ages are typically set at 18) are now examining the research and experience of the United States with increasing interest. Professionals in the areas of public health and traffic safety, as well as other professionals and citizens, are beginning to see the benefits of the age-21 drinking law in the United States, and they are initiating in their own countries the debate on the most appropriate age for legal access to alcohol.

(SEE ALSO: *Accidents and Injuries from Alcohol; Driving, Alcohol, and Drugs; Driving Under the Influence; Social Costs of Alcohol and Drug Abuse*)

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MINNESOTA MULTIPHASIC PERSONALITY INVENTORY (MMPI) This is a self-report test containing 550 statements that can be answered true or false (Levitt & Durkworth, 1984). It was first published in 1943 for use in routine diagnostic assessment. As one of the most widely used psychological tests, the MMPI is sometimes given to alcoholics and drug users to evaluate the psychological effects of substance use as well as the personality characteristics of substance abusers.

The MMPI is scored in subunits or scales. Eight scales comprise the main parts of the clinical profile, which is a standard way of describing the patient's personality features in relation to population norms. The clinical scales measure hypochondriasis, depression, hysteria, psychopathic deviancy, paranoia, psychasthemia, schizophrenia, and hypomania.

The MMPI has three main applications to the diagnosis and study of substance-use disorders. First, it has been used to evaluate the effects of alcohol and drug abuse. Several studies (Pettinati et al., 1982; Babor et al., 1988) have found that MMPI clinical scales measuring depression, paranoia, and other psychiatric symptomatology tend to be higher than normal when alcoholics are drinking—but return to the normal range during periods of abstinence. Second, the MMPI has been used to identify subtypes of alcoholics and drug users that might benefit from specialized treatments. For example, several studies have found three types of alcoholics based on their MMPI profiles: neurotic, psychotic, and psychopathic (Conley, 1981; Nerviano & Gross, 1983). Third, the MMPI has been used in the development of screening tests. The MacAndrew scale (MacAndrew, 1965), for example, is used to measure impulsivity, pressure for action, and acting-out potential that may lead to alcoholism and drug abuse. Persons who score high on the MacAndrew scale are therefore considered to be at risk for substance-use disorders.

(SEE ALSO: *Addiction Severity Index; Diagnostic and Statistical Manual; Disease Concept of Alcoholism and Drug Abuse; Michigan Alcoholism Screening Test*)

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THOMAS F. BABOR

MINORITIES AND DRUG USE *See* Ethnicity and Drugs

MMPI *See* Minnesota Multiphasic Personality Inventory

MONEY LAUNDERING Obtaining the proceeds of crime has generally been but the first step for profit-motivated criminals. The use of those often has required a second step, whether it be to convert the money into form usable form for licit or illicit purposes, disguise its origins, avoid tax consequences, or make it possible to transport. As the quantity of money to be derived from illegal activity increases, the "laundering" of that money becomes more necessary with the internationalization of commerce, parallel markets, and increased technology. Money laundering has become more sophisticated as a consequence.

The International Financial Action Task Force, convened in 1989 by the G-8 Economic Summit, defines money laundering as "the process by which one conceals the existence, illegal source, or illegal use of the crime proceeds to make those proceeds appear legitimately derived." There are three steps to laundering funds: introducing the proceeds of criminal activity into the legitimate economy (commonly referred to as "placement"), engaging in financial transactions designed to limit the ability to trace the funds (commonly referred to as "layering"); and making the funds available for use (commonly referred to as "integration").

In fact, depending on the objectives of individual criminals as far as convenience and security are concerned, the laundering process can be effected with as few as one and as many as a dozen discrete steps. In its most familiar form, hundreds of thousands of dollars in drug proceeds are taken to a financial institution and exchanged for a cashier's check, which the trafficker can carry around (or out of the country) with much less suspicion than suitcases full of cash. A slightly more involved scenario entails taking the same cash to the same bank, where it is deposited into an account and then sent by wire transfer to a bank in a foreign country, probably a jurisdiction renowned for the relative secrecy it affords customers like the hypothetical drug dealer.

In even more elaborate schemes, the same funds are wire transferred around a circuit of accounts in different countries, bearing the names of legitimate businesses. After the transfer reaches its final destination abroad, the owner in the United States arranges a sham transaction to bring the funds back into this country, often as the proceeds of a purported loan. There are literally countless varieties of laundering schemes, limited only by the imaginations of criminals and a more widespread impatience with transferring one's funds too far away.

Traditionally money laundering was conducted by the same individuals who committed the underlying criminal activity. Today, the sophistication of the process has given rise to the professional money launderer. But as money laundering has become more invaluable for criminals and criminal networks, governments have increasingly come to see the process as a potential vulnerability in the business of crime and have increasingly sought to curtail and prosecute it.

The United States began its legislative efforts to crackdown on money laundering in 1970 by requiring the reporting of cash transactions as part of the Bank Secrecy Act. As now modified, $10,000 in cash deposited in a financial institution or paid to a business will trigger the reporting requirements by the recipient of the funds. And with the Money Laundering Control Act of 1986, codified as 18 USC 1956 and 1957, Congress made it a crime to move certain illegally obtained funds through the commercial or banking system. Enforcement of anti-money laundering legislation was not only accomplished through the traditional penalties of incarceration and fines, but enhanced with powerful forfeiture remedies. Finally, since 1988, federal legislation has required banks to report "suspicious transactions." Individual states have sought to control money laundering with their own statutory and regulatory schemes. Internationally, the Financial Action Task Force and Interpol have approved resolutions, protocols, and recommendations calling for nations to pass legislation that would make money laundering a crime, require reporting of

suspicious transactions, permit forfeiture, and allow extradition in money laundering cases.

U.S. anti laundering legislation is complex and often controversial but, what is perhaps most remarkable is the fundamental change in enforcement policy it represents, wrought by the requirement that non-law enforcement entities be compelled to engage in the systematic reporting of potential illegal activity. As a result, compliance programs requiring the recipient of funds to know its customer's business and to establish baselines from which suspicious activities can be identified are now the norm. For better or for worse, money laundering has brought the private world of commerce into the public field of law enforcement.

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MONITORING THE FUTURE *See* High School Senior Survey

MONOAMINE A monoamine is an amine that has one organic substituent attached to the nitrogen atom (as RNH_2). SEROTONIN is such an amine, one that is functionally important in NEUROTRANSMISSION. Chemically, monoamines include the catecholamines (derived from tyrosine) and the indoleamines serotonin and melatonin (derived from the amino acid tryptophan). Acetylcholine also has only a single (but trimethylated) amine, while histamine (a diamine formed from histidine) stretches the condition only slightly. Neurotransmitters in this class share several properties—nanomolar concentrations/milligram protein; neurons (nerve cells) that contain thin, generally unmyelinated axons to many brain regions; and their receptors (except for the cholinergic nicotinic receptor and one of the ten or so subtypes of serotonin receptors) employ second-messenger coupled transduction. Monoamine neurotransmitters are often involved in the action of mind-altering drugs and have been well studied.

(SEE ALSO: *Dopamine*, *Neurotransmitters*)

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FLOYD BLOOM

MOOD AND DRUGS *See* Research: Measuring Effects of Drugs on Mood

MOONSHINE Moonshine (white lightning) is the colloquial term for illegally produced hard liquor—whiskey, rum, brandy, gin, and vodka. The term probably originated around 1785, when it was recorded in a British book on vulgar language—used to describe the white (clear) brandy that was smuggled to the coasts of Kent and Sussex in England. In the New World, moonshine was made in homemade stills, usually from corn, especially in rural areas in the southern United States—before, during, and after Prohibition—and continues to be made today. The ethanol (drinking ALCOHOL) content is usually high, often approaching 80 percent (160 proof). First-run moonshine contains a number of impurities, some of which are toxic, so it is necessary to double and triple distill the liquor to purify it for drinking.

(SEE ALSO: *Alcohol: History of Drinking; Legal Regulation of Drugs and Alcohol; Still*)

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MORNING GLORY SEEDS The seeds of the morning glory, genus *Ipomoea* of the family Convolvulaceae, contain many lysergic acid derivatives, particularly lysergic acid amide. The hallucinogenic properties of some of these derivatives are not known. The seeds can be ingested whole; they can be ground and used to prepare a tea; or the active compound can be extracted using solvents. The seeds have also been used as a source of precursors for the synthesis of LYSERGIC ACID DIETHYLAMIDE (LSD). Since the seeds contain

Figure 1
Morning Glory

lysergic acid derivatives, people ingesting morning glory seeds may feel "different"; however, the experience is not identical to an LSD-type "trip," even though the seeds are marketed on the street as an LSD equivalent.

Although morning glory seeds are easy to purchase legally, many varieties (those sold by reputable garden-supply distributors) have been treated with insecticides, fungicides, and other toxic chemicals—as well as with compounds that will induce vomiting if the seeds are eaten.

(SEE ALSO: *Hallucinogenic Plants; Mescaline*)

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MORPHINE Morphine is a major component of OPIUM, a product of the poppy plant (PAPAVER SOMNIFERUM or *P. album*). Named after Morpheus, the Greek god of sleep, morphine is a potent ANALGESIC (painkiller) that is widely used for moderate to severe PAIN. Morphine is one of approximately twenty ALKALOIDS in opium; it was first purified in 1806 and, by the mid-1800s, pure morphine was becoming widely used in medicine. At approximately the same time, the hypodermic needle and syringe was developed, which permitted the injection of the drug under the skin (subcutaneous, S.C.), into muscles (intramuscular, I.M.), or directly into the veins (intravenous, I.V.). Together, these routes of administration are termed parenteral. Injections provide rapid relief of pain and can be used in patients who are unable to take medications by mouth. These advantages led to the wide use of morphine injections during the American Civil War (1861–1865). At that time, the intense euphoria and addictive potential of these agents following injections was not fully appreciated, leading to the addiction of a large number of soldiers. Indeed, morphine was not illegal and was sold over the counter; ADDICTION soon became known as the Soldier's Disease.

Since that time, a major objective of pharmaceutical companies has been to develop, for medication purposes, a nonaddictive analgesic with the potency of morphine. The concepts of PHYSICAL DEPENDENCE and addiction were not clearly differentiated until the late twentieth century, and it is likely that most of those early addicts were attempting to prevent the onset of WITHDRAWAL symptoms. Today very few patients become addicted to opiates, despite the fact that with continued administration all will become physically dependent—this may reflect our better understanding of the drugs plus our ability to take a patient off medications without precipitating withdrawal symptoms.

Morphine produces a wide variety of actions, some desired and others not. The definition of a desired action and a side effect depends on the reason for using the drug. For example, opiates such as morphine can be used to treat diarrhea—but their constipating actions are usually considered an undesirable side effect when they are used to treat pain.

Clearly, the control of pain remains the most important use for morphine. Morphine and other OPIATES relieve pain without interfering with traditional sensations. Patients treated with morphine often report that the pain is still there but that it no longer hurts. Morphine works through *mu* opiate RECEPTORS located both within the brain and the spinal cord. Morphine has a number of other actions as well. Its ability to constrict the pupil is one of the most widely recognized signs of opiate use. In addition, morphine produces sedation and, at higher doses, morphine will depress respiration.

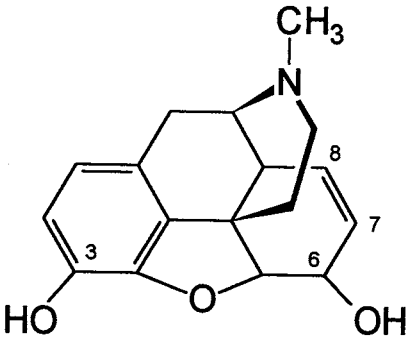

Figure 1
Morphine

Very high doses of morphine will stop breathing entirely—a common occurrence in overdoses.

Morphine also has a major influence upon the gastrointestinal tract, which is the basis for its antidiarrheal effect. Here, morphine decreases the motility of the stomach and intestine, through local actions on the organs themselves, as well as through control systems located within the brain and spinal cord. Other systems can be affected as well. Morphine produces a vasodilation, in which the peripheral blood vessels are relaxed. This can lead to significant drops in blood pressure when a person shifts from a lying to a standing position as the blood is pooled in the legs. This ability to pool blood in relaxed blood vessels can be used clinically to treat conditions such as acute pulmonary edema, an accumulation of fluid within the lungs, which occurs in acute myocardial infarctions (heart attacks). Increasing the capacity of the vascular system by relaxing the blood vessels permits the reabsorption of the lung fluid. Finally, morphine and similar drugs, such as CODEINE, are also effective agents in the control of coughing.

All these effects of morphine can be easily reversed by ANTAGONISTS. NALOXONE is the most widely used antagonist. Given alone, it has virtually no actions; however, low doses of naloxone are able to block or reverse all the actions of morphine described above.

Morphine is given either by mouth or by injection. Oral administration is associated with a significant metabolism of the drug by the liver, explaining its lower potency as compared to that attained by injections. From three to six times more morphine must be taken by mouth to produce the same effects as an injected dose. Thus, higher doses are needed when giving the drug orally. Morphine injections can be given either intramuscularly, subcutaneously, or intravenously. Continuous infusions are also becoming more common, but their use is restricted to physicians expert in the treatment of pain. Morphine has a relatively short half-life in the body, around two hours, and it is usually given to patients every four to six hours. It is extensively metabolized. In the late 1980s, it was discovered that one of these metabolites, morphine-6β-glucuronide, is very potent, far more potent than morphine itself. The importance of this compound with a single morphine dose is probably not great; however, with chronic dosing, the levels of morphine-6β-glucuronide in the blood actually exceed those of morphine—so this metabolite may be responsible for most of morphine's actions. Since this metabolite is removed from the body by the kidneys, special care must be taken when giving morphine to patients with kidney problems.

One common problem associated with morphine is nausea. This is difficult to understand, since nausea does not occur in all patients and often is seen with one drug but not others. This lack of consistency raises questions about whether it is a specific receptor-mediated action or whether it may be a nonspecific side effect.

With chronic use, morphine has a progressively smaller effect, a phenomenon termed TOLERANCE. To maintain a constant action it is necessary to increase the dose. Along with tolerance, morphine also produces physical dependence. Physical dependence (physiological dependence; neuroadaptation) develops as the body attempts to compensate for many of morphine's actions. As long as a person continues to receive the drug, no symptoms are noted. Abrupt cessation of the drug or the administration of an antagonist, such as naloxone, produces a constellation of symptoms and signs termed the withdrawal syndrome. Early symptoms include a restlessness, tearing from the eyes and a runny nose, yawning, and sweating. As the syndrome progresses, one sees dilated pupils, sneezing, elevations in heart rate and blood pressure, and gooseflesh—which is responsible for the term "cold turkey." Cramping and abdominal pains are also common.

Physical dependence (or neuroadaptation) is a physiological response to repeated dosing with morphine and is seen in virtually all patients. Physical dependence, however, is not the same as addiction (drug dependence). Drug dependence (addic-

tion) is defined as drug-seeking behavior, whereas physical dependence is simply a physiological response to the medication. While addiction is common among drug abusers, it is rare when morphine is used for appropriate medical conditions. The reasons for this difference are not clear, and they remain a major issue in understanding and treating morphine addiction.

(SEE ALSO: *Addiction: Concepts and Definitions; Diagnostic and Statistical Manual; Opiates/Opioids; Opioid Complications and Withdrawal*)

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GAVRIL W. PASTERNAK

MOTHERS AGAINST DRUNK DRIVING (MADD) This organization works to reduce DRUNK DRIVING and to help the victims of drunk-driving ACCIDENTS. Many of MADD's members are volunteers who have personally suffered from the results of drunk driving. This national organization was founded by Candy Lightner, whose thirteen-year-old daughter, Cari, was killed by a drunk driver on May 3, 1980. Ms. Lightner was outraged to learn that only two days previously the driver had been released from jail, where he had been held for another hit-and-run drunk-driving crash. Although he had been arrested for drunk driving several times before, he was still driving with a valid California license. Candy Lightner decided to begin a campaign to keep drunk drivers off the road, so that other mothers would not have to suffer the anguish that she was experiencing. On September 5, 1980 (Cari's birthday), MADD was incorporated.

Since then, MADD has evolved into an organization with millions of members and hundreds of local chapters across the United States. Chapters have also been started in Canada, Great Britain, New Zealand, and Australia. Membership is not restricted to mothers of victims or to the victims themselves. Everyone who is concerned about the drunk-driving issue is welcome to join. Funding for

Candy Lightner, the founder of Mothers Against Drunk Driving, holds a photograph of her daughter, Cari, who was killed by a drunk driver on May 3, 1980. (AP Photo)

the organization comes from membership dues and contributions; MADD also applies for and receives grants from federal and state governments and private organizations. Paid staff are employed to provide leadership on the state and national levels. MADD is involved in three major kinds of activity: (1) advocacy for stricter drunk-driving laws and better enforcement, (2) promotion of public awareness and educational programs, and (3) assistance to victims.

THE LEGISLATIVE AGENDA

According to MADD, drunk driving is a violent crime. One of its rallying slogans is, "Murder by Car Is Still Murder!" Over the years, MADD members have worked to generate public support for passage of stricter drunk-driving legislation, punitive sanctions, and more consistent enforcement measures aimed at deterring drunk driving. In the 1980s, intense lobbying efforts were undertaken for the passage of laws making twenty-one the minimum legal age for drinking (now in force in all 50 states). The group believes that this measure has saved thousands of young lives that would have been lost in drunk-driving crashes.

MADD has also lobbied for changes in judicial procedures that would make the system more responsive to victims of drunk driving. For example, in many states victims had been barred from the courtroom during the trial of their own drunk-driv-

ing cases, because their testimony (or even their presence) might prejudice the jury. Owing to the efforts of MADD and other groups, victims' rights bills have now been passed in all states. These ensure that victims will be notified about court hearings and, in most states, allowed to testify about the impact of the crime on their lives. Other lobbying efforts have sought to close legal loopholes that drunk drivers were using to avoid punishment. For example, drivers might have refused to take a breath or blood test for intoxication and have been allowed to plead guilty to a lesser charge. In other cases, drivers were allowed to claim that despite their high blood-alcohol content (BAC), their driving was not really impaired.

MADD has been instrumental in the passage of over 1,000 tougher drunk-driving laws that close these loopholes and institute other deterrence measures, such as mandatory jail sentences for drunk drivers. MADD also supports efforts to require offenders to undergo treatment for alcoholism and/or drug dependency, if this is deemed necessary.

PUBLIC AWARENESS AND EDUCATION

MADD is involved in various efforts to raise public awareness and concern about drunk driving. The "National Candlelight Vigil of Remembrance and Hope" is held in many locations each December, drawing victims together to give public testimony to the suffering that results from drunk driving. During the "Red Ribbon Tie One On for Safety" campaign, which takes place between Thanksgiving and New Year's Day, MADD encourages citizens to attach a red ribbon to their car as a reminder to themselves and others to drive sober. MADD's well-known public awareness campaign of the past used the slogan, "Think . . . Don't Drink and Drive" in public-service announcements on radio and television and in print materials. A more recent campaign, "Keep It a Safe Summer" (KISS) emphasized the need for sobriety during recreational activities that involve driving, boating, or other risky activities. MADD also provides curriculum materials for schools and each year sponsors a poster and essay contest for children on the subject of drunk driving.

ASSISTANCE TO VICTIMS

Programs that provide aid to victims of drunk-driving crashes constitute the heart of MADD's mission. Support groups help victims share their pain with others who understand their feelings. MADD members send "We Care" cards to victims of recent crashes. Specially trained victim advocates offer a one-on-one personal relationship with victims, trying to respond to both their emotional and practical needs. Victims are briefed on their legal rights and on the judicial procedures relevant to their cases. They can call a toll-free number (1-800-GET MADD) for information and for help in case of crisis. MADD also offers death-notification training for police and specialized training for other community professionals, such as clergy and medical workers, who are called upon to assist victims.

"20 × 2000"

Since the founding of MADD in 1980, the percentage of alcohol-related traffic fatalities has steadily decreased from almost 60 percent to around 50 percent. MADD's goal "20 × 2000" seeks to reduce that proportion by an additional 20 percent by the year 2000. Intensified efforts will focus on more effective law enforcement, increased sanctions, and prevention programs that include education for youth and more responsible marketing and service practices in liquor establishments.

(SEE ALSO: *Blood Alcohol Concentration, Measures of; Blood Alcohol Content; Breathalyzer; Dramshop Laws; Driving, Alcohol, and Drugs; Driving Under the Influence; Legal Regulation of Drugs and Alcohol; Minimum Drinking Age Laws; Psychomotor Effects of Alcohol and Drugs; Remove Intoxicated Drivers; Students Against Destructive Decisions*)

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DIANNE SHUNTICH

MPTP To circumvent the laws regarding controlled drugs, a chemist attempted to synthesize a derivative of MEPERIDINE. By synthesizing a new derivative not specifically covered by the CONTROLLED SUBSTANCES ACT and existing Drug Enforcement Agency laws and by synthesizing the drug and selling it within the same state, the chemist had hoped to profit but to avoid violation of the laws. This DESIGNER DRUG approach was being widely used to avoid prosecution for selling drugs of abuse—however, in this case a side product was also formed in this reaction, MPTP (1-methyl-4-phenyl-1,2.3,6-tetrahydropyridine). People who bought this mixture on the street quickly developed a neurological syndrome virtually indistinguishable from Parkinson's disease. Initially, the cause of this problem remained unknown. With intense investigation, the blame was placed on the side product in the reaction, MPTP. MPTP had long been used as an intermediate in chemical synthesis and was commercially available. The ability of MPTP to provoke a Parkinson-like syndrome helped explain a report from years ago of a chemist working with this compound suddenly developing a Parkinson-like disease.

The Parkinson-like syndrome is very similar to the symptoms originally described in Parkinson's disease. The most notable aspects of the syndrome are the marked cog-wheel rigidity of the muscles, along with a generalized decrease in movement usually associated with problems initiating the movement. Patients often have difficulty with fine motor skills, such as writing, and with walking, which usually becomes a series of small, shuffling steps termed a "festinating gait"; their greatest problem is starting and stopping. Diminished blinking coupled with a limited facial expression can be very prominent and is termed "masked facies." In Parkinson's disease, patients also have a pill-rolling tremor and a tendency to fall, because

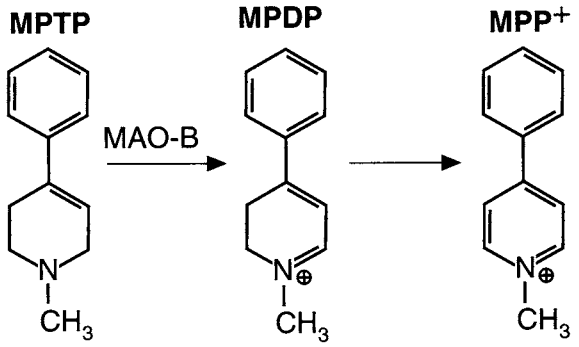

Figure 1
MPTP Conversion to MPDP and MPP+

of problems with blood pressure and the reflexes important to maintaining posture.

Pathologically, Parkinson's disease is noted for a degeneration of pigmented nuclei within the brain, including the substantia nigra. The loss of the dopaminergic NEURONS in the substantia nigra that project to the part of the brain called the striatum is responsible for the motor problems, while the degeneration of other areas of the brain, including the locus coeruleus, are presumably responsible for the autonomic problems. The cause of Parkinson's disease is still not known; treatment is symptomatic. Early studies demonstrated the ability of anticholinergic medications to help with many of the motor symptoms, especially the tremor. However, the drug of choice in the 1990s is L-dopa, a precursor of DOPAMINE. Unlike dopamine, which does not traverse the blood-brain barrier, L-dopa is readily transported into the brain where it is taken up into neurons and converted to dopamine—thereby helping to reduce symptoms caused by loss of dopamine-containing neurons. Replacement of the dopamine can markedly limit the severity of the motor symptoms; however, the duration of this benefit is often limited to only about five years, presumably due to the continued progression of the disease.

MPTP does not bind to OPIOID RECEPTORS and it has no opioid activity, although it is a side product in the synthesis of a meperidine analog. When ingested, it is taken up into neurons containing a catecholamine transporter, greatly limiting the neurons affected. Once in the cell, the drug is converted by the enzyme monoamine oxidase (type B) in a series of steps to another compound, MPP +, which is believed to be responsible for its toxic

actions. The need for the transporter to take up the toxin into the cells partially explains its selective toxicity within the brain. There, this drug destroys the same groups of pigmented catecholinergic neurons affected in Parkinson's disease, including the substantia nigra and the locus coeruleus. The greater sensitivity of pigmented neurons to the toxin is still not completely understood. One hypothesis has been put forward: The color in the neurons is due to the pigment melanin, which actively binds the toxin. Therefore, it has been suggested that this binding results in the accumulation of very high levels of the drug, which persist in the neurons for long periods of time, enhancing its toxicity.

Clinically, MPTP produces a syndrome virtually identical to that seen in Parkinson's disease, but Parkinson's is a progressive degenerative disease, which, over the period of many years, gradually leads to a variety of difficulties with thought and memory. It is not thought that MPTP produces a similar global, diffuse loss of function. The marked similarity, though, has led to the speculation that Parkinson's may be due to the exposure to a toxin similar to MPTP. Since the toxicity of MPTP depends on its conversion by type B monoamine oxidase (MAO-B), it has been suggested that inhibition of this enzyme may prove beneficial. Seligine is a selective MAO-B inhibitor, and early clinical trials suggest that the progression of Parkinson patients taking this medication may be slower than in the control groups.

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GAVRIL W. PASTERNAK

MULES *See* Slang and Jargon

MULTIDOCTORING Multidoctoring, or double-doctoring, is the practice of obtaining medications from more than one physician without informing the other physician(s) involved of any medication already prescribed. Almost always, the medications involved are PSYCHOACTIVE medications, which may then be abused or misused. Individuals who engage in this behavior may be obtaining the medication for their own use or for the purpose of diverting it to sell on the street. People who seek drugs for the purpose of selling them on the street are often very convincing in their appeals and can get the physician to prescribe the particular drug they are after without even mentioning it by name. In Canada and the United States, legislation prohibits people from acquiring a narcotic prescription without informing the physician of other narcotics that have already been for them prescribed that month. Failure to do so results in criminal charges. Physicians can record a patient's response to the question about other prescribed narcotics, and psychoactive drugs in general, as a means of discouraging multidoctoring.

Physicians themselves may be involved at various levels in multidoctoring and the diversion of drugs to the street. These are the physicians termed "script doctors," who willfully prescribe controlled substances to people seeking them, or who prescribe them as a result of being misled or simply uniformed about the prevalence of multidoctoring and the substances involved. Educating the public regarding the risks of prescription-medication abuse and increasing the skills of physicians in recognizing patients engaged in multidoctoring will help to decrease the diversion and misuse of prescription drugs.

(SEE ALSO: *Controls: Scheduled Drugs; Iatrogenic Addiction*)

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KAREN PARKER

MYTHS ABOUT ADDICTION AND ITS TREATMENT As of the year 2000, many medical facts about the causes of addiction, its nature, the best ways to treat its symptoms, and the possibility of devising a full, permanent cure remain unknown. However, research has already established that the myths listed below are false. Unfortunately, these common myths cause the general public and even many physicians to be needlessly unsympathetic to addicts.

Myth: Addiction is an acute condition, like a broken leg or pneumonia.

Fact: Addiction is a chronic disorder, like arthritis, high blood pressure, asthma, or diabetes.

Myth: Addiction ends when detoxification removes all of the abused substance from the addict's body.

Fact: Changes in the pathways of the brain, which had been caused by the abused substance, persist long after the last particle of the abused substance has left the body.

Myth: Addiction ends when the pain following detoxification (the withdrawal syndrome) is gone.

Fact: At the end of the withdrawal process, pain caused by the body's dependence on the abused substance stops, but the underlying addictive disorder (the cause [or set of causes] which made the person liable to become addicted in the first place) remain.

Myth: When a patient relapses (returns to addiction) after detoxification, then the detoxification of this patient must have failed as a treatment.

Fact: As a chronic disorder, addiction needs ongoing treatment, not just a one-time detoxification. One does not expect a single injection of insulin to cure a diabetic, or any single administration of medicine to relieve a patient forever of arthritis, asthma, or high blood pressure. Each treatment is successful if it improves the condition at the time; each needs to be repeated, often throughout the rest of the patient's life.

Myth: Once an addict is detoxified, as long as he or she does not take the abused substance (or a different abused substance) again, any medical, social, and occupational difficulties that had been associated with the addiction disappear.

Fact: Medical, social, and occupational consequences may last long after an addict has stopped taking any abused substance. Let us assume, for example, that because of alcoholism a person has lost an eye while driving drunk, has been divorced for cruelty and non-support, and has been fired from a job. Getting sober (detoxification) and remaining sober (compliance with the prescribed treatment) do not restore the eye, and usually do not rebuild the broken marriage or regain the lost job. Active alcoholism in an individual may be gone, perhaps forever, but the destruction it may have caused often lasts indefinitely.

Myth: Once an addict is detoxified, as long as he or she does not take the abused substance (or a different abused substance) again, any changes in the pathways of the brain that had been caused by the abused substance disappear, and the brain returns to a more fully healthy state.

Fact: The brain usually returns to a better state of health than when the addiction was at its worst, but it takes a very long time to return completely to the health it enjoyed before the substance abuse began. For many addicts, part of the brain damage is permanent.

Myth: A single, simple course of treatment ought to produce a permanent total cure in an addict.

Fact: As a chronic disorder, addiction needs a lifelong treatment, like diabetes, asthma, arthritis, and high blood pressure.

Myth: Since most persons treated for addiction relapse sooner or later, treatment is by definition unsuccessful, and it makes no sense to try it.

Fact: Treatment is not unsuccessful because further treatments are needed. Suppose a diabetic is brought to the Emergency Room unconscious from extremely high blood-sugar, is treated with insulin, regains consciousness, and reduces the blood-sugar level to normal. The patient will probably need insulin every day for the rest of his or her life, but this emergency treatment was certainly successful. With addiction, as with diabetes, we must see treatment as an ongoing process, successful if at the time it reduces the severity of the disorder. It unfortunately does not have a permanent fix, like setting a broken bone or surgically removing all of a cancer. The goal is improvement, not cure.

Myth: Addiction is voluntary; addicts "bring it on themselves." Everyone has enough free will not to become an addict.

Fact: The choice to try an addictive substance for the first time may be voluntary. Freedom even in this choice may be weakened by such factors as peer pressure, an inherited biological condition predisposing one to a craving for this substance, or a valid reason for taking it once (for example, as a

pain-killer prescribed by one's physician). But as the person slips from the first use to repeated use to misuse to full-fledged addiction and chemical dependence on the substance, freedom of choice diminishes and usually disappears.

Myth: There are no degrees of addiction. It is an all-or-none condition. A person is either a nonaddict and never takes the tiniest amount of an abused substance or is a hopeless addict whose life centers on enjoying maximum amounts of the abused substance (or substances) all day every day for life.

Fact: At one extreme, there is an occasional addict who is satisfied with a low dose of an abused substance and who functions at a normal level at home and on the job. At the other extreme is the addict who regularly takes such huge doses of the abused substance as to pass out in a life-threatening coma. There is, indeed, a formal system for measuring the severity of a patient's addiction and the success of treatment at any given moment. It is called ASI (for Addiction Severity Index). It considers such factors as whether the patient's substance abuse is decreasing, whether the patient is functioning better socially and enjoying better general health (rarely a complete return to the state before the first use of the abused substance), and to what degree, if any, the patient presents a danger to public health and safety (treatment of an alcoholic who continues to drink but has stopped driving after drinking as a result of psychotherapy would be a partial success).

Myth: If treatment were possible, it would cost millions of dollars to treat a single patient. Treatment would cost more than putting a young person in prison for life. In terms of dollar value, treatment would cost even more than a single addict would be apt to steal in a lifetime.

Fact: One study in California showed that the benefits of treatment outweighed the cost of treatment at least four-to-one and as high as twelve-to-one, depending on the type of substance abused and the type of treatment employed. It is nontreatment which costs the United States billions of dollars a year.

Myth: Even if methadone keeps an addict away from heroin, the methadone itself will leave the patient drugged and dangerous, so the patient might as well have stayed on heroin.

Fact: Methadone simply does not cause a drugged state, or even the appearances of a drugged state.

Myth: Even if methadone keeps an addict away from heroin and even if the methadone does not seem to leave the patient drugged and dopey, the patient could function successfully only at undemanding jobs such as raking leaves or checking out books in a library. Even this relatively fortunate patient would be, in effect, in a dangerous position in a job requiring quick reflexes or motor skills, a job such as driving a subway train or operating a fork-lift.

Fact: Many persons on methadone can safely drive trains and run fork-lifts. Some people on methadone cannot do so. The difference between these two groups is not caused by the methadone, but by factors such as lack of education (we don't want people driving trains or busses who can't read traffic signs or safety notices), physical problems (a patient who lost both eyes while driving drunk obviously cannot drive anything), or psychological problems (a patient who panics to the point of paralysis or fainting should not drive). Methadone will not create or increase a danger even for these high-risk jobs, but neither will methadone remove a risk caused by a previously existing condition.

JAMES T. MCDONOUGH, JR.

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NA *See* Narcotics Anonymous (NA)

NALOXONE Naloxone is an OPIOID ANTOGONIST (i.e., a blocker of morphine-like agents) commonly used to reverse the actions of drugs such as morphine. In the early 1990s, it was the treatment of choice for reversing the life-threatening effects of opioid overdose. Structurally, naloxone is very closely related to OXYMORPHONE, both compounds being derivatives of the opium alkaloid thebaine. Indeed, the structural differences between oxymorphone and naloxone are minimal; they are restricted to a simple substitution on the nitrogen atom. Oxymorphone has a methyl group whereas naloxone has an allyl substitution. This small substitution changes the pharmacology of the compound dramatically. Whereas oxymorphone is a potent ANALGESIC with actions very similar to MORPHINE, naloxone has no analgesic actions by itself and instead has the ability to antagonize, or reverse, virtually all the effects of morphine-like drugs. This ability to reverse opiate actions has proven valuable clinically. However, giving naloxone to opiate addicts will immediately precipitate WITHDRAWAL symptoms.

Naloxone is rapidly metabolized in the liver to inactive compounds, resulting in a relatively brief duration of action. When naloxone is used clinically to reverse the actions of morphine and other OPIATES, care must be taken to ensure that the drug being reversed does not last longer than the naloxone. Should that happen, a patient may be revived by naloxone only to relapse back into a coma or even die from the side effects of the initial opioid AGONIST. Despite its effectiveness following injection, naloxone is not very active when given orally; this, together with its short duration of action, prevents its widespread use as a treatment for opioid addiction.

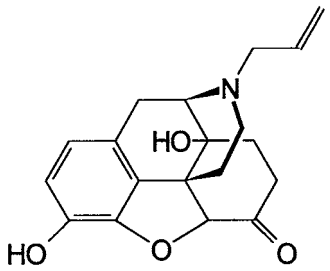

Figure 1
Naloxone

(SEE ALSO: *Naltrexone; Naltrexone in Treatment of Drug Dependence; Opioids: Complications and Withdrawal*)

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NALTREXONE Naltrexone (brand name ReVia) is an OPIOID ANTAGONIST (i.e., a blocker of substances with morphine-like actions), with a structure very similar to another antagonist, NALOXONE. It also closely resembles the potent ANALGESIC (painkiller) OXYMORPHONE. The differences between naloxone and naltrexone are restricted to a simple substitution on the nitrogen atom, with naltrexone having a methylcyclopropyl group, yet this small substitution changes the pharmacology of the compound dramatically. Naltrexone has no analgesic actions by itself and has the ability to antagonize, or reverse, virtually all the effects of morphine-like drugs. Like naloxone, naltrexone will precipitate WITHDRAWAL in physically dependent people.

Naltrexone is rapidly metabolized in the liver, but one of its metabolites is 6-naltrexol, which has some activity and a longer duration of action. In the 1990s, naltrexone was used to treat opiate addiction and for rapid opioid detoxification. Its greater potency than naloxone, along with its greater and longer activity after oral administration, has made this the antagonist of choice (for clinicians) in the treatment of opioid addiction.

In the early 1990s, several research groups reported that naltrexone, when given to alcoholic men following detoxification, reduced the likelihood of relapse to ALCOHOL. This finding secured to support the hypothesis that some of the reinforcing (euphoric) effects of alcohol are due to interactions with naturally occurring opioid systems in the brain.

A study published in 1999 supported this conclusion (Davidson et al., 1999). The findings suggested that naltrexone reduces the desire and craving for alcohol while sometimes increasing the negative side effects, including headaches. Naltrexone has been shown to be especially effective when combined with behavioral therapy.

(SEE ALSO: *Naltrexone in Treatment of Drug Dependence; Treatment: Alcohol; Treatment Types: Pharmacotherapy*)

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REVISED BY REBECCA MARLOW-FERGUSON

NALTREXONE IN TREATMENT OF DRUG DEPENDENCE Naltrexone (brand names Trexan^{R}, Revia^{R} [U.S.], Nalorex^{R} [France, U.K.]) is a synthetic antagonist of opiate (morphine-like) drugs, which blocks their actions without itself having any opiate effects. Naltrexone differs from most other pure opiate antagonists in having a relatively long duration of action (at least 24 hours) and being effective when taken by mouth. These characteristics have led to its clinical use as a long-term or maintenance treatment for OPIATE and OPIOID dependence after detoxification. Naltrexone is also being studied experimentally as a possible treatment for cigarette smoking and eating disorders, and was approved in 1995 for treatment of alcoholism.

The use of opiate ANTAGONISTS as treatment for opiate dependence was first proposed by William Martin and Abraham Wikler and their colleagues at the U.S. Addiction Research Center in the early 1960s. They hypothesized that chronic administration of an opiate antagonist, by blocking the pleasurable or rewarding effects of opiate drugs, would lead to the extinction of drug-seeking and drug-taking behavior—since the addict would no longer receive any pleasurable effects from taking

an opiate. With abstinence from opiates, PHYSICAL DEPENDENCE and any chronic withdrawal syndrome would dissipate, removing important factors that cause craving for opiates. They suggested that antagonist treatment would have several advantages over treatment with an opioid such as METHADONE. Since antagonists do not produce any pleasurable effects, the addict would have little incentive to misuse the medication or divert it to illegal channels. Chronic use of an antagonist would not produce physical dependence, and an overdose of antagonist would not cause life-threatening opiate effects such as suppression of breathing. Use of the antagonist in nondetoxified opioid addicts, however, would cause an acute but not life-threatening withdrawal.

HISTORY

The earliest studies of opioid antagonists were not satisfactory, because of drawbacks in the then available antagonists. For example, NALOXONE was short-acting and not very effective when taken by mouth. Nalorphine and cyclazocine had some kappa-opioid effects (i.e., were not pure antagonists), which produced unpleasant side effects.

Further work was stimulated by the SPECIAL ACTION OFFICE FOR DRUG ABUSE PREVENTION created by President Richard M. Nixon in June 1971 as part of a "war on drugs." The 1972 funding legislation for this office called for research on "long-lasting, nonaddictive, blocking and antagonist drugs . . . for the treatment of heroin addiction." Eventually twenty-two studies with naltrexone (which had been synthesized by Blumberg and Dayton in 1965) were conducted at various treatment programs in the United States. These studies demonstrated the safety and effectiveness of naltrexone after detoxification as a long-term treatment for opiate dependence, leading to its marketing in North America and Europe. Its effectiveness, however, was defined in terms of blocking the effects of HEROIN, not in the success of changing the behavior of heroin users.

TREATMENT

Naltrexone is usually used in conjunction with counseling and other rehabilitation services, as part of a structured and monitored treatment program. The best treatment results tend to occur in highly motivated, psychologically healthy addicts who are employed and well-functioning socially, especially when they face severe economic or legal consequences for failing treatment. For example, addicted health professionals whose treatment is required by their professional licensing boards and monitored as a condition of continued licensure will regularly take naltrexone for several years and remain abstinent from opiates. Some programs have reported five-year success rates as high as 95 percent. Most street addicts (e.g., those with unstable living situations who support their drug use by criminal activity) refuse to take naltrexone or, if started in treatment, quickly drop out. This is believed due to the lack of reward effect. Many such addicts prefer maintenance treatment with the synthetic opiate methadone and others find even methadone nonrewarding, so they relapse.

Fifty milligrams of naltrexone block the effects of 25 milligrams of heroin for 24 hours, so the typical weekly naltrexone dose is 350 milligrams. The actual medication schedule is adjusted to the individual patient and may range from 50 milligrams every day to 150 milligrams every third day. Patients are put on the least frequent medication schedule possible to enhance patient cooperation and reduce the number of clinic visits. To further reduce medication scheduling, researchers are working on a depot form of naltrexone that can be injected once a month and which slowly releases the medication into the body.

Care must be taken to avoid administering naltrexone to individuals still physically dependent on opiates. In opiate-dependent individuals, an antagonist will precipitate an acute opiate withdrawal syndrome. While not life-threatening, this syndrome can be extremely uncomfortable, with symptoms such as abdominal cramps; diarrhea; muscle, joint, and bone pain; runny nose (rhinorrhea); and goose bumps (piloerection). To avoid this situation, naltrexone is not administered to patients until they have been free of opiate drugs for at least seven to ten days to allow dependence to wear off. To confirm the absence of dependence, patients may be challenged with the short-acting antagonist naloxone before starting on naltrexone. To shorten the required opiate-free period, some programs are experimenting with combined administration of naltrexone and CLONIDINE, a medication that reduces symptoms of opiate withdrawal.

Naltrexone was shown to reduce the rate of relapse of full-blown compulsive drinking by detoxified alcoholics, although it did not substantially increase the number who were totally abstinent. In one research study, naltrexone seemed to reduce craving for alcohol. In contrast with opioid addicts, alcoholics were more willing to take naltrexone.

(SEE ALSO: *Treatment/Treatment Types; Wikler's Pharmacologic Theory of Drug Addiction*)

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NARA *See* Narcotic Addiction Rehabilitation Act

NARCANON/SCIENTOLOGY *See* Cults and Drugs

NARCOTERRORISM *See* Terrorism and Drugs

NARCOTIC The term derives from the Greek *narkōtikos*, meaning benumbing. It was originally used (since the fourteenth century) to refer to drugs that produced a stupor associated with pain relief (analgesia), primarily OPIUM and its derivatives, the morphine-like strong ANALGESICS, or the opium-like compounds (OPIOIDS)—these, in moderate doses, dull the senses, relieve pain, and induce profound sleep but in large doses cause stupor coma, or convulsions.

During the nineteenth century, the term was widely used to include a number of agents that produced sleep. Toward the end of the nineteenth century, the term came to imply drugs that could lead to addiction, and so by the turn of the twentieth century, "narcotic" came to describe drugs as diverse as opioids and COCAINE. During the twentieth century, the term became widely used in a legal context to refer to psychoactive drugs and drugs of abuse—those subject to restriction—as "addictive narcotics," whether in fact the agents were physiologically addictive and narcotic or not. This imprecise usage has left the term nebulous, although it is still used extensively in the media and by the general population. The term is no longer used in scientific discourse to categorize drugs.

(SEE ALSO: *Drug Types; Opiates/Opioids; World Health Organization Expert Committee on Drug Dependence*)

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NARCOTIC ADDICT REHABILITATION ACT (NARA) Public Law 89-793, the Narcotic Addict Rehabilitation Act (NARA), was passed by Congress in 1966. This legislation was designed to allow the use of the federal courts and criminal-justice system to compel drug addicts to participate in treatment. Several developments provided the context for this legislation. In the early 1960s, the problem of NARCOTIC drug use and ADDICTION were perceived to be increasing. There was also a perception that treatment was not particularly effective and that the RELAPSE rate was high. In response, California, in 1961, and New York, in 1962, passed legislation permitting the CIVIL COM-

MITMENT of narcotic addicts; that is, they could be compelled to accept treatment even if they had committed no crime but could be shown to be using illicit narcotic drugs. In both of these states the legislatures also provided substantial funds to establish residential facilities where addicts could be treated initially as well as aftercare programs to provide supervision following their release from the residential facilities. Several other states, including Illinois, passed similar civil commitment legislation, but only New York and California launched massive programs to implement compulsory treatment and civil commitment.

In January 1963, the Presidential Advisory COMMISSION ON NARCOTIC AND DRUG ABUSE appointed by President John F. Kennedy made a number of recommendations, including the enactment of a federal civil commitment statute that could provide an alternative to prison for confirmed narcotic or marijuana abusers convicted of federal crimes. The advisory commission also recommended increased assistance to states and municipalities to develop and strengthen their own treatment programs.

As passed by Congress, NARA had four titles, or main parts: *Title I* provided that eligible addicts charged with a federal offense could choose civil commitment or treatment instead of prosecution. After being examined by clinicians at a treatment center, an addict, if found suitable, could be committed to the custody of the surgeon general for thirty-six months of institutional treatment and aftercare. *Title II* provided for civil commitment after conviction. *Title III* stated that even if no federal crime had been committed, an addict or a related individual could petition the U.S. attorney in the district of residence and, if local facilities were unavailable, the U.S. District Court could commit the person to custody of the surgeon general for treatment. *Title IV* provided for funding to states and localities to establish or expand treatment for addicts.

Treatment under NARA began to be provided in 1967. The two U.S. PUBLIC HEALTH SERVICE HOSPITALS—in Lexington. Kentucky, and Fort Worth, Texas—which had been treating both addicted federal prisoners and voluntary patients, were redesignated "Clinical Research Centers" and became the sites for the institutional phase of treatment for addicts committed to the Surgeon General under NARA. Aftercare was provided by local programs supported by contracts with the NARA program administered by the Division of Narcotics within the National Institute of Mental Health (NIMH).

From 1967 through 1973, the two clinical centers admitted more than 10,000 NARA patients, 5 percent under Title I, 2 percent under Title II, and 93 percent under Title III. Women made up 15 percent of admissions. Race and ethnicity were noted for admissions between 1970 and 1973, during which time the designations and distribution were as follows: Anglo 43 percent, black 47 percent, Puerto Rican 1 percent, Mexican American 9 percent.

Many of the patients referred were found "not suitable for treatment" (38% at Fort Worth and 51% at Lexington), a designation that generally meant they were too disruptive or antagonistic. Some of this unsuitability was deliberate. Many of those under Title III, while not being charged with a federal crime, were under court pressure because of state or local crimes; as part of plea bargaining with local courts, they agreed to accept commitment under NARA Title III. They quickly learned that the centers would not require them to stay in residence, nor would NARA officials compel them to stay in aftercare. Once released from the centers as "not suitable," they would find ways to convey to the local courts how motivated for treatment they still were and how puzzled they were not to be offered treatment.

The general approach to treatment during the residential phase was based on THERAPEUTIC COMMUNITY principles, which delegate many responsibilities to former addicts and to patients participating in the program. The average duration of the residential phase of treatment was intended to be about 6 months, but of those admitted for examination, only about 35 percent were discharged to aftercare as having completed the residential phase. A number of studies have been conducted on the effectiveness of the NARA program, including aftercare. One study found that only 38 percent of the 35 percent that completed the residential phase remained in aftercare for the full six months after discharge from residential treatment. Reasons for attrition included death, disappearance, recommitment, conviction, and incarceration. One study by Gold and Chatham in 1971 found that 46 percent of addicts in aftercare had used an illegal drug during the month preceding the interview;

about 50 percent were working. Another study found that 87 percent had used narcotics during the first six months after the residential phase; 65 percent had become readdicted.

While this rate of readdiction did not seem as bleak as that seen after the discharge of the early cohorts from Lexington, it was not seen as particularly successful—given the high cost of the six-month residential phase and the high attrition rates. Because of the attrition, the readdiction rate, while not inevitable, was occurring among only the better candidates. Another study by Mandell and Amsel (1973) compared the outcome of those treated compared to those found "not suitable" for treatment. The difference in outcome between the two groups was not significant.

While the legal authority for federal civil commitment remained in effect through the early 1990s, the actual application of NARA felt into disuse in the mid-1970s as more federal prisons developed programs for Title II offenders and as more communities developed their own treatment programs. The use of treatment under civil commitment also declined, because the involvement of courts and expensive legal procedures made it far more expensive than voluntary treatment. In 1971, the Fort Worth facility was closed and turned over to the Bureau of Prisons. The Lexington facility experienced the same fate in 1974.

(SEE ALSO: *California Civil Commitment Program; Civil Commitment; Coerced Treatment; New York State Civil Commitment Program*)

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NARCOTICS ANONYMOUS (NA) Even though the origins and strategies of Narcotics Anonymous (NA) are closely intertwined with those of ALCOHOLICS ANONYMOUS (AA), NA has devised its own unique adaptations to them. There is no question that NA's roots were in the AA program, but it soon came to realize its uniqueness and had to give AA's program its own "spin." Briefly sketched—an energetic, relatively new AA member, while doing twelve-step work in 1944, recruited an alcoholic who was also an abuser of MORPHINE (he used this drug to avoid hangovers). The AA program helped the recruit with alcohol, but not with morphine. He soon found himself an involuntary patient in the U.S. PUBLIC HEALTH SERVICE HOSPITAL in Lexington, Kentucky.

In the meantime, his AA sponsor, who was much puzzled by AA's help with alcohol but not other drugs, was transferred to Frankfort, Kentucky, near the Lexington Hospital. He (dubbed "Houston" in a *Saturday Evening Post* article) reportedly repeated to himself, "I was convinced that the TWELVE STEPS would work as well for drugs as for alcohol" (Ellison, 1954:23). As a result, Houston called on Dr. V. H. Vogel, the director of the Lexington Hospital, and told him of his convictions and his partial success with his AA "pigeon." Further, he offered to start a group directed at drugs in the hospital and Dr. Vogel agreed. The first meeting was on February 16, 1947. Weekly meetings have gone on ever since.

In 1948, an addict known as Dan returned to the hospital from New York City for the seventh time; after a period of severe withdrawal, he began attending the meetings begun by Houston the year before. Dan, Houston, and Houston's former AA "pigeon" spent many hours together apart from the regular meetings. From these discussions Dan experienced a miraculous change, focusing enthusiastically on the twelfth step of AA. In high spirits, he returned to New York hoping to form the first group outside Lexington Hospital—and to call it Narcotics Anonymous. Dan looked up others whom he had known at Lexington and suggested weekly meetings. Only three responded: a barber, a housepainter, and a waiter. No organization was then willing to provide them with a room for a meeting until the Salvation Army provided one. Slips plagued the first few months, but three of the original four remained committed. Slowly, the group grew in size despite disputes over policy—for ex-

ample, should withdrawal from drugs be done "cold turkey" at home or within institutional care? The group finally decided to encourage the latter.

As NA emerged, it faced a dilemma. On the one hand, it wished to use the basic AA strategies and program that were directed solely against alcohol. On the other hand, it attracted, as did AA itself, many who abused a rather wide variety of drugs besides alcohol. At first, NA attracted mainly HEROIN users; later, abusers of BARBITURATES, AMPHETAMINES, and MARIJUANA began to appear at meetings. As the NA groups spread from New York City to other cities, AA groups began to thrash out a policy on the matter that further encouraged the formation of NA groups. The policy came to be known as "cooperation, but not affiliation" between AA and NA. The result was that AA freely offered their steps and traditions to NA for adaptation but steadfastly clung to their singleness of purpose—namely, to encourage alcoholics only to join. Thus, NA had to deal with a variety of drugs, not a sole prominent one, such as alcohol.

In their meetings, NA members tended to focus on the differences between the various drugs they had abused, thereby creating considerable chaos. Slowly, however, they decided on a radical change in the wording of step one. Rather than "We admitted we were powerless over drugs," they decided on "We admitted we were powerless over our ADDICTION." In other words, what all members had in common was a belief that they suffered from a disease of addiction. They pass on their experiences and hopes to the addict who still suffers; they do not become embroiled in the differing features of the various drugs to which members were addicted. In this respect, they are quite different from Cocaine Anonymous, a group that focuses on only one drug, cocaine.

(SEE ALSO: *Addiction: Concepts and Definitions; Disease Concept of Alcoholism and Drug Abuse; Rational Recovery; Treatment Types: Self-Help and Anonymous Groups*)

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NATIONAL ASSOCIATION OF STATE ALCOHOL AND DRUG ABUSE DIRECTORS The National Association of State Alcohol and Drug Abuse Directors, Inc. is a private, not-for-profit organization comprised of State Alcoholism Agency and State Drug Agency directors. The association promotes and supports the development of alcohol and drug abuse prevention and treatment programs in each state. It provides a variety of services to the states including training, technical assistance, the collection and analysis of data, and the spread of information and technology. It is a tax-exempt organization that does not engage in political activities. It was incorporated in 1971 to support the State Drug Agency Directors and was expanded in 1978 to include The State Alcoholism Agency Directors.

The association's objectives are to:

- Promote the development of alcohol and drug abuse programs in each state;
- Facilitate the evaluation, spread, and exchange of alcohol and drug abuse information among members and other interested parties;
- Aid federal and state governments in the development and execution of alcohol and drug abuse programs;
- Encourage the federal government to interact with the states in the planning and use of government resources;
- Identify common and different alcohol and drug abuse problems among the states and assist in the development of programs tailored to each state's need; and
- Identify problems that require study and research.

In its aim to serve as an educational and informational organization, the association produces several publications. It's studies and publications have been widely cited. The annual report, entitled *State Alcohol and Drug Abuse Profiles*, provides information on state fiscal resources, services, model products, drug trends, and special needs populations. The annual report provides invaluable information which allows for program comparisons between states, replication of creative programs and services by other states, and the dissemination of policy issues. The *State Substance Abuse Quarterly* is distributed to members, National Prevention Network members, and other interested parties. In addition, the association produces "*Special*

Reports", which cover a variety of topics and are published several times each year. Also, it publishes a series of reports covering the effectiveness of alcohol and drug treatment including *Alcohol and Other Drug Treatment: Policy Choices in Welfare Reform* and *Investment in Treatment for Alcohol and Other Drug Problems: It Pays*.

The association has an annual meeting in conjunction with the National Prevention Network.

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NATIONAL COMMISSION ON MARIHUANA AND DRUG ABUSE In response to a substantial increase in drug-use patterns in American society during the 1960s and a swirling controversy about changing the marijuana laws to legalize the substances, in 1970, the U.S. Congress established the National Commission on Marihuana and Drug Abuse. The commission was directed to conduct a two-year study, the first on MARIJUANA and the second on "the causes of drug abuse and their relative significance." The commission was composed of thirteen members, four appointed by the Congress (two each from the Senate and the House) and nine appointed by the president. The chair of the commission was Raymond P. Shafer, former governor of Pennsylvania, and the vice chair was Dana L. Farnsworth, M.D., the director of Student Health Services at Harvard University.

In March 1972, the commission issued its first report, *Marihuana: A Signal of Misunderstanding*, which recommended decriminalization of possession of marijuana for personal use. The commission's final report, *Drug Use in America: Problem in Perspective*, was issued in March 1973. The 500-page report was supplemented by 1,000 pages of appendices. In its report, the commission summarized its findings concerning the patterns of drug use in the United States, psychosocial and institutional influences on drug-using behavior, and the social impact of drug dependence and drug-induced behavior. The commission also proposed a framework for policymaking and made specific recommendations in the areas of legal regulation, prevention, treatment and rehabilitation, and research.

The most enduring impact of the commission's final report probably lies in its efforts to revise the vocabulary of the drug field. The commission insisted that ALCOHOL be recognized as the major "drug" problem in the United States; it recommended that the term "drug abuse" be eschewed in favor of more descriptive terminology concerning drug-using behavior. For example, the commission developed a typology of drug-using behavior (experimental, recreational, situational, intensified, and compulsive use) and emphasized the need for different social responses for different patterns of use. In another important contribution, the commission fostered the development of information systems for monitoring changes in drug-using behavior in U.S. society, including national surveys of drug-using behavior among high-school students and in the general population.

The commission strongly endorsed the national treatment strategy, codified in the Drug Abuse Office and Treatment Act of 1972, which aimed to create a national network of treatment services and to establish appropriate incentives for people to seek these services voluntarily. In addition, the commission sought to reorient the rule of the criminal law in implementing a policy of discouraging drug use. In the short term, the commission concluded, the criminal sanction should be retained, but should be utilized primarily as leverage for entry into prevention and treatment programs. In regard to government organization, the commission recommended that the law-enforcement and public-health dimensions of national drug-abuse prevention policy be combined into a single agency.

(SEE ALSO: *Commissions on Drugs; Marihuana Commission; U.S. Government*)

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NATIONAL COUNCIL ON ALCOHOLISM AND DRUG DEPENDENCE (NCADD)

This is the ninth largest voluntary health organization in the United States and the country's major public advocate for the prevention and treatment of alcohol and other drug problems. Working through hundreds of local affiliate councils, state councils, and its New York City and Washington offices, NCADD sponsors prevention and education programs, information and referral services, scientific and clinical consensus development, public policy advocacy, and other related activities.

NCADD was established in 1944 as the National Committee for Education on Alcoholism. As the organization grew, its name and scope enlarged. It became the National Committee on Alcoholism in 1950, was renamed the National Council on Alcoholism in 1957, and assumed its present name in 1990.

The NCADD was the idea of a single individual, Marty Mann; she was its director until her retirement in 1968 and its guiding spirit until her death in 1980. Mrs. Mann was the first woman to recover from alcoholism through the fellowship of Alcoholics Anonymous (AA). During the early years of her recovery, she became increasingly aware that the United States was uninformed about the disease of Alcoholism. The resulting stigma and prejudice kept alcoholics and their families from receiving the medical, social, and spiritual help they needed to recover. The structure and traditions of AA prevented it from becoming a public-health agency similar to those concerned with promoting prevention, treatment, and research for polio, tuberculosis, cancer, and heart disease. With the support of the Yale Center of Alcohol Studies, the council was incorporated and an office was established in the New York Academy of Medicine building in New York City. In 1950, it became independent of Yale. Ruth Fox, a psychiatrist who had helped found the council, became its first medical director in 1958. In 1969, she was succeeded by Frank A. Seixas, an internist.

During its early years, the council's activity consisted mainly of developing literature and presenting lectures to professional and lay groups on the concept of alcoholism as a disease and of organizing local affiliates to pursue this educational process in their own communities. By 1947, a survey of American adults showed that 36 percent believed alcoholism to be a disease, a remarkable increase from 6 percent who held this view in 1943. As interest in alcohol and drug problems expanded, the council developed and then published in 1972 the first set of medical criteria for the diagnosis of alcoholism. In 1976, it sponsored Operation Understanding, in which fifty-two men and women known for their contributions in the areas of government, medicine, industry, science, journalism, and the arts publicly revealed their histories of recovery from alcoholism.

These and other activities have made NCADD an important force in the nation's development of service systems and health policy related to alcohol and other drug problems. NCADD helped establish the first industrial alcoholism programs, the first research society devoted to alcoholism, the first public education campaigns to promote the concept of alcoholism and other drug dependence as diseases, the movement to recognize the special needs of Women with substance-related problems, and the nation's effort to understand and prevent Fetal Alcohol Syndrome (FAS) and other effects in the fetus.

NCADD is also a leader in the U.S. campaign against alcohol-related highway Accidents and in promoting appropriate treatment services for substance-dependent pregnant and postpartum women and their children. Through its local affiliates, NCADD provides direct services, including education and prevention, in school and community settings, as well as information, intervention, and referral counseling, local alcohol- and drug-awareness campaigns, and other related activities.

(See also: *American Society of Addiction Medicine; Association for Medical Education and Research in Substance Abuse; Disease Concept of Alcoholism and Drug Abuse; Parents Movement; Women for Sobriety*)

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NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE (NHSDA) The National Household Survey on Drug Abuse (NHSDA) is the primary source of statistical information on the use of illegal drugs by the population of the United States. Conducted periodically by the federal government since 1971, the survey collects data by administering questionnaires to a scientifically selected sample of persons age twelve and older living in the nation. The primary purpose of the survey is to estimate the prevalence of illegal drug use (i.e., the number of people using illegal drugs) in the United States, and to monitor changes in prevalence over time.

Legal drugs, such as ALCOHOL and TOBACCO, are also covered by the survey. Prevalence rates (the percentage of the population using any type of drug) for various population subgroups and for various types of drugs are generated from the survey data; these rates are compared by analysts to provide insight into which population groups are most prone to illicit drug use—which drugs are most commonly used. These basic statistics are used by the federal government in planning federal policies and funding priorities related to substance abuse. Statistical reports, containing the survey estimates and descriptions of the surveys, have been routinely published. The raw survey data are also available on data tapes, which are widely used by substance-abuse researchers studying the EPIDEMIOLOGY of substance abuse, and the results of these studies are published in professional journals.

HISTORY OF THE NHSDA

The NHSDA traces its origin to a survey conducted by the NATIONAL COMMISSION ON MARIHUANA AND DRUG ABUSE (1970–1972). The commission required baseline data on the public's beliefs, attitudes, and use of marijuana, to satisfy its charge of developing recommendations for legislation and administrative actions in helping to deal with the illicit drug problem. Through a private contractor, they conducted two surveys, in 1971 and 1972. The NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) continued the survey in subsequent years (1974, 1976, 1977, 1979, 1982, 1985, 1988, 1990, and 1991) to satisfy the continuing need for current data. Starting in 1990, the survey was conducted annually. In 1992, sponsorship of the survey was transferred to the newly created SUBSTANCE ABUSE AND MENTAL HEALTH SERVICES ADMINISTRATION (SAMHSA). All the surveys were conducted by private contractors selected by the government.

Expansion of the survey took place in 1985 with the implementation of a new sample design that had larger samples of African Americans and HISPANICS (resulting in a sample size of 8,038). Further expansions took place in 1990, with the intensive sampling of the Washington D.C., metropolitan area as a part of the survey and in 1991, with the addition of five more oversampled metropolitan areas and an increase in the national sample component (for a total sample of 32,594 in 1991). The metropolitan oversampling was continued through 1993, but beginning in 1994 the survey was scaled back to a national sample of about 18,000 interviews. All surveys conducted from 1971 through 1991 were done at a particular time of year, usually spring or fall. In 1992, a continuous data collection design was implemented—with quarterly samples and January to December data collection. A major revision to the survey questionnaire was also implemented in 1994, to improve the validity and reliability of the survey estimates. That year, the NHSDA began using an improved questionnaire and estimation procedure based on a series of studies and consultations with drug survey experts and data users. Because this new methodology produces estimates that are not directly compa-

rable to previous estimates, the 1979-1993 NHSDA estimates presented in the 1998 report were adjusted to account for the new methodology that was begun in 1994.

The 1998 NHSDA employed a sample of 25,500 persons. This sample included augmented samples in California and Arizona (4,903 and 3,869 respectively).

DESCRIPTION OF THE SURVEY METHODOLOGY

Since its inception, the NHSDA has undergone various design changes affecting primarily the sample design, as described above.

Target Population. Prior to 1991, the NHSDA covered all persons age twelve and older living in households in the forty-eight contiguous states. Beginning in 1991, this was modified so that the survey covers the civilian noninstitutionalized population aged twelve years old and older within the fifty states. In addition to including all household residents (except persons on active MILITARY duty), it includes the residents of noninstitutional group quarters (e.g., shelters, rooming houses, dormitories) as well as residents of civilian housing on military bases. Persons excluded from the target population are those with no fixed address, residents of institutional quarters (e.g., jails and hospitals), and active-duty military personnel.

Sample Selection. A complex multistage sample design is used to select people to be respondents in the survey. The first stage of sampling is the selection of nonoverlapping geographic primary sampling units (PSUs), consisting of counties or metropolitan areas. For the second stage of sampling, area segments (constructed from U.S. Census block groups or enumeration districts) are selected within each PSU. Field staff count and list all dwelling units within sample segments and mark their location on a map. A *dwelling unit* is either a housing unit, such as a house or apartment, or a group-quarters unit, such as a dormitory room or a shelter bed. From these listings, a sample of dwelling units is then selected by sampling staff, and interviewers are assigned to contact these dwelling units.

Prior to arrival at the sample dwelling unit (SDU) an introductory letter is mailed to the SDU, briefly explaining the survey and requesting participation. When the interviewer visits the SDU, a brief screening interview is conducted that involves listing all SDU members along with their basic demographic data on a screening form. The interviewer identifies which SDU member(s) will be asked to participate in the survey, based on the composition of the household. This selection process is designed to provide the necessary sample sizes for specified population groups.

Questionnaire Administration. Interviewers control the questionnaire administration, but to enhance respondent confidentiality, drug-use questions are answered by respondents on self-administered answer sheets that are not reviewed by interviewers. As the respondent records the answer choices and completes each answer sheet, they are placed in an envelope. At the end of the interview process, all materials are sealed in this envelope by the respondent and mailed to the data-processing site with no personal identifying information attached.

Data Processing. All questionnaires are received by mail at a data-processing site, where they are checked for critical identification and demographic data and then all data are entered onto a computer data base. Consistency checks and other editing is done, after which statistical tables showing estimates of prevalence rates for various drugs are produced. Data are generally released to the public about six months after the end of data collection. Public use data files are available one to two years after completion of data collection.

STRENGTHS AND LIMITATIONS OF THE NHSDA

Strengths. The major strengths of the NHSDA are its size, continuity, and national representativeness. The survey has a sample large enough to allow comparisons of drug-use prevalence among many different population subgroups each year and over time. The length of the questionnaire and amount of data collected provides a rich data base for examining the characteristics of drug abusers, the relationships of drug use with many demographic and other variables, and the changing patterns of drug use over time. The methodology used, while expensive, has been extensively evaluated and found to be effective (relative to other methodologies) in eliciting valid data from respondents. Through intensive call-back procedures, participation rates in the NHSDA have been excellent. The

1998 participation rate for the screening questionnaire was 93 percent and the participation rate for the main questionnaire was 77 percent.

Limitations. The survey does not cover certain populations likely to have heavy illicit drug use, such as the homeless and prison populations. While these missing populations, because they are small, make little difference in estimating MARIJUANA or ALCOHOL prevalence, rarer behaviors such as HEROIN or CRACK use may be severely underestimated by the NHSDA. Data validity from the survey is also in question because of the self-report methods employed and the voluntary nature of the survey.

MAJOR FINDINGS OF THE SURVEY

The NHSDA has tracked the changing nature of drug abuse since 1971. At the time of the first survey, about 10 percent of the population age twelve and older had ever used illicit drugs. This was estimated to be more than double the rate of lifetime use as of the early 1960s. In 1998, an estimated 13.6 million persons or 6.2 percent of the American population of 12 years of age or older were current illicit drug users, meaning they had used an illicit drug in the month prior to interview. The report for current use showed that more than one drug had been used by some of the total 13.6 million, with a breakdown of this figure as follows: Some 11 million reported using marijuana or HASHISH; an estimated 1.8 million cocaine; and 130,000 heroin. The rate of current use of inhalants by Americans has remained steady since 1991 (between 0.3-0.4 percent of the population). The rate of current use of HALLUCINOGENS and PRESCRIPTION DRUGS was estimated at 0.7 percent and 1.1 percent respectively in 1998. By 1998, the estimated number of persons who had tried methamphetamine in their lifetime was 4.7 million (2.1 percent of the population). Current use of illicit drugs reached a peak in 1979 when the estimate was 25 million, or 13.7 percent of the population.

All the NHSDAs conducted since 1971 have shown that marijuana is the most commonly used illicit drug, with current use at 5 percent in 1998. Marijuana initiation among youths 12-17 was at its highest level ever from 1995-1997. Current cocaine use reached a peak in 1985 at 3.0 percent, but the survey showed declines in cocaine use after 1985, to 0.7 percent in 1992. The percentage of current cocaine use did not change significantly between 1992 and 1998.

The NHSDA has shown varying rates of use in different segments of the population. The highest rates of current illicit drug use were found among young people age 18-20 (19.9 percent) in 1998. The rates of use generally decline in each successively older age group, with only 0.7 percent of persons age 50 and older reporting current illicit use.

The surveys have also shown that while illicit drug use occurs in all segments of society, prevalence rates have been greatest among males; in metropolitan areas; and among high-school dropouts. According to the 1998 report, although the rate of drug use was higher among the unemployed, most drug users were employed. The rate of current illicit drug use was also found somewhat higher among blacks (8.2 percent) than among whites (6.1 percent) and Hispanics (6.1 percent). With respect to absolute numbers in the 1998 report, however, most current illicit drug users were white.

The increase in marijuana use among youths age 12-17 has important implications for substance abuse prevention and treatment efforts. In terms of prevention, there is an obvious need to focus immediate attention on children and adolescents. In the long run, the expanding pool of young people using illicit drugs will probably result in continuing pressure on the substance abuse treatment system in future years, as many new drug users progress to addiction and require intervention.

(SEE ALSO: *Drug Abuse Warning Network; Drug Use Forecasting Program; High School Senior Survey; U.S. Government Agencies: National Institute on Drug Abuse*)

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NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM (NIAAA) *See* U.S. Government Agencies

NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) *See* U.S. Government Agencies

NATIONAL PARENTS RESOURCE INSTITUTE (PRIDE) *See* Prevention

NATIVE AMERICANS, DRUG AND ALCOHOL USE AMONG *See* Alcohol: History of Drinking; Ethnicity and Drugs

NATURAL HISTORY OF NARCOTIC USE *See* Opioid Dependence: Course of the Disorder over Time

NEEDLE AND SYRINGE EXCHANGES AND HIV/AIDS The first syringe exchange (SE) program was begun in 1984 in Amsterdam, the NETHERLANDS, out of concern for the spread of hepatitis B among INJECTING DRUG USERS (IDUs). While the hepatitis B virus, hepatitis C virus, and human T cell lymphotropic virus can all cause fatal illness and are all spread through multiperson use (''sharing'') of drug-injection equipment, the threat of human immunodeficiency virus (HIV) has clearly become the dominant force in implementing needle- and syringe-exchange programs throughout the world.

HIV is the causative agent for ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS). As of 1995, HIV infection is eventually fatal; there is no permanently effective treatment for HIV infection. Large-scale vaccination studies began in the late 1980s, and have continued through the 1990s, focusing on some 27 different vaccines (Henderson, 1999). HIV has now been reported among IDUs in sixty countries, from all continents except Antarctica, and from both industrialized and developing nations.

A disturbing facet of HIV infection among injecting drug users is the potential for the rapid spread of the virus through a local population of IDUs. In Edinburgh, Scotland, HIV spread, after the introduction of the virus, into the local population to infect over 40 percent of the local IDUs within two years (Robertson, 1990). In Bangkok, Thailand, the percentage of HIV-infected IDUs (HIV seroprevalence) increased from 2 percent to over 40 percent in less than one year (Vanichseni et al., 1992). In the state of Manipur, India, over 50 percent of the local population of IDUs were infected with HIV within one year after the introduction of the virus into the group. The rapid spread of HIV among IDUs results from a lack of awareness of HIV/AIDS as a local threat and from mechanisms, such as shooting galleries (places where addicts ''shoot up'' together) and dealers' works, that allow large numbers of the population to be exposed to the virus through infected needles and syringes (Des Jarlais et al., 1992). In the United States, injection drug use accounts for 36 percent of AIDS cases overall. In 1998 alone, 31 percent of the 48,269 AIDS cases reported were IDU-related.

Once HIV becomes well established within a population of IDUs, their homosexual and heterosexual partners and transmission to developing fetuses (perinatal) become additional significant problems. In most developed countries, IDUs are the predominant source for both heterosexual and perinatal transmission of HIV. Since AIDS was identified as an epidemic in the United States, 31 percent of all AIDS cases among men have been attributed to injection drug use as compared to the 59 percent of all cases among women (CDC, 1999).

The need to reduce HIV transmission among and from injecting drug users has led to a variety of prevention programs; as a result, there are approximately 113 exchange programs active in 80 U.S. cities in 30 states (Bowdy, 1999). The programs have had differing degrees of effectiveness, although there is evidence that ''education-only'' programs (i.e., those that do not provide the physical means for behavior change) are the least effec-

tive. In almost all industrialized and in some developing countries, increasing legal access to sterile (or uncontaminated) injection equipment has become the most common HIV/AIDS prevention strategy for IDUs. This strategy has included both increased over-the-counter sales of sterile injection equipment and syringe-exchange programs, in which IDUs can turn in used injection equipment for sterile equipment at no cost. A study of a Canadian program in the province of Quebec showed that simple equipment exchanges were not enough. To succeed in reducing the total number of IDUs, transitional and basic support services needed to be part of the program (Belanger, et al., 2000).

Increasing legal access to sterile injection equipment has been politically controversial in several industrialized countries, notably the United States and Sweden, and in many developing countries. Concerns have been raised as to whether increased legal access would lead to increased injection of illicit drugs and whether increased legal access would appear to "condone" illicit drug use or "send the wrong message" about illicit drug use (Martinez, 1992). The decision to support needle exchange programs (NEPs), often lies at the state level. Perhaps the more controversial issue is legalization—or criminalization—of syringe possession. As of 2000, in an effort to reduce the spread of HIV through injection drug use, many states changed laws making it illegal to purchase, sell, or possess syringes without prescriptions. Other states (e.g., New Hampshire) renewed their NEPs. Unfortunately, in most states it is more a political, rather than a public health issue (*AIDS Alert*, 2000).

The empirical data on these questions will be reviewed below, but first it is important to address operational issues involved in needle-exchange programs—to specify how needle exchanges actually work before addressing evaluations of their outcomes.

ORGANIZATIONAL CHARACTERISTICS OF PROGRAMS

At first glance (and regrettably, in much of the public debate about needle-exchange programs thus far), the operation of a program seems quite simple—one would merely select a location and provide staff who could trade new injection equipment for used. In practice, since the exchanges are service-delivery programs, the organization of the services is critical to their effectiveness. Some programs are heavily utilized—for example, the Amsterdam programs exchange approximately 6 million needles and syringes per year in a city with an estimated 3,000 injection drug users. In contrast, the first legal program in New York City traded fewer than 1,000 needles and syringes per year in a city with an estimated 200,000 injecting drug users.

As of 2000, there have been only two comparative studies of the organizational characteristics of the programs (Stimson et al., 1988; Lurie & Reingold, 1993). According to the Stimson study, the most important aspect of an exchange program is "user-friendliness"—which includes such practical considerations as convenient location and convenient hours of operation but also addresses some of the philosophical issues involved.

Perhaps the most vital element of user-friendliness is the nonjudgmental attitude of the staff toward the participants in the exchange. Participants in a user-friendly program are treated with dignity and respect. They are not stigmatized as morally and psychologically impaired simply because they inject psychoactive drugs. The participants are presumed to care about their health and to be capable of taking actions to preserve their health and the health of others.

User-friendliness also requires that exchanges offer multiple services. Other concerns need to be addressed beyond the provision of sterile injection equipment; the sexual transmission of HIV also needs to be prevented, which includes the distribution of condoms without cost. Moreover, the trusting relationships that gradually develop between staff and participants lead to the discovery of other health and social-service needs, especially the need for drug-abuse treatment. The exchange service should be able to respond positively to such needs, either through referral or through on-site provision of assistance. Failure to do so would undermine the trusting relationships between staff and participants.

There is as yet no consensus as to which additional services should be offered on site and which ones through referral—or even a set of available guidelines for how an individual exchange program should decide which additional services to offer on site and which to offer through referral. However, a broad range of additional services are presently being offered on site, with some programs offering conventional drug-abuse treatment, self-help re-

covery groups, women's support groups, tuberculosis screening and treatment, and Bible study groups.

The need to provide on-site (or link to other) services means that exchange programs should be considered a part of a system of services for preventing HIV infection among injecting drug users, rather than as self-sufficient HIV prevention programs.

THE EFFECTIVENESS OF THE EXCHANGES

Studying the effectiveness of an HIV prevention program that facilitates sustained risk reduction is extremely difficult. Research ethics require that comparison subjects be provided with some intervention to reduce their chances of HIV infection, and it is not easy to determine an appropriate comparison condition for a program. Should the comparison subjects be told/permitted to purchase sterile injection equipment from pharmacies? Should they be told to purchase sterile injection equipment through an illicit market? or find some method of disinfecting their own injection equipment?

The logical unit of analysis in an exchange evaluation would be the needs of the local population of injecting drug users rather than the needs of individual drug users. If HIV-infected drug users participate in exchanges—returning their needles and syringes to the exchange rather than passing them on to other injectors—those who do not participate in the exchange would then still be protected against HIV infection. Using communities as the unit of analysis in a clinical trial, however, would be extremely expensive, and it is doubtful that many communities would accept random assignment to experimental or control conditions.

No needle-exchange study as of 2000 has approached a randomized clinical trial. Most studies have measured HIV risk behavior prior to and after participation in an exchange, or have compared risk behavior among exchange participants with that of some other group of injecting drug users. Conclusions about the effectiveness of needle-exchange programs must thus be drawn from the consistency of findings across many methodologically limited studies, rather than rely on a single or small group of methodologically rigorous tests of needle exchange. It should be noted, however, that a consensus panel of the National Institutes of Health in February 1997 concluded that needle exchange programs in general, "show reduction in risk behavior as high as 80 percent in [IDUs], with estimates of a 30 percent reduction of HIV" (Fuller, 1998). In addition, the Centers for Disease Control, the American Medical Association, and the American Public Health Association, have all in some measure acknowledged the amalgam of data pointing toward needle exchange programs as being successful in reducing the incidents of HIV (*AIDS Alert*, 2000).

Jason Farrell, Executive Director of the Positive Health Project, shows syringes to Senior Peer Educator Virgilio Cintron at the agency's offices in New York City, March 6, 2000. The project runs 160 syringe exchange programs for drug users in the U.S. (AP Photo/Jeff Geissler)

Drug Injection. A common concern expressed by opponents to exchange is that the programs would increase the frequency of illicit drug injection. However, research studies have consistently found that such exchange is not associated with any

detectable increase in drug use on either a community or an individual level (Des Jarlais & Friedman, 1992). The most recent review emphasized that "there is no evidence that needle exchange programs increase the amount of drug use by needle exchange clients or change overall community levels of noninjection or injection drug use" (Lurie & Reingold, 1993). Of the eight relevant studies analyzed in this review, three found reductions in injection associated with needle exchange, four found mixed or no effect, and one found an increase in injection compared with the controls. Data from the New York City exchange evaluation (which were not available at the time of Lurie & Reingold's 1993 review) indicate a modest *decrease* in the frequency of injection among participants using needle exchange (Paone et al., 1995).

Moreover, although opponents have often expressed an additional concern—that exchange programs would attract new injectors—the overwhelming number of IDUs participating in exchanges have long histories of drug injection. The mean length of time usually ranges from five to ten years or more. Typically only 1 to 2 percent of exchange participants initiated drug injecting within the previous year. If providing sterile injection equipment had induced large numbers of people to begin injecting drugs, then the numerous studies to date should have observed substantial numbers of new injectors participating in programs.

HIV Injection Risk Behavior. Consistent findings across studies indicate declines in self-reported frequencies of injection with potentially HIV-contaminated needles (Paone et al., 1993). The magnitude of the reduction is difficult to estimate, because studies have used different metrics for risk behavior; some studies have used differences in pre- and post-exchange measurements, while other studies have compared participants with various other groups of drug injectors. Nonetheless, the trend observed from participants in a program has been a reduction in risk behavior, through injection of contaminated equipment, ranging from 50 percent to 80 percent. No studies, however, have shown anything approaching complete elimination of risk behavior among needle-exchange participants.

Exchange programs probably attract drug injectors who are relatively concerned about their health, and it is possible that, even in the absence of exchange programs, these injectors would seek alternative ways of reducing HIV injection risk, such as purchasing sterile injection equipment from pharmacies or on the illicit market. Thus the present data do not permit a conclusion that exchange programs are necessary to reduce risk behavior leading to HIV infection. However, the possibility of alternative methods for reducing injection risk behavior does not imply that an exchange program is not effective in reducing such behavior.

Nevertheless, the fact that very few new injectors participate in exchange programs may be considered a limitation on their current effectiveness. Since IDUs are typically exposed to hepatitis B and C within the first few years of injecting drugs (Hagan et al., 1993), new injectors may already be infected with these blood-borne viruses before they start to obtain sterile injection equipment from an exchange program. Moreover, in cities with high HIV-seroprevalence, even new injectors may be at high risk for HIV infection. In New York City, the estimated seroconversion rate among new injectors is 6.6 per 100 person-years at risk (Des Jarlais et al., 1994). The new injectors may become infected with HIV before they even begin to participate in an exchange program.

Sexual Risk Behavior. While all exchange programs address sexual transmission of HIV to some extent, fewer studies have examined the effect that the program has had on sexual-risk reduction among participants. Moreover, the findings from these few studies are ambiguous. Very few HIV prevention programs for injecting drug users have had consistent success in changing the sexual behavior of IDUs, particularly those with "regular" sexual partners (Friedman et al., 1994). The one exception might be programs that provide HIV counseling and testing, since drug injectors who know they are infected with HIV are more likely to change their behavior to reduce the chances of transmitting HIV to others (Vanichseni et al., 1993).

Effects on HIV and Hepatitis B Transmission. Research data on exchange programs has produced a body of consistent findings with regard to reduced risk behavior through drug injection. Studies within the programs of HIV seroprevalence and HIV seroincidence tend to validate the self-reported risk reduction. Seroprevalence rates have usually stabilized after a program has been implemented, and the rates of new infections among

participants have ranged from zero to less than 1 per 100 person-years at risk to a moderate 4 per 100 person-years at risk in Amsterdam. While there is as yet no definite evidence that participation in a needle exchange reduces the chances of HIV infection, the available HIV seroprevalence and seroincidence data are largely consistent with this hypothesis.

The same behaviors that transmit HIV infection (multiperson use of injection equipment and unprotected sexual behavior) also transmit hepatitis B. The epidemiology of these viruses is similar in most countries, and injecting drug users are at high risk for infection with both viruses.

Studies on the effects of exchange-program participation and new hepatitis B infection among drug users in several cities have shown actual declines (Hagan et al., 1991), further validating self-reported risk reduction and indicating that exchange programs do have a large-scale effect on AIDS risk behavior among injecting drug users.

Discarded Syringes. Exchange programs create an economic value for used needles and syringes—they can be traded for new injection equipment. Thus exchanges have the potential for reducing the amount of used and damaged equipment that is just discarded in the community. Indeed, the one study that systematically examined the amount of discarded injection equipment before and after implementation of an exchange program found a significant reduction in needles and syringes left on sidewalks and in the streets (Oliver et al., 1992)—where anyone might touch it and become a potential victim.

THE "MESSAGE" OF EXCHANGE PROGRAMS

Objections that exchange programs will lead to increased illicit drug use or that they will not lead to reductions in HIV risk behavior can be addressed through empirical studies. Such studies show consistent findings of *no* increase in illicit drug injection and consistent reductions in HIV risk behavior (although it has not yet been possible to translate the reductions in risk behavior into empirically grounded reductions in HIV transmission rates).

A common objection to the programs, however, is that they "condone" or "send the wrong message" about illicit drug use. The symbolism of a government providing the equipment needed for the injection of illicit drugs seems to contradict society's fundamental disapproval of illicit drug injection; and exchange participants do not misinterpret a need to prevent HIV infection as indicating a reversal of prevailing societal attitudes toward the injection of psychoactive drugs.

The important political message in the programs is not that the injection of drugs like HEROIN and COCAINE is a social good but that previous policies on illicit drug use cannot cope with a public-health catastrophe such as HIV infection among injecting drug users, their sexual partners (and theirs), and their children. The "war on drugs" or "ZERO TOLERANCE" approach focused on reducing the use of illicit drugs. It was clearly impractical, however. The ability to treat drug users so that they will never take drugs again is also clearly limited, and letting drug injectors, their sexual partners, and their children die of HIV infection is clearly inhumane—and they have potential for spreading HIV into the rest of society.

Needle-exchange programs suggest the possibility of greatly reducing the individual and social harm associated with drug use through means other than simply reducing drug use or the drug supply. Making the distinction between reducing drug-related harm and reducing drug use per se is the fundamental premise of a new approach to drug policy that has been termed "harm reduction" or "harm minimization." Harm-reduction practices existed before HIV/AIDS and exchange programs and extend well beyond HIV/AIDS issues, but they have come to be recognized as a prototype of the harm-reduction approach in general.

The harm-reduction perspective itself is in a period of rapid development, so it is not possible to state its fundamental principles definitively, but there are at least four common assumptions in descriptions of the approach:

1. Pragmatism is valued over idealism. The nonmedical use of both licit and illicit psychoactive drugs is likely to continue indefinitely, so policies should be formulated on a realistic basis rather than on the basis of a utopian drug-free society.
2. Reducing drug use, particularly very heavy (dependent, addictive) drug use, is the most desirable but not the only means of reducing the individual and social harms associated with psychoactive drug use. Exchange programs to pre-

vent HIV infection are a clear example of reducing harm without necessarily reducing drug use. (Designated-driver programs are another example of harm reduction—reducing the harm associated with alcohol use without necessarily reducing alcohol use.)

3. In general, drug-related harm is likely to be reduced through integrating drug users into society rather than stigmatizing them and treating them as social outcasts.
4. While drug addiction clearly restricts an individual's ability to control his or her own behavior, drug users are still capable of making rational choices and should be offered choices among different ways of reducing the harm that drug misuse causes them and society.

The harm-reduction perspective is thus quite different from the war on drugs-zero tolerance perspective. Harm reduction is also distinct from the LEGALIZATION of all psychoactive drugs. The individual and social harms of drugs are not likely to be minimized by the mass marketing of drugs. NICOTINE/TOBACCO is a prime example of how large-scale harm has been created through uncontrolled merchandising of an addictive drug.

Rather than base policy on a utopian ideal of a drug-free society or the equally implausible ideal of a society that freely uses psychoactive drugs without problems, the harm-reduction perspective calls for basing policy on a flexible pragmatism. Specific harms associated with specific types of drug use can be identified, and concrete steps can be taken to reduce those specific harms. Exchange programs to reduce HIV infection among injecting drug users and their social contacts are a prototypical example of a concrete action for reducing drug-related harm. The message sent by exchange programs thus should not be read as "drug injecting is good" but rather that drug policies should be based on their pragmatic effects instead of on their symbolism.

(SEE ALSO: *Alcohol and AIDS; Complications: Route of Administration; Injecting Drug Users and HIV; Substance Abuse and AIDS*)

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NERVOUS SYSTEM DAMAGE *See* Brain Structures and Drugs; Complications: Neurological

NETHERLANDS, DRUG USE IN THE Since the 1970s, drug use in the Netherlands has been shaped by a national policy that the Dutch characterize as "harm reduction." To other nations, however, the policy has made the Netherlands the center of European drug use and drug trafficking. The continuing clash between the Dutch perception and the perception of outsiders shows no signs of abating. Dutch officials argue that heroin use is declining, while critics contend that Dutch youth have access to every type of drug.

After the explosion of drug use in the 1960s and 1970s, the Dutch government moved away from prohibition policies. The primary goal of the new policy was to reduce the harm that drugs caused to both the individual and to society. A corollary to this approach was that the efforts to control drugs should not cause more harm than the drugs themselves. The pursuit of this policy by the Netherlands has often resulted in bitter controversy with neighboring countries, which complain that the Dutch drug policy had undermined their own drug-control efforts.

The drug policy of the Netherlands has been characterized by two main principles—the separation of markets and the normalization of drug problems. The separation of markets principle is based on the idea that drugs can be classified pharmacologically according to their socially acceptable risks and that drug markets should be controlled on the basis of this classification. For example, in many societies ALCOHOL is a drug regarded to have acceptable risk, and the market for alcohol is legal for adults, with varying degrees of government regulation. The Dutch have decided that cannabis (MARIJUANA, HASHISH) is also a drug of acceptable risk and therefore should be separated from the markets for HEROIN and COCAINE, which have an unacceptable risk. Because of international regulations, however, the cannabis market cannot be equated with the alcohol market. Thus cannabis trafficking still remains illegal in the Netherlands, although it has a low law-enforcement priority in many jurisdictions. The so-called AHOJ-G policy for marketing cannabis requires limited advertising; no hard drugs—cocaine or heroin—are allowed to be sold or on the premises; no social nuisance; no youths under 16 years of age; only small amounts—less than 30 grams—can be sold. This policy regulates the system of cannabis-selling coffeehouses that have sprung up in most Dutch cities. Additional local regulations require that the coffeehouses provide recreational facilities, such as pool tables, so that something more than cannabis is offered to the customers.

The second main principle of the Netherlands drug policy is the normalization of drug problems. This principle recognizes that much of the harm attributed to the use of hard-drugs, such as heroin, is based on negative definitions that are held by

A man lights up a pipe at "Cannibis Castle" outside Nijmegen, the Netherlands, November 24, 1998. A marijuana user's mecca, the castle, owned by Sensi Seed Company, produces some of the most potent strains of the drug. (AP Photo/ Dusan Vranic)

society and internalized by the drug users. The principle of normalization leads to multiple efforts to reintegrate the heroin user into the community and to fight against his or her stigmatization. This is done by an extensive system of METHADONE MAINTENANCE PROGRAMS (a widely used pharmacotherapy for heroin users), counseling, and social-service support. In addition, drug users are encouraged to organize self-help groups and to mobilize for positive changes in their own subcultures, all in the interest of increasing both their participation in and their responsibility for the development of the drug-use context.

Although the Dutch maintain these policies on drug use, the laws against drug trafficking and the consumption of hard drugs are at least as tough as those of other European nations. However, the Netherlands had emerged in the late 1990s as the leading manufacturer of synthetic drugs such as ecstasy. Moreover, drug enforcement officials in Europe and the United States see the country as a drug supermarket, where smugglers are relatively free to move drugs across borders.

The Dutch government has argued that its policies are working. It cites evidence that the population of heroin addicts is stable and rapidly aging, suggesting that heroin is out of fashion with young people. However, critics note that between 1988 and 1997, heroin addicts treated at Dutch methadone programs increased from 6,500 to 9,800, an increase of 50 percent. In addition, the government points out that the mortality rate among drug users is low, due to effective methadone programs. The number of addicts infected with HIV is very low, which is attributed to methadone programs, needle-exchange programs and counseling. While the government acknowledged that marijuana use had gradually increased in the 1990s, the rate of cannabis use was lower than that of the United States.

Research continues to play an important role in reformulating the system of Dutch drug use. A number of universities, along with private and governmental institutions, conduct research in almost every area of drug use. In general, this research seems to show that the drug policy of the Netherlands has been functioning positively. For example, it seems that the goal of reducing the secondary effects of drug abuse (e.g., AIDS, VIOLENCE) is being reached. Studies of cocaine use in nondeviant social groups in Amsterdam and in the general population of Rotterdam provide evidence that patterns of use do not always lead to negative consequences, although it is difficult to say who can use without experiencing harm. A longitudinal study of heroin addicts indicates that the normalization policy has been effective in diverting the career of heroin addicts from criminal to conventional, but has been less effective in getting heroin users clean. Nevertheless, Dutch policies remain controversial.

(SEE ALSO: *Needle and Syringe Exchanges and HIV/AIDS; Sweden, Drug Use in*)

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NEUROLEPTIC Neuroleptic includes any of a group of drugs that are also called ANTIPSYCHOTICS. Neuroleptics are used as medications in the treatment of acute psychoses of unknown origin, including mania and SCHIZOPHRENIA. The prototype neuroleptic drugs are chlorpromazine (Thorazine), haloperidol (Haldol), clozapine (Clozaril), lithium (Lithonate), and thioridazine (Mellaril). Some of the newer drugs include risperidone (Risperdal), quetiapine (Seroquel), and olanzapine (Zyprexa). The site of action for these drugs (receptor site) is the central nervous system where they produce antipsychotic effects.

These drugs are also used for antianxiety, although other agents are more effective and do not have the long-term side effects that neuroleptics do. Drug therapy alone is not entirely effective in treating psychoses, and it is used in combination with acute and long-term support and medical care. Some neuroleptics are also used in the treatment of nausea, vomiting, alcoholic hallucinosis, neuropsychiatric diseases marked by movement disorders (e.g., Huntington's disease and Gilles de la Tourette's syndrome), pruritus, and intractable hiccough.

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NEURON The gross anatomy of the central nervous system—the brain and spinal cord—was studied in some detail during the seventeenth and eighteenth centuries, but not until the nineteenth century did scientists begin to appreciate that the central nervous system (CNS) was composed of many millions of separate cells, the neurons (also called nerve cells). This discovery had to await technical improvements in the microscope and the development of specialized stains that permitted scientists to observe the microscopic anatomy of the nervous system.

HISTORY

In the 1870s, the Italian anatomist Camillo Golgi developed such a special staining technique, and he and other scientists were then able to observe, under the microscope, the fine structures of the cells of the nervous system. Yet Golgi may not have fully appreciated that what seemed to be an extended network of nerve tissue, in reality, were millions of distinct neurons with fine fibrils touching each other. It was the Spanish scientist, Santiago Ramón y Cajal, who was credited with expounding the neuron theory. In 1906, Golgi and Ramón y Cajal shared the Nobel prize in physiology/medicine for their discoveries on the nature of the nervous system.

Even after the concept of separate neurons was generally accepted, there was controversy for many years about how the separate neurons communicated with each other. At the end of the nineteenth century, many scientists believed they did so by means of electric impulses. Others believed there was a chemical messenger that allowed neurons to influence each other. Around 1920, ACETYLCHOLINE was discovered, the first of many nerve messengers that would be discovered during the subsequent decades.

FUNCTION

The neuron is the basic functional cellular unit of nervous system operations; it is the principal investigational target of research into the actions of addictive drugs and ALCOHOL. An essential feature of the cellular composition of the brain is the high density of extremely varied, heterogeneously shaped neuron groups (see Figure 1). To understand the specialized aspects of neurons and their

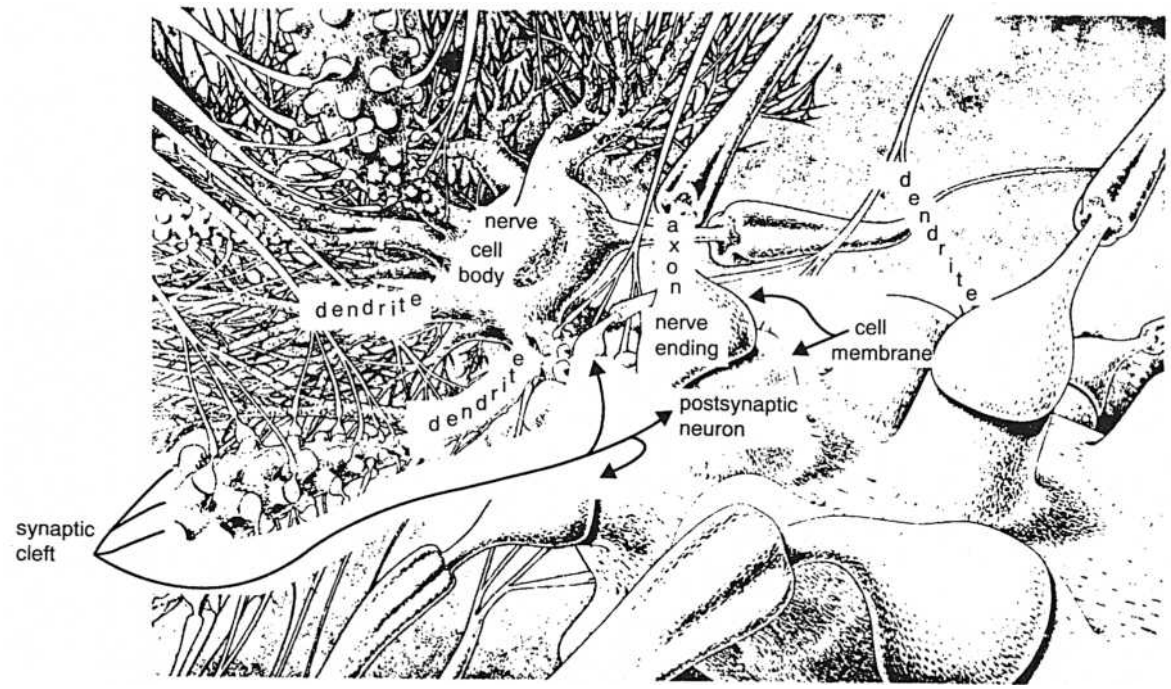

Figure 1
Neuronal Complexity. The complexity of the neuronal network in the brain is demonstrated by this bundle of neurons, which form a vast and ramified structure with their cell bodies, outgrowths, and intercellular contact points.
SOURCE: Modified from Figure 1, in M. J. Kuhar's Introduction to Neurotransmitters and Neuroreceptors, in *Quantitative Imaging*, edited by J. J. Frost and H. N. Wagner. Raven Press, New York, 1990.

function, therefore, requires a discussion of the general structural and functional features characteristic to all neurons and the degree to which unique variations form consistent subsets of neurons.

Neurons share many cellular properties that distinguish them significantly from other cell types in other tissues; those changes within the cell's regulatory processes of greatest interest to researchers of addictive drugs, however, depend on features that form distinctions within the class of cells called neurons. Furthermore, the assembly of individual neurons into functional systems, through highly precise circuitry employing highly specified forms of chemical interneuronal transmission, allows for the sensitivity of a brain to addictive drugs.

In some organs of the body—such as the liver, kidney, or muscle—each cell of the tissue is generally similar in shape and function. Within that tissue, all perform in highly redundant fashion to convert their incoming raw material into, respectively, nutrients, urine, or contractions, which establishes the function of the specific tissue. In the nervous system, the variously (heterogeneously) shaped neurons (see Figure 2), supported by an even larger class of similarly (homogeneously) shaped non-neuronal cells, termed *neuroglia*, convert information from external, or from internal, sources into information ultimately integrated into programs for the initiation and regulation of behavior.

This integrative conversion of sensory information into behavioral programs results from the rich interconnections between neurons, and it depends on the extremely differentiated features of neurons—their size and shape; their extended cell-surface cytoplasmic processes (dendrites and axons); and their resultant interconnections that establish the sources of their incoming (afferent) information

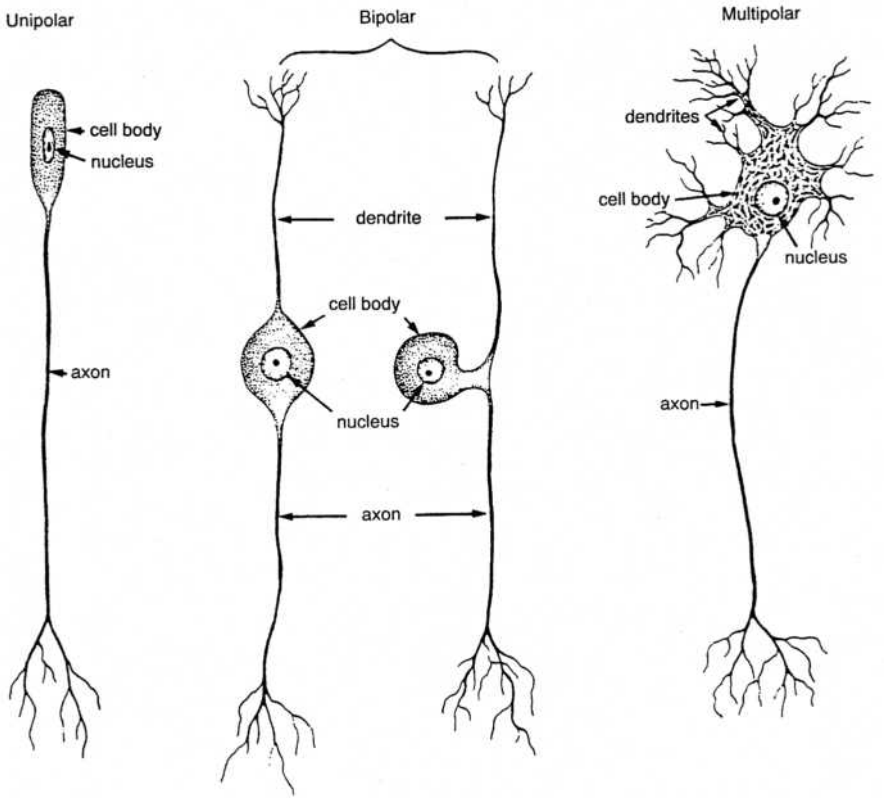

Figure 2
Three Types of Neurons

and the targets of their outgoing (efferent) communication (see also Figure 4).

COMMON FEATURES

As cells, neurons share some features in common with cells in all other organ systems (see Figure 3). They have a *plasma membrane* acting as an external cell wall to form a distinct boundary between the environment inside (intracellular) and outside (extracellular) the cells. The intracellular material enclosed by the plasma membrane is termed the *cytoplasm*. Like all other cells (except red blood cells), neurons have numerous specialized intracellular organelles, which permit them to maintain their vitality while performing their specialized functions.

Thus, neurons have *mitochondria* (singular, mitochondrion), by which they convert sugar and oxygen into intracellular energy molecules, which then fuel other metabolic reactions. Neurons have abundant *microtubules*, thin intracellular tubular struts, by which they form and maintain their often highly irregular cell structure. Neurons are also rich in a network of intracellular membranous channels, the *endoplasmic reticulum*, through which they distribute the energy molecules, membrane components, and other synthesized products required for functioning. Like other cells that must secrete some of their synthesized products for functioning, as neurons do with their neurotransmitters, some parts of the endoplasmic reticulum, the *smooth endoplasmic reticulum*, are specialized for the packaging of secretion products into storage particles, which in neurons are termed *synaptic vesicles*. At the center of the pool of cell material, the *cytoplasm*, neurons possess a *nucleus*, which, as in other nucleated cells, contains the full array of the genetic information characteristic of the individual organism. From this nucleus, selected subsets of genetic information are expressed to provide for the general shared and the specific unshared features of the cell. The nucleus of the neuron cell is enclosed within a membranous envelope that, as in

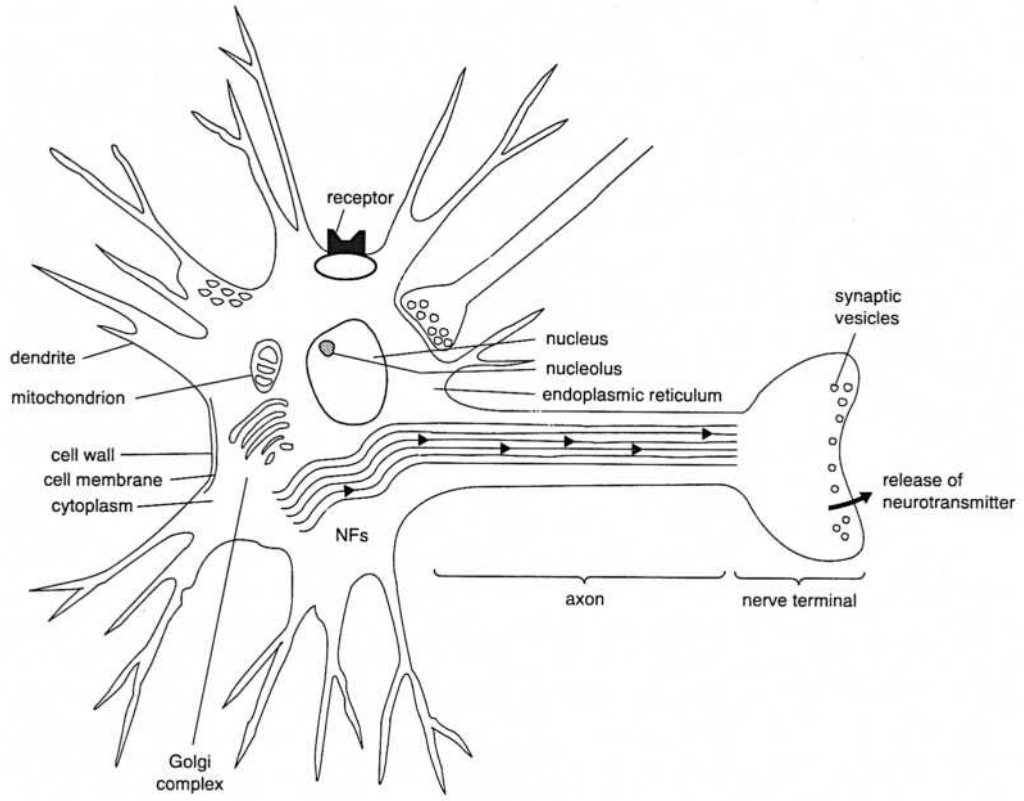

Figure 3
Features of the Neuron

many other types of cells, exhibits multiple nuclear pores through which information can be conveyed to and from the nucleus.

UNIQUE FEATURES

The plasma membrane of neurons differs from that of non-neuronal cells in that it contains special proteins, termed *voltage-sensitive ion channels*. Such channels are conceptually small tubular proteins embedded in the membrane of the neuron, which, when activated under specific conditions, allow positively charged ions of sodium, potassium, and calcium to enter the neuron. The existence of such electrically sensitive channels permits the neuron to become electrically excitable. The expression and selective distribution (compartmentalization) of such electrically excitable channels along its efferent processes, the axons, permit neurons to conduct signals efficiently for long distances; this also accounts for the bioelectrical activity of the brain assessed by *electroencephalography (EEG)*. Similarly, the distribution of such electrically excitable ion channels along the receptive surfaces of the neurons (its dendrites and cell body [soma]) allows them to conduct and integrate signals from all over the extended shape of the neuron.

The smooth endoplasmic reticulum of the neuron is somewhat more elaborate and extensive than other cells that secrete their products; this specialized and extensive smooth endoplasmic system is termed the *Golgi complex* (or *Golgi apparatus*). Discovered accidentally, it was a useful marker for staining the nervous system to distinguish neurons from other cells of the brain when under inspection by microscope.

The nucleus of neurons is often highly elaborated, with multiple creases or infoldings, exhibiting complex configurations, within which are typically dense accumulations of cytoplasmic organelles, and almost always a very distinctive intranuclear clustering of genetic material, the *nucleolus*. Differentiated neurons—neurons whose developmental stage is past the step at which cell-

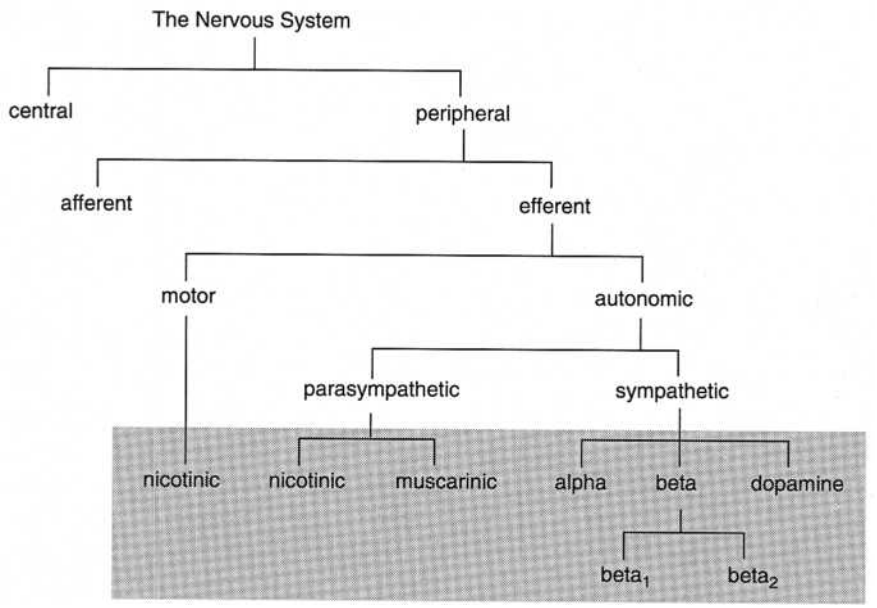

Figure 4
Relationship of Receptor Types. Efferent nerves in the peripheral nervous system. (Receptor subdivisions for alpha and dopaminergic receptors are not included.)

type dedication has occurred—are unable to undergo cell division, in distinct contrast to comparably metabolically active cells in such complex tissues as liver, kidney, muscle, or skin. As a result, mature neurons can repair themselves, up to a point, but are unable to regenerate themselves or respond to their growth factors in a manner that would in other tissues lead to cell division and replacement.

The most distinctive cellular feature of neurons is the degree to which they express unique patterns of size and shape. In mammals, all neurons have highly irregular shapes; such shape variations are categorized in terms of the number of cell surface extensions, or neuronal processes, that the neuronal subset expresses, as in Figure 2.

Some neurons have only one cellular process extending from the surface of a round or nearly round cell body; this form of neuron, a *unipolar* neuron, is typical of invertebrate nervous systems. Typical unipolar neurons are the cells of the dorsal root ganglia, in which a single efferent axon conducts information toward or away from the cell body through a branched axon.

Most neurons of the central nervous system of mammals are multipolar. That is, in addition to the efferent axon, which may also have many subsets of secondary axons, called *collateral branches*, that stem from the main efferent process axon, elaborations may also be expressed from the cell body surface. The latter elaborations are termed *dendrites*, because their shape resembles the limbs of trees. Dendrites protrude from the cell body, and they, as well as the cell body, constitute the receptive surfaces of the target neuron onto which the afferent connections make their synaptic connections.

DISTINGUISHING NEURONS

Since neurons come in so many shapes and sizes, early investigators of the brain sought to make distinctions among them, based in part on their locations, their sizes and shapes, and the connections they could be shown to receive or emit. Every scientist who worked in the formative era of brain research sought to describe a unique subset of neurons that were forever after named for their initial describer or the unique property defined. Thus, we have *Betz neurons*, large layer V-VI neurons of the motor cortex, and *Purkinje neurons*, the major output neurons of the cerebellar cortex, as well as neurons named for their shapes and appearance—*pyramidal neurons* of the cerebral and hippocampal cortices, *mitral* and *tufted neurons* of the olfactory bulb, and *granule cell neurons* of the cerebellar, hippocampal, and olfactory cortices. The last mentioned have relatively compact cell bodies, densely packed together, giving the brain a granular appearance by optical microscopy.

Dendrites and axons exhibit highly distinctive morphological patterns. The surfaces of dendrites and axons can be distinctive in the shapes of their branches. This permits fine discrimination among neurons (stellar, or star-shaped, neurons; chandelier neurons; or mossy or climbing axon fibers). Some neurons exhibit dendrites whose surfaces are smooth (aspiny); others are highly elaborated (spiny), which may serve to enlarge the receptive surfaces and enhance the degree to which such neurons may integrate afferent information.

Similarly, the morphology and stability of the axons may also be highly variable. Some neurons direct their axons to highly constrained targets in a more or less direct route; others may be highly branched, with multiple collateral branches to integrate communications from one cell cluster to many divergent targets. To provide the essential support of anabolic and secretory materials within these highly elaborated cellular structures, neurons have evolved an efficient form of intracellular transport, an energy-dependent, microtubule-guided, centripetal and centrifugal process by which organelles are dispensed to and returned from the distal processes (as well as probable macromolecular signals sensed by pinocytotic-like [fluid uptake] incorporation of such signals by distal dendrites and axons). Such signals may serve as local growth-regulatory factors, allowing even the nondividing neurons to alter their shape and connections in response to activity and signals received from their afferent sources.

NEURONAL IDENTITY

An individual neuron may be referred to on the basis of its size (magnocellular, parvicellular). A layer or "nuclear" cluster of neurons may be referred to by shape (pyramidal, mitral), the morphology of its axon terminals (i.e., *basket cells*, whose axon terminals make basket-shaped terminations on their targets), and its position in a sensory or motor circuit. In the latter classification scheme, those neurons closest to the incoming sensory event or to the outgoing motor-control event are termed *primary sensory* or *motor* neurons, respectively, whereas neurons at more distal positions of circuitry from the primary incoming or outgoing event are termed *secondary*, *tertiary*, and so on, depending on their position in that hierarchy.

In addition to these morphological qualities, neurons may also be separately distinguished on the basis of the functional systems to which they are connected (visual, auditory, somatosensory, proprioceptive, attentional, reinforcing, etc.) and on the basis of the neurotransmitters they employ to communicate with the neurons to which they are connected (cholinergic, adrenergic, GABA-ergic, etc.). Each of those features provides for a multidimensional definition of virtually every neuron in the brain.

(SEE ALSO: *Brain Structures and Drugs; Neurotransmission; Neurotransmitters; Receptor; Drug; Reward Pathways and Drugs*)

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FLOYD BLOOM

NEUROTRANSMISSION NEURONS (nerve cells) communicate chemically by releasing and responding to a wide range of chemical substances, referred to in the aggregate as NEUROTRANSMITTERS. The process of *neurotransmission* refers to this form of chemical communication between cells of the central and peripheral nervous system at the anatomically specialized point of transmission, the SYNAPSE (synaptic junctions). Thus, it is convenient to conceive of "the" neurotransmitter for a specific instance of synaptic connections between neurons in one brain location (the source neurons) and their synaptic partner cells (the target neurons) in another neuronal location. For example, the phrase "dopaminergic neurons of the nigro-accumbens circuit" refers to the DOPAMINE-transmitting synaptic connections between the brain neurons of the substantia nigra and their targets in the NUCLEUS ACCUMBENS. Current concepts of neurotransmission, however, require a broader view; they would consider as neurotransmitters all the chemical substances that a given neuron employs to signal the other neurons to which it is anatomically connected (its synaptic targets) and through which that neuron may also be able to influence other neuronal and nonneuronal cells in the adjacent

spatial environment of its circuitry (nonsynaptic targets).

In some cases—more frequent in invertebrate nervous systems, in more primitive vertebrates, and in the embryonic nervous system than in the adult mammalian nervous system—neurons may also communicate "electrically," by direct ionic coupling between connected cells, through specialized forms of intercellular junctions referred to as "gap junctions," or *electrotonic junctions*. Such electrotonic transmission sites are of relatively little direct concern to the actions of addictive drugs and ALCOHOL. In contrast, it is the more pervasive process of chemical neurotransmission that underlies the main molecular and cellular mechanisms by which addictive drugs act—and through which the nervous system exposed to such drugs undergoes the adaptations that may lead to DEPENDENCE, HABITUATION, WITHDRAWAL, and the more enduring changes that persist after withdrawal from the once-dependent state.

The critical characteristic of a substance designated as a neurotransmitter is the manner in which it is made and secreted. To qualify as a neurotransmitter, the release of the substance must be coupled to neuronal activity according to two rather stringent functional rules (see Figure 1).

1. The transmitter substance must be synthesized by the transmitting neuron. In most cases, the substance is made well in advance and stored in small organelles (synaptic vesicles) within the terminal axons of the source neuron, ready for eventual release when called upon.
2. The transmitter substance must be released by that neuron through a special form of activity-dependent, calcium ion (Ca^{2+})-selective, excitation-secretion coupling. Substances released through other nonactivity-coupled and non-Ca^{2+}-coupled mechanisms may be regarded as excretion (as with metabolic byproducts to be degraded), rather than secretion.

The synaptic junction is the site at which the axons of the source neuron physically make most intimate contact with the target neuron to form an anatomically specialized junction; concentrated there are the proteins that mediate the processes of transmitter release (from the presynaptic neuron) and response (by the postsynaptic neuron). Indirect evidence for some neurotransmitter systems has suggested to some scientists a general concept of *nonsynaptic* interneuronal communication, sometimes also referred to as *paracrine* or *volume-transmission* communication, in which the neurotransmitter released by a designated set of presynaptic terminals may diffuse to receptive neurons that are not in anatomic contact. The sets of chemical substances that neurons can secrete when they are active can also influence the non-neuronal cells, such as the cells of the vascular system (the glia) and the inflammatory-immune cells (the microglia).

The activity of neurons can also be modified by substances released from the non-neuronal cells of the central or peripheral nervous system, substances often termed *neuromodulators*. This same term, however, is frequently applied to the effects of neuron-produced transmitter substances whose mechanisms of action and whose time course of effect differ from those of the classic junctional neurotransmitter acetylcholine.

The current research on neurotransmitters and neuromodulators pertinent to drugs and alcohol is devoted to (1) understanding how exposure to addictive drugs may regulate the genes that control the synthesis, storage, release, and metabolism of known neurotransmitters; (2) identifying new substances that may be recognized as neurotransmitters, whose effects may be related to the effects of or reactions to addictive drugs and alcohol; (3) understanding the molecular events by which neurons and other cells react to neurotransmitters in both short-term and long-term time frames (a process often termed *signal transduction*, which cells of the nervous system share with most other cells of the body) and how these processes may themselves be perturbed by the influence of addictive drugs and alcohol; and (4) understanding the operations of neuronal communication in an integrative context of the circuits that release and respond to specific transmitters, and the way in which these neuronal circuits participate in defined types of behavior, either normal or abnormal.

NEUROTRANSMITTER ORGANIZATION

There are three major chemical classes of neurotransmitters.

1. *Amino acid transmitters*: glutamate (GLU) and aspartate are recognized as the major excitatory

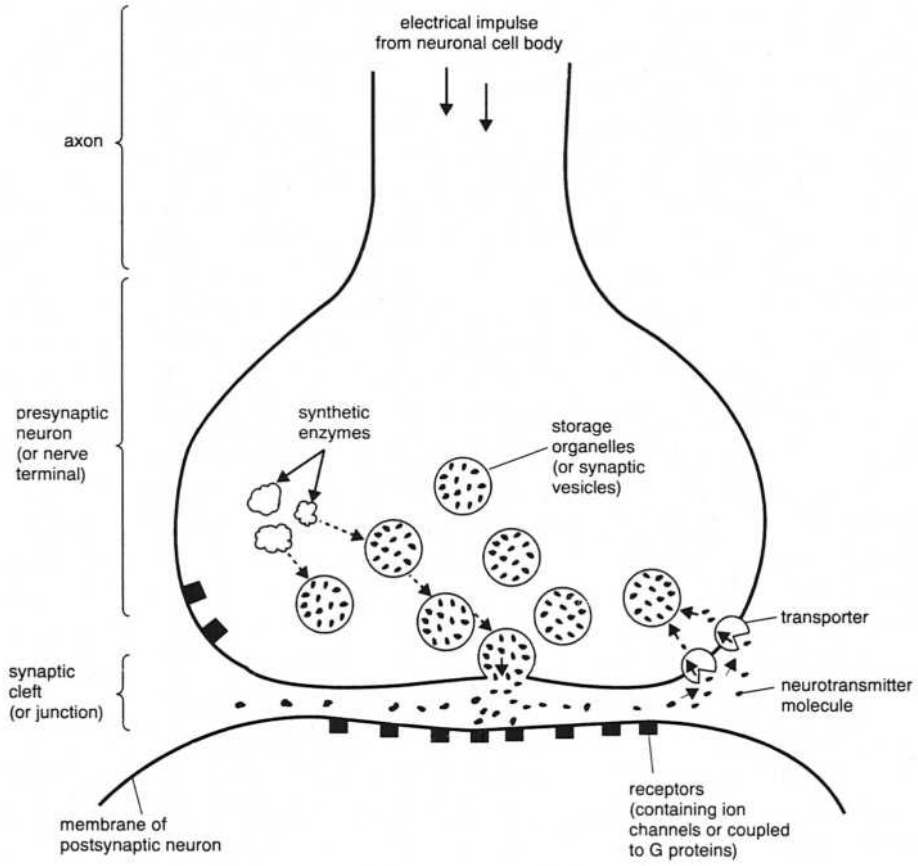

Figure 1

Synapse. Nerve ending from one neuron forms a junction, the synapse, with another neuron (the postsynaptic neuron). The synaptic junction is actually a small space, sometimes called the synaptic cleft. Neurotransmitter molecules are synthesized by enzymes in the nerve terminal, stored in vesicles, and released into the synaptic cleft when an eletrical impulse invades the nerve terminal. The electrical impulse originates in the neuronal cell body and travels down the axon. The released neurotransmitter combines with receptors on postsynaptic neurons, which are then activated. To terminate neurotransmission, transporters remove the neurotransmitter back into the nerve terminal that released it.

transmitting signals; GAMMA AMINOBUTYRATE (GABA) and glycine are the major inhibitory transmitters. These transmitter substances occur in concentrations of one millionth part per milligram (μM/mg) protein. Since they are considered the most frequently employed transmitter substances, they have been linked to many aspects of the actions of addictive drugs.

2. *Aminergic transmitters*: ACETYLCHOLINE, epinephrine (also called adrenaline), NOREPINEPHRINE (also called noradrenaline), DOPAMINE, SEROTONIN, and histamine. The aminergic neurons constitute a minor population of neuronal transmission sites, as reflected in the fact that their concentrations in the brain are roughly 1/1000th that of the amino acid transmitters or one billionth part per milligram (nM/mg protein). Because of their divergent anatomy (a few clusters of aminergic neurons may project onto literally millions of target neurons in many locations of the brain) and the ability of their synaptic signals to produce long-lasting effects, the aminergic neurons represent a very powerful subset of transmission conditions that is important to the effects of addictive drugs. Of particular relevance are the dopaminergic neurons—for their pertinence to the sites of reward for stimulants, opiates, and certain aspects of etha-

nol (alcohol) action—and the noradrenergic and serotonergic neurons—for their association with the phenomena of drug adaptation and tolerance.

3. *Neuropeptides*: of which there are dozens. Peptides are molecules containing a specific series of 2–50 amino acids, chemically arranged in a specialized "head-to-toe" chemical linkage known as a peptide bond. The order and number of the linked amino acids determine the linear structure of the peptide. In the nervous system, peptides, in general, occur in still lower concentrations than do the two prior classes of transmitter, namely at 10–100 trillionth part per milligram (pM/mg) protein. A revolutionary finding has emerged here in concepts of brain system interactions: It would now seem that neuropeptides are almost certainly never the sole signal to be secreted by a central neuron that contains such a signaling molecule, but rather accompany either an amino acid or an amine transmitter (at intrasynaptic terminal concentrations a thousand to a millionfold higher), such sites may even contain a second or third peptide as well.

Neuropeptides are of interest to the molecular and cellular mechanisms of addictive drug and alcohol action, because they may provide the postsynaptic receptors through which the drugs act (as in the case of the opiates and possibly the case for the natural BENZODIAZEPINES) or modify the effects of the presynaptic transmitters (as in the case of the peptide cholecystokinin that accompanies some forms of dopaminergic transmission, through which stimulants act and may modify responses to that amine if cosecreted).

Because of the ability to read the linear sequences of the amino acids, it has become clear that many of the neuropeptides share select small sequences and thus conceptually constitute "families" of peptides. For example, the opioid peptides all share one or more repeats of the amino-acid sequence tyrosine-glycine-glycine-phenylalanine; thus, each of the opioid-peptide genes leads to the expression of a different pre-prohormone by different sets of neurons of the central and peripheral nervous system. The existence of the shared amino-acid sequences implies that at some point in evolution, there may have been only one opioid-peptide signal, which was then duplicated and modified for use by the increasing number of neurons that came with the evolution of the mammalian brain. Such family relationships also exist for other peptide families (oxytocin/vasopressin; the tachykinin peptides; the secretin/glucagon-related peptides; the pancreatic polypeptide-related peptides), whose amino-acid sequences have shown great conservation over large domains of the evolutionary tree, attesting to the high signal quality of these molecules and the transductive mechanisms of their receptors. Other peptides, such as somatostatin and gonadotropin-releasing hormone, have no known family relationships as yet—but the discovery process here is probably not complete.

OTHER TRANSMITTER CANDIDATES

Other kinds of molecules may also be made within neurons to play auxiliary roles in intercellular transmission in the nervous system—from purines like Adenosine Triphosphate, lipids like arachidonic acid and prostaglandins, and steroids similar to those made and released by the adrenal cortex and the gonads. These substances may, in some cases, act as intracellular second messengers to underlie the effects of the aminergic and peptidergic transmitters (see below); they therefore have implicit relevance to the effects of the addictive drugs whether or not they may also serve as primary transmission signals.

Investigators have revealed that under some conditions active neurons may synthesize gaseous signals, such as nitric oxide and carbon monoxide, which can carry rapidly evanescent signals over short distances. The effects of these transmission-related substances will undoubtedly become of increasing importance to the explanations of the mechanisms of action or adaptation to the addictive drugs.

SIGNAL TRANSDUCTION ORGANIZATION

Aside from the chemistry of the neurotransmitter substances, further insight into their role in the actions of addictive drugs arises from the viewpoint of their synaptic physiology and their underlying mechanisms of signal transduction. When neurons respond to neurotransmitters, the ultimate changes in the excitability and metabolic activity of the

responding neuron generally require changes into or out of the cell in the flow of ions (natural chemical elements of the extracellular fluid)—some with positive charge (sodium, potassium, and calcium) and others with negative charge (chloride).

As a general rule, it would appear that every neurotransmitter has more than one form of postsynaptic receptor through which its effects are mediated. Before the ability to characterize these receptors through molecular genetics, such receptor subtypes were identified on the basis of the comparative pharmacological potency of synthetic AGONISTS or ANTAGONISTS of the natural transmitter. With the advent of molecular cloning, however, an even finer subtyping would appear to be required, since many of the conclusions on receptor pharmacological patterns were based on analyses of tissue fractions that undoubtedly contained many molecular forms. A major effort in the future will be to link more explicitly the molecular and pharmacological characterization of neurotransmitter receptor subtypes and to determine which of them are most critical to the effects of, and adaptations to, addictive drugs.

Three major formats have been revealed for the transductive process.

1. *Directly regulated ion channels*. Here the ion channel to be opened is formed by the units of the receptor molecule itself, as recently established by direct cloning of several such receptor-ionophores. Such receptors are now known to be the motif of the nicotinic-cholinergic receptors of the neuromuscular junction and the central nervous system, as well as for the three types of glutamate receptor, the several isoforms of the $GABA_A$ receptor, the glycine receptor, and at least one form of a serotonin receptor.

   Common features of these receptors are (a) they are composed of several (3–5) subunits, called monomers, that apparently may be combined in differing ratios (so-called multimeric recombinations) by various neurons to constitute the "holoreceptor"; (b) each monomer consists of four presumed transmembrane domains; and (c) discrete sections of the receptor monomer, either within the membrane or the cytoplasm, account for their voltage and chemical sensitivity, and for the ease and duration of openings in the ion channel.

2. *Indirectly regulated ion channel-receptors*. This form is based on the similarities between the visual pigment rhodopsin—the molecule used by photoreceptor neurons (rods, cones) to transduce light into signals to other neurons of the retina—and the beta-adrenergic receptor—one of the types of receptors regulated by the amine norepinephrine. This general form of transducing molecule was later found to be the form also used by the cholinergic muscarinic receptor, as well as by most serotonin and all known dopamine receptors, plus all the known peptide receptors.

   The common features of this class are (a) the receptor is a single molecule, with seven transmembrane domains; (b) activation of these receptors by their signaling molecules leads to further interactions of the receptor with other large proteins, some of them enzymes, within or near the plane of the membrane; and (c) the eventual indirect regulation of the ion channel, either the opening or closing of the channel, is then mediated through small molecular intracellular second messengers, such as the calcium ion (Ca^{2+}) or the products of the associated enzymes, yielding intracellular second-messenger molecules, such as cyclic adenosine monophosphate (cAMP), or a lipid such as an inositol phosphate, diacyl-glycerol, or an arachadonic acid catabolite. The essential common second step of such transduction cascades is that the activated receptor interacts with a guanosine triphosphate (GTP)-binding protein (termed a *G-protein*) composed of three monomer subunits. The G-protein complex dissociates to activate the enzyme making the second messenger and, at the same time, hydrolyses the GTP and reassociates to end the cycle of signal generation. The second messenger consequences of this form of transduction, however, may be more enduring—activating one or more enzymes (protein kinases or phosphatases) that can add or remove phosphate groups on structural proteins or other enzymes, to activate or inactivate them. Such events can significantly shift the metabolic state of the responding cell and eventually regulate the expression of its specific genes. One such gene target is the immediate early genes of the nervous system, the proto-oncogenes, discovered some years ago because

of the mutated forms used by oncogenic viruses, which induce cancer in non-neuronal cells.

3. *The receptor-enzyme*. This third major molecular motif of signal transduction has been elucidated recently; although it is already clear that this form does exist in the mammalian brain, it has been studied more in non-neuronal systems. This motif's characteristics are that the receptor for some peptides is itself the enzyme guanylate cyclase, which is directly activated by receptor-ligand binding, leads to an intracellular generation of cyclic guanosine monophosphate, and then to a cascade of events similar to that described for AMP.

SYNAPTIC INTERACTIONS

Most neurons receive synaptic input simultaneously from hundreds of other neurons, each of which employs its own mix of transmitters. The transductive processes underlying these individual events can influence the intensity and duration of the subsequent responses, thereby integrating incoming signals and providing the basis by which activity in assemblies of interconnected neurons results in behavioral output by the brain.

To gain insight into the basis by which the events of neurotransmission can lead to multineuronal programs of interaction, such as those required to initiate responding for an addictive drug, requires knowledge both of the anatomical substrate over which such programs of neuronal activity take place and of the effects of the neurotransmitters at each of the cellular elements of such an interactive ensemble of neurons.

(SEE ALSO: *Addiction: Concepts and Definitions; Brain Structures and Drugs; Limbic System; Tolerance and Physical Dependence*)

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FLOYD BLOOM

NEUROTRANSMITTERS A neurotransmitter is any chemical substance (the first recognized was ACETYLCHOLINE) that NEURONS (nerve cells) secrete to communicate with their target cells (glands, muscles, and other neurons). Neurotransmitters diffuse from their sites of release—from the presynaptic nerve terminal—across the synaptic cleft, to bind to receptors on the external surface of the postsynaptic cell. Activation of these receptors allows for the transmission of commands (excitation, inhibition, and other more complex forms of regulation) from the presynaptic neuron to the postsynaptic cell.

A neurotransmitter is released from a nerve ending, interacts with specific RECEPTORS, and is then either transported back into the presynaptic neuron or destroyed by metabolic enzymes in the synaptic cleft.

Chemically, neurotransmitters are amino acids, amines, or peptides. Peptide transmitters commonly coexist and may be cosecreted with amino acid or amine transmitters.

(SEE ALSO: *Dopamine; Endorphins; Neurotransmission; Norepinephrine; Serotonin*)

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FLOYD BLOOM

NEW YORK STATE CIVIL COMMITMENT PROGRAM The New York State Civil Commitment Program was the largest and most expensive drug treatment program of its kind during the 1960s and 1970s. Modeled after the CALIFORNIA CIVIL Addict Program (CAP), it was established in the early 1960s in response to the dramatic rise of New York's heroin-addict population. The first reaction to the problem was expressed in the Metcalf-Volker Narcotic Addict Commitment Act of 1962, which sent arrested addicts to state mental-hygiene facilities for treatment. The total failure of this program prompted New York Governor Nelson Rockefeller to substantially modify and expand the program in 1966 by creating a Narcotic Addiction Control Commission (NACC). NACC was established to administer the New York State Civil Commitment Program, which involved a major statewide network of residential treatment centers.

Six different types of centers handled the following phases of treatment: examination and detention; detoxification, orientation, and screening; residential treatment and rehabilitation; temporary return; indefinite return; and halfway houses. Those who were eligible for treatment at a center included addicted individuals who had been arrested or convicted for a felony or misdemeanor, who had been involuntarily committed by their family or a friend, or who had volunteered to be treated. The treatment process consisted of a period of commitment within the institution, followed by community aftercare. Clients were under the control of the agency for an average of twenty-five months, of which ten months was spent in residence at the institution (Winick, 1988).

THE PROGRAM'S DEMISE

The program reached its peak in 1970 when twenty-four state facilities with 4,100 beds and a staff of over 5,000 provided services to 6,600 addicts. Followup studies of the program at this time were few, but they tended to indicate some positive outcomes (Winick, 1988). After 1970, the program began to lose public support and became a regular political target because of charges of cost overruns, allegations of staff brutality, and questionable administrative procedures (Winick, 1988). There was also a general change in philosophy that drew politicians away from supporting state-run institutions and toward recommending community-based treatment. In addition, political leaders began to move away from rehabilitation and toward harsh criminal sanctions for persons possessing or selling narcotics.

Governor Rockefeller announced in 1971 that he had lost confidence in the New York program and initiated a two-thirds cutback in budget and clients. The number of occupied beds steadily diminished because of these cuts and by 1979 the last two centers shut down(Winick, 1988). From 1966 to 1979, the program had cost approximately $1 billion. By the time the program was closed, each resident was costing an average of $29,000 per year, as compared with $8,500 for a resident in a THERAPEUTIC COMMUNITY and $14,500 for a prison inmate (Winick, 1988). In 1980, the state legislature repealed the civil commitment law.

WHY THE PROGRAM FAILED

Poor planning played a major part in the failure of the program (Winick, 1988). Due to political pressure, the first eight facilities opened in less than a year. Staffing was an immediate problem. The directors of the treatment facilities had inadequate administrative or clinical experience, since they were mostly political and civil service appointees (Inciardi, 1988). Facilities also were ill chosen and they too contributed to staffing deficiencies. NACC purchased underused prisons from the New York Department of Corrections and used them as treatment facilities. Many of the former prison guards were maintained as rehabilitation officers who performed both a counseling and custodial function. These officers were inadequately trained for their new positions, and they often disciplined program participants too harshly (Inciardi, 1988). The result was an environment that did not offer therapeutic benefits and was not conducive to behavioral change.

The screening of candidates for the program, moreover, was not consistent, and the criteria for completion of the program were ambiguous. The reentry and aftercare programs were equally ill

equipped to handle the task at hand. The aftercare "officers" had no authority to arrest a client for violation of aftercare conditions, and their caseloads were too large to allow close supervision. As a consequence, a great number of parolees fled or stopped reporting (Winick, 1988).

Apart from programmatic failings, the civil commitment program began just as political leaders started to move away from rehabilitative models. Governor Rockefeller provides a telling example. By the early 1970s, when heroin addiction showed no signs of abating, Rockefeller decided that the criminal justice system should be directed more forcefully at drug users. In 1973, a group of statutes, popularly known as the Rockefeller laws, went into effect. These laws imposed mandatory prison sentences on those that possessed or sold drugs. These sentences, even for first-time offenders, were very long. Repeat offenders could receive life imprisonment. With the Civil Commitment Program unable to produce reliable and cost-effective results, the impulse to incarcerate drug users proved almost irresistible.

(SEE ALSO: *California Civil Commitment Program; Civil Commitment; Coerced Treatment for Substance Offenders; Narcotic Addict Rehabilitation Act; Prisons and Jails; Rockefeller Drug Laws*)

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NIAAA *See* U.S. Government Agencies: National Institute on Alcoholism and Alcohol Abuse

NICOTINE This is a PSYCHOACTIVE chemical substance found in TOBACCO products, including cigarettes, cigars, pipe tobacco, and smokeless tobacco such as chewing (spit) tobacco and oral and nasal SNUFF. The nicotine molecule is composed of a pyridine ring (a 6-membered nitrogen-containing ring) with a pyrrolidine ring (a 5-membered nitrogen-containing ring).

Nicotine can occur in two forms. The active form, called L-nicotine, is found in tobacco plants of the genus Nicotiana. These are chiefly South American plants of the nightshade family (Solanaceae)—annuals cultivated since pre-Columbian times for their leaves, especially *Nicotiana tabacum*. The inactive form, D-nicotine, is not present in tobacco leaves but is formed, to a small extent, in the combustion of tobacco during smoking. These two forms are stereoisomers, meaning that even though they are both nicotine, they have different three-dimensional structures. In pure form, nicotine is a colorless liquid, but it turns brown on exposure to air.

Nicotine is water-soluble and transfers from tobacco to cigarette smoke readily, because it vaporizes easily. Once it is in the body, conditions are ideal for rapid distribution to blood and tissues because nicotine is a weak base, and when un-ionized under alkaline conditions, such as those found in the blood stream, it crosses cell membranes easily.

The primary natural source of nicotine is the tobacco plant, but nicotine is also found in some amount in related plants. Small amounts are in foods of the nightshade family, such as tomatoes and eggplants. Consumption of nicotine has not been limited to the use of plants in which it naturally occurs. In 1828, the German scientists Posselt and Reiman isolated nicotine from tobacco leaves, and since then it has been added to other products. For example, it is widely used as an insecticide in such products as Black Leaf 40, which contains 40 percent nicotine sulfate.

EFFECTS OF NICOTINE

The first pharmacological studies of nicotine were initiated in 1843 by Orfila. Nicotine is an

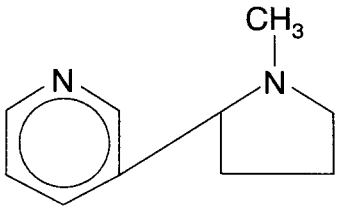

Figure 1
Nicotine

alkaloid that affects major organs, such as the heart and brain. It also affects the body at the cellular level.

Effects in the Body. The actions of nicotine in a human body are complex. They depend on the amount of nicotine given, the route of administration (e.g., by mouth or intravenously), the time over which the dose is given, and the individual's history of exposure to nicotine. In high doses, nicotine produces nausea, vomiting, convulsions, muscle paralysis, cessation of breathing, coma, and circulatory collapse. Such high doses are seen after accidental absorption of a nicotine-containing insecticide or an overdose of nicotine.

In lower doses, such as those used by people who consume tobacco products, the effects are very different. They include a speed up in heart rate and blood pressure; increased force of contraction of the heart; constriction of blood vessels in the skin, producing cool, pale skin; constriction of blood vessels in the heart; relaxation of skeletal muscles; increased body metabolic rate; and the release of hormones such as epinephrine (adrenaline), NOREPINEPHRINE, and cortisol into the bloodstream. Nicotine's effects on the brain are very complex because nicotine works in part by enhancing the release of chemicals that transmit information from one neuron to another (NEUROTRANSMITTERS) by brain cells. For example, nicotine enhances the release of DOPAMINE, which may produce pleasure; norepinephrine, which may suppress appetite; acetylcholine, which produces arousal; SEROTONIN, which may reduce anxiety; and beta ENDORPHIN, which may reduce pain. The development of addiction to nicotine in tobacco users is attributed in part to many of the effects of nicotine that people find desirable.

Effects of Nicotine in Cells. Nicotine binds (attaches) to RECEPTORS on cell membranes that normally bind a neurotransmitter called ACETYLCHOLINE. Acetylcholine, like other neurotransmitters, is a chemical released by nerve endings in the body that binds to certain receptors on cells and activates them. The activated cells communicate messages to other nerves or produce specific actions on body organs. Nicotine activates only certain of the receptors that bind acetylcholine. These receptors are now called nicotinic cholinergic receptors. Using the selective action of nicotine on cholinergic receptors, scientists are able to observe their activity separately from muscarinic cholinergic receptors, receptors activated by a chemical called muscarine. Nicotinic cholinergic receptors are located at the ganglia in the autonomic nervous system, where there are specialized areas for communications between nerves, in the adrenal gland, at the neuromuscular junctions, where nerves attach to and activate muscles, and in many parts of the brain.

The greatest number of nicotine cholinergic receptors in the BRAIN are found in the hypothalamus, hippocampus, thalamus, midbrain, brain stem, and many parts of the cerebral cortex. Nicotine acts on sensory receptors, including those that mediate pain sensations. The effects of nicotine on these specific receptors have been an important tool in studying the effects of neurotransmitters on cell receptors and on the nervous system as a whole. In addition, these studies provide information about the widespread effects of nicotine introduced into the body during tobacco use.

DEVELOPMENT OF PHYSICAL DEPENDENCE ON NICOTINE

Nicotine is the chemical substance responsible for PHYSICAL DEPENDENCE on tobacco products. During the development of physical dependence on a drug such as nicotine, brain chemistry and function change. They return to normal in the presence of nicotine and come to depend on the drug for normal function.

The change that results in normal function in the presence of nicotine is called neuroadaptation or TOLERANCE. When tolerance develops after a period of use of nicotine, or of any drug, the same dose produces less of an effect than previously. Tolerance develops to many of the effects of nicotine. It is well-known that people smoking their first cigarette often experience nausea and vomiting. However, after repeated exposure to cigarette smoke,

these effects disappear. Their disappearance is the development of tolerance to the toxic effects of nicotine in the cigarette smoke. Tolerance also develops to the more desirable effects of nicotine such as pleasure and alertness.

The development of tolerance is associated with changes in the brain, such as an increased number of nicotinic cholinergic receptors found in the brains of smokers studied at autopsy. The changes in the brain correspond to a state in which the tolerant brain comes to depend on nicotine for normal functioning. This state is called physical dependence.

Physical dependence also means that abstinence or WITHDRAWAL symptoms occur when a person who has taken a drug on a regular basis stops taking it. Physical dependence on nicotine has been clearly demonstrated. Thus a person who stops using tobacco after his or her body has adapted to the presence of nicotine will experience withdrawal symptoms in the form of irritability, restlessness, drowsiness, difficulty concentrating, impaired job performance, anxiety, hunger, weight gain, sleep disturbances, slow down in heart rate, and a strong urge for nicotine. In general, withdrawal symptoms are opposite to the effects produced by nicotine when a person who is not tolerant uses it. Thus a person will start using tobacco primarily to experience the desired effects of nicotine, but once the ADDICTION develops, use of tobacco may be chiefly to prevent the emergence of unpleasant withdrawal symptoms. Use of a drug to prevent withdrawal is common in people who are addicted to a drug.

ABSORPTION OF NICOTINE FROM TOBACCO

Nicotine, which is absorbed into the body when tobacco products are used, can be absorbed by different routes and at different rates. Some products deliver nicotine in smoke that is inhaled. In tobacco smoke, nicotine is present in droplets that also contain water and tar. These droplets are carried by gases that include carbon monoxide, hydrogen cyanide, and nitrogen oxides. Such suspended droplets carried by gas are called an aerosol. When the aerosol is inhaled, the droplets are deposited in the small airways of the lungs, from which nicotine is absorbed into the blood stream. After absorption through the lungs, blood containing nicotine moves into the heart and then into the arterial circulation, including the brain. Nicotine reaches the brain within 10 to 15 seconds after a puff on a cigarette. This rapid delivery of nicotine to the brain produces more intensive effects than following slower delivery and provides the close temporal link between SMOKING and the development of addiction.

Nicotine is absorbed into the body in other ways. It can be absorbed in the mouth even if not inhaled in pipe or cigar smoke. In addition, not all tobacco products deliver nicotine through smoke. Chewing tobacco consists of shredded tobacco or plugs of tobacco that are enhanced with licorice and other flavorings. These products are periodically chewed, and the saliva generated is spat out, hence the term *spit tobacco*. Oral snuff is finely cut tobacco. A portion of oral snuff, called a pinch, is placed between the lip and the gum. Nicotine is absorbed from these forms of tobacco more slowly than from inhaled smoke, but the total amount absorbed is similar. Nasal snuff is finely powdered tobacco that is sniffed into the nose, where nicotine is rapidly absorbed.

DOSES OF NICOTINE TAKEN IN TOBACCO

The dose of nicotine absorbed from a cigarette is on average about 1 milligram (mg). The average user smokes about 25 cigarettes a day, an average nicotine intake of 20 to 30 mg daily. The average amount of nicotine absorbed from chewing tobacco or snuff per day is similar to that obtained from cigarettes. A person who smokes 25 cigarettes a day will absorb about 200 grams of nicotine in 20 years of smoking.

NICOTINE-CONTAINING MEDICATIONS

Nicotine is available as a medication, used to assist people in quitting smoking (see articles on NICOTINE DELIVERY SYSTEMS and TREATMENT of smoking and TOBACCO abuse). These medications are meant to provide nicotine to smokers as a substitute for nicotine formerly consumed from tobacco use. Nicotine medications reduce withdrawal symptoms and increase the likelihood that the individual will quit tobacco use. Two forms of nicotine medication are currently available. Nicotine chewing gum (nicotine polacrilex, also known as Nicorette) consists of nicotine in a gum that slowly

releases nicotine during chewing. Each gum is typically chewed for about 30 minutes. People chew up to 16 pieces per day when trying to quit smoking.

Nicotine patches are applied to the skin. They release nicotine slowly through the skin over 16 or 24 hours, depending on the patch used.

Both forms of nicotine-replacement medication deliver doses of nicotine equivalent to that taken in by the average tobacco user. Nicotine chewing gum delivers about 1 to 2 mg per piece. Nicotine patches deliver from 5 to 21 mg, depending on the patch and its strength.

ELIMINATION OF NICOTINE FROM THE BODY

Nicotine in the body is eliminated primarily by breakdown by the liver. The rate of breakdown is such that the level of nicotine in the blood falls about one-half after two hours. This rate is also known as a half-life of two hours. The primary breakdown product of nicotine is cotinine. Cotinine levels in the body are about 10 times higher than those of nicotine. The half-life of cotinine is 16 hours, and cotinine persists in the body for 4 days after a person stops smoking. Cotinine levels can be measured as an indicator of how much nicotine a person is taking in.

NICOTINE ADDICTION

Addiction to nicotine is well documented. The development and characteristics of nicotine addiction are described in detail in a report from the U.S. Surgeon General published in 1988. In this report, *The Health Consequences of Smoking: Nicotine Addiction*, the surgeon general presents criteria for nicotine addiction including the following:

1. Highly controlled or compulsive use. Smokers have great difficulty abstaining. Seventy percent of the 45 million smokers in the United States today report that they would like to quit and can not.
2. Psychoactive effects. Nicotine, as described earlier in this article, has pronounced effects on the brain.
3. Drug-reinforced behavior. Tobacco use is motivated by a desire for the effects of nicotine. People do not smoke cigarettes that do not contain nicotine. Very few people choose to smoke cigarettes that deliver very low doses of nicotine. (See also the article on tobacco.)

Other factors lead to the conclusion that nicotine is addictive:

1. It is used despite harmful effects. Most people know that smoking is harmful to their health and continue to smoke. Many people who have nicotine-related diseases are still unable to quit.
2. RELAPSE following abstinence. Most smokers can quit for a few days or even weeks (abstinence), but most of these smokers return to smoking within a month. Typically, it takes four or five attempts before a smoker is successful at quitting permanently.
3. Recurrent drug cravings. Most smokers have an intense craving or urge to smoke when they have not smoked for some period of time.
4. Tolerance
5. Physical dependence
6. Pleasurable effects

The last three factors were described previously.

Smokers carefully regulate nicotine intake to maintain desired levels of nicotine in the body. Such careful regulation is further evidence that nicotine is addictive. Smokers keep the amount of nicotine obtained from cigarettes constant in two ways.

1. When people are given cigarettes that are labeled as low-yield (see tobacco history for detailed discussions of yields), they smoke more intensively to obtain the same dose of nicotine they were used to obtaining from the higher-yield cigarettes.
2. When they are forced to cut down on the number of cigarettes they smoke each day, they will take in more nicotine per cigarette. Thus when smoking is restricted, smokers tend to maintain the nicotine in their bodies at close to levels maintained during unrestricted nicotine intake.

BEHAVIORAL ASPECTS OF TOBACCO ADDICTION

People continue to smoke both because they enjoy the direct drug effects of nicotine and because use of nicotine becomes associated with other pleasures through learning—for instance, when the pleasurable effects of nicotine occur repeatedly in the presence of specific cues or events in the envi-

ronment. Eventually, those cues and events become a signal to smoke. For example, people often smoke after meals, while drinking a cup of coffee or an alcoholic beverage, during a break from work, while talking on the phone, or while with friends who smoke. After smoking in these situations hundreds of times, the user may find that these situations themselves produce a powerful urge for a cigarette.

There are other learned pleasures that keep people smoking independent of the pharmacological effects of nicotine. Handling of smoking materials, and the taste, smell, or feel of tobacco smoke in the throat, all can become associated with the effects of nicotine and then become pleasurable in themselves. A person who tries to quit must learn to give up not only the pharmacological actions of nicotine but also the aspects of smoking that have become pleasurable through learning. Urges aroused after learning an association between aspects of the environment and the pleasures of smoking prompt relapses in many people who have already overcome withdrawal from nicotine and quit tobacco use.

Smokers report many other reasons for their habit. For example, many smokers, particularly women, smoke to maintain lower body weight. Others seem to use tobacco to control mood disturbances, such as DEPRESSION or ANXIETY.

COMPARISON OF ADDICTION TO NICOTINE AND OTHER DRUGS

Nicotine addiction is similar to and as powerful as addiction to other drugs, such as HEROIN, ALCOHOL, and COCAINE. All these drugs have psychoactivity and produce pleasure. They increase the likelihood that people will spend time looking for them and engaging in rituals while taking them and that users will continue to take them in the face of risk to their well-being and health. The psychoactivity of nicotine is subtle and does not interfere with normal functioning in daily life. Thus nicotine's psychoactivity differs from that of heroin and cocaine, which produces more intense euphoria and may be disruptive to everyday functioning. Despite this difference, nicotine is addictive. A subtle psychoactive effect, especially when experienced with each puff of smoke, taken hundreds of times a day, exerts a powerful effect on behavior over time. The magnitude of effect becomes apparent when each puff of cigarette is considered as a dose of nicotine. A smoker who takes 8 puffs per cigarette and smokes 20 cigarettes per day is receiving up to 160 doses of nicotine per day. The dosing is equivalent to 58,400 doses a year, or 1,168,000 doses after 20 years of smoking.

When difficulty in quitting and relapse after attempting to quit are compared, it becomes apparent that nicotine is even more addictive than other drugs of abuse. Ninety percent of all people who smoke cigarettes are addicted and have difficulty quitting. In contrast, only about 10 percent of people who drink alcohol at all have difficulty controlling use and would be classified as addicted. The percentage of occasional versus addicted users of heroin and cocaine is not known, but when multidrug users are asked about which drug they would have most difficulty giving up, the choice is most commonly nicotine (that is, cigarettes). Relapse rates among adults after cessation of alcohol, heroin, and tobacco use are similar.

NICOTINE ADDICTION IN YOUTH

Ninety percent of all tobacco users begin smoking before the age of 20. The earlier in life one starts smoking, the more likely he or she is to become a regular smoker and the more cigarettes he or she will smoke as an adult. The development of addiction in youth involves a series of steps including

- a trying stage
- experimentation
- regular smoking
- nicotine addiction

The typical interval between trying and addiction is 2 to 3 years.

Initially, young people smoke for social and psychological reasons. The motivations include the influence of parents and friends who are smokers, and the positive images of smoking perpetuated in television and movies and in advertisements in magazines, at music and sports events, and on billboards. Personal factors also play a role. Some include poor school performance, low self-esteem, poor self-image, sensation seeking, rebelliousness, failure to take seriously the adverse effects of tobacco use, and depression or anxiety. While early stages of smoking usually consist of occasional sessions with friends, tolerance develops and withdrawal symptoms are experienced between cigarettes as smoking becomes more frequent. Many

youths report withdrawal symptoms and difficulty quitting. They consider themselves addicted to tobacco.

TREATMENT OF NICOTINE ADDICTION

Treatment of nicotine addiction is discussed in the articles entitled *Treatment: Tobacco*. The approach may be summarized as follows. Initial therapy usually does not include drugs. Smokers are encouraged to pick a day and just stop (go cold turkey). Some smokers participate in formal behavioral therapies, such as those available in smoking-cessation clinics. Those who are unable to stop on their own or with behavior therapies are more likely to be highly addicted to nicotine and are candidates for pharmacological (drug) therapy. The main drug therapies for smoking are nicotine-containing medications such as chewing gum or transdermal (skin) patches.

(SEE ALSO: *Addiction: Concepts and Definitions; Adolescents and Drugs; Reward Pathways and Drugs; Tobacco: Smokeless; Tolerance and Physical Dependence; Withdrawal: Nicotine*)

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NICOTINE DELIVERY SYSTEMS FOR SMOKING CESSATION Several nicotine delivery systems have been devised to assist nicotine-dependent cigarette smokers to quit smoking. The aim of nicotine replacement therapy (NRT) is to provide temporary relief of smoking withdrawal symptoms such as irritability, anxiety, hunger, restlessness, drowsiness, and craving for cigarettes. Meanwhile, the smoker learns to resist smoking in a variety of situations that have been repeatedly associated with smoking in the past. Eventually, the goal is to relinquish the alternative source of nicotine, which is not as addictive as cigarettes. Quitting smoking, which is such a difficult task for many, is thereby simplified by breaking the process into two steps:

(1) giving up the habit of smoking while retaining some of the effects of nicotine, and
(2) relinquishing the nicotine, perhaps weeks or months later.

While using an alternative nicotine delivery system, smokers also avoid the intake of hazardous smoke components such as carbon monoxide and cancer-causing "tar."

Nicotine chewing gum. Nicotine chewing gum was the first alternative nicotine delivery system to be approved as a smoking cessation aid. Nicotine is contained in a gum resin and is slowly released upon chewing. Nicotine gum is available in two strengths, containing either two milligrams or four milligrams of nicotine. Of that amount, about half is released on chewing, which is comparable to the amount of nicotine delivered from one or two cigarettes. Unlike cigarette smoking, which delivers the nicotine rapidly into the bloodstream through the lungs, nicotine from the chewing gum is slowly absorbed through the cheeks. Most of the nicotine that is swallowed does not reach the general circulation, because after being absorbed from the small intestine, it is destroyed as it passes through the liver. The use of nicotine gum has been shown to double success rates in smoking cessation. Problems with the gum include unpleasant taste, jaw soreness, stomach upset from nicotine that is swallowed, and inconsistent levels of nicotine in the bloodstream.

Nicotine skin patches. Partly to overcome the unpleasant side effects of nicotine chewing gum, nicotine skin patches were developed to release a controlled amount of nicotine directly through the skin. Nicotine is easily absorbed through the skin, and it is possible to provide a steady delivery of approximately 21 to 22 milligrams per day, equivalent to the amount of nicotine delivered from about twenty cigarettes (one pack). However, as with nicotine chewing gum, the nicotine is delivered much more slowly than from cigarettes, and the peak blood levels are thus lower than those obtained from cigarettes. The patches are applied once a day, and after using full-strength patches for at least 4 weeks, reduced-strength weaning patches can be used to gradually withdrawal from nicotine.

Use of the patch has been shown to double or triple success rates in quitting smoking; a small proportion of patients (less than 10%) do experience skin irritation from wearing the patches.

Nicotine nasal spray. Some researchers have speculated that a more rapid absorption of nicotine than is achieved with patches or gum would more closely simulate the effects of cigarettes desired by smokers and increase success rates in smoking cessation. A nicotine nasal spray is available for smoking cessation treatment; it delivers 1 milligram of nicotine (equivalent to the delivery of a typical cigarette) with each use (one spray per nostril). Unlike other modes of NRT, the nasal spray delivers nicotine to the bloodstream very rapidly, within a few minutes. Some studies have suggested the nasal spray might be particularly advantageous for more highly dependent smokers. Problems with the spray include local irritation caused by nicotine, which can result in sneezing, a runny nose, watering eyes, and a cough.

Nicotine inhaler. A fourth mode of NRT resembles a cigarette in size and shape, and releases a nicotine vapor when a smoker puffs on it. However, the dose of nicotine released in each puff, which is limited by the vaporization of nicotine at room temperature, is much less than with cigarette smoking. Intensive use (eighty inhalations over 20 minutes) releases, on average, 4 milligrams of nicotine, of which 2 milligrams is absorbed. Although termed an ''inhaler,'' studies have shown that the nicotine vapor is deposited mainly in the mouth, and hence absorption rates resemble that of nicotine gum. The inhaler can provide some of the behavioral and sensory characteristics associated with smoking and may therefore be appealing to smokers seeking a weaning tool that provides these components. However, the sensory effects of nicotine also can produce adverse effects, including mouth irritation and cough.

Commonalties across NRT products. Each of the four NRT systems discussed has been shown to facilitate smoking cessation, approximately doubling or tripling abstinence rates over placebo. They are effective even in the absence of a formal behavior therapy program, although behavioral treatment in combination with the nicotine replacement further enhances success rates. Interestingly, success rates are similar across the different methods, although more research needs to be done to determine whether different types of smokers will benefit more from one treatment than another.

What is missing from nicotine replacement? One might suppose that with the varied nicotine replacement techniques available, success rates in smoking cessation treatment would be higher than the typical long-term outcome (e.g., at one year) of 10 to 20 percent. Unfortunately, the vast majority of smokers relapse to cigarettes, raising the question of what is missing from NRT that cigarettes provide. It has been widely believed that the rapid absorption of nicotine from the lung during cigarette smoking accounted for the unique addictiveness of cigarettes; however, some doubt has been cast on this interpretation in view of the modest efficacy of the nasal spray despite extremely rapid absorption of nicotine, and by laboratory studies indicating that even rapid intravenous nicotine injections do not reproduce the enjoyable aspects of cigarette smoking. Research has suggested at least two other key components may be missing from NRT. One component alluded to above, consists of the sensory and behavioral cues associated with inhalation upon which smokers have become dependent. Although the nicotine inhaler provides some of these cues, it does not deliver tobacco taste or replace what smokers find to be enjoyable sensations of inhaling cigarette smoke. A second component that may also be important entails non nicotine constituents in tobacco that inhibit an enzyme (monoamine oxidase) important to the breakdown of neurotransmitters in the brain (e.g., dopamine), which in turn may mediate the chemical reward of nicotine. Methods of replacing these missing components are being developed and may yield further improvements in treatment efficacy.

Bupropion. Bupropion was the first non nicotine pharmaceutical to be approved by the U.S. food and Drug Administration for smoking cessation treatment and had been marketed previously as an antidepressant. However, it is efficacious in smoking cessation treatment even for smokers who are not depressed. Although the mechanism of action relevant to smoking cessation has not been elucidated, bupropion raises the level of brain neurotransmitters involved in drug reward, such as dopamine and norepinephrine. Bupropion has also been shown to block the action of nicotine at certain receptors. Clinical trials have demonstrated that bupropion approximately doubles success

rates over placebo, and the most frequent side effects include insomnia and dry mouth.

Combination approaches. Many potential combination approaches have yet to be thoroughly evaluated; they may increase success rates beyond those of any one technique alone. This has already been seen in the enhancement of success rates with NRT by behavior therapy programs. Additional treatment combinations may include the use of two or more nicotine delivery systems at the same time. A patch might provide a steady baseline level of nicotine, which could be supplemented as the need arises by the use of gum, nasal spray, or the inhaler. Another promising combination may be NRT plus bupropion, which some research suggests may have additive benefits. Combinations of NRT and techniques that provide some of the missing components of tobacco discussed above may also be considered. These and other possibilities need to be tested in future research because smoking has proven to be a more formidable adversary, as well as a more tenacious addiction, than many would have initially suspected.

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JED E. ROSE

NICOTINE GUM *See* Nicotine Delivery Systems for Smoking Cessation

NICOTINE PATCH *See* Nicotine Delivery Systems for Smoking Cessation

NIDA *See* U.S. Government Agencies: National Institute on Drug Abuse (NIDA)

NITRITES *See* Inhalants

NITROUS OXIDE *See* Inhalants

NOREPINEPHRINE Also referred to as adrenaline, it is a catecholamine NEUROTRANSMITTER known to be involved in the action of some addicting drugs. It is the biochemical product of DOPAMINE and the enzyme dopamine-beta-hydroxylase. It is the major neurotransmitter for the sympathetic nervous system, as well as for several sets of long axon, multiple-branched neurons (nerve cells) of the central nervous system. After release from nerve terminals onto its RECEPTORS, much of it is recaptured or removed from extracellular spaces by an uptake mechanism, or TRANSPORTER, located in the nerve terminal membrane. This transporter is an important drug target for antidepressants and psychostimulants. Monoamine oxidase is a well-known enzyme that breaks down norepinephrine.

Norepinephrine holds an important place in the history of drug studies. It was discovered as an active chemical in the body many years ago. The availability of pharmacological agonists and antagonists helped reveal its physiologic role in the body. Also the development of histochemical methods in the 1960s and 1970s for its direct light microscopic visualization led to a detailed understanding of the many neurons that contain it. Noradrenergic receptors, termed alpha and beta, can act independently or synergistically to mediate the activity of norepinephrine and related drugs. Brain noradrenergic neurons in the nucleus locus ceruleus are well char-

acterized in general and are activated during withdrawal from addictive drugs.

FLOYD BLOOM
REVISED BY MICHAEL J. KUHAR

NUCLEUS ACCUMBENS The nucleus accumbens is a group of NEURONS that is part of the limbic system and located near the midline in the frontal region, beneath the frontal lobe. Anatomically, it has been divided into the shell and core, with the shell perhaps being more important for the actions of drugs of abuse. It is one of the most important structures in the brain for studies of drug addiction because it is believed to be involved in reward, reinforcement, and unpredictably positive experiences. Nucleus accumbens is known to include neurons that contain GABA and acetylcholine and other neurotransmitters. It receives important input from dopaminergic neurons located in the ventral midbrain that are also involved in reward and reinforcement. It has output projections back to the ventral midbrain and other areas.

This nucleus is thought to be involved in the action of many different drugs of abuse, especially psychostimulants whose actions on the nucleus accumbens have been well studied. Destruction of neurons in this structure or its inputs disrupts psychostimulant self-administration by rodents, and psychostimulants and other drugs of abuse cause an efflux of dopamine from this structure. Because of its small size, it has been difficult to study, and, at this time, it is being studied in humans and nonhuman primates to determine its relevance to human drug and stimulant abuse.

JAMES E. SMITH
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NUTMEG Nutmeg, the common spice obtained from the aromatic seed of the tree *Myristica fragrans* (native to the Moluccas, the spice islands of the East Indies), has been used for centuries for food and medicinal purposes. It has some HALLUCINOGENIC activity when consumed in large amounts. Since nutmeg is found in most kitchens, including food-preparation areas found in prisons, it has been used by prisoners. Therefore, it has been removed from ready access in prisons to the tighter control of drugs of abuse; Malcolm X wrote about such use.

Figure 1
Nutmeg

Nutmeg contains elemicin and myristicin, whose structures have some similarities to the hallucinogen MESCALINE as well as to the Psychostimulant AMPHETAMINE. It has been hypothesized that elemicin and myristicin might be metabolized in the body to form an amphetamine- and/or mescaline-like compound, but this has not been proven. The effects of nutmeg have been reported to have some similarities to those produced by MARIJUANA; however, the large amounts of nutmeg that must be ingested to get behavioral effects can cause dry mouth and thirst, increases in heart rate, vomiting and abdominal pain, severe headaches, agitation, and panic attacks.

(SEE ALSO: *Lysergic Acid Diethylamide and Psychedelics; Plants, Drugs from*)

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NUTRITION, ALCOHOL, AND DRUGS
See Complications: Nutritional

O

OBESITY This term derives from the Latin (*obesus*, meaning "to eat up"), and it came into use in English in the early 1600s to mean a condition characterized by excessive bodily fat. Excess body weight is associated with the increased storage of energy in the form of adipose tissue. Standard criteria for obesity are (1) greater than 20 percent above ideal body weight (IDW) for a given height, as determined from actuarial tables, or (2) body mass index (BMI), defined as weight in kilograms divided by height in meters squared ($kg \div m^2 =$ BMI), greater than 27 for men and greater than 25 for women.

Obesity represents the upper end of a body-weight continuum, rather than a qualitatively different state. Obesity can derive from a variety of causes, but a significant genetic contribution has been demonstrated.

Being overweight to a statistically significant above-average degree or having proportionately more body fat than average is believed to be due primarily to genetic factors that influence appetite, metabolism, and activity levels. Most notably, obesity is more prevalent (ten times more likely) in persons whose parents, brothers, or sisters are obese. Studies in identical twins have clearly demonstrated that genetics plays a major role. For example, nonidentical twins raised together were less similar in weight than identical twins raised apart.

Beyond the genetic component, researchers have been examining the role of hormones, most specifically leptin, a hormone secreted by fat tissue that affects the brain's appetite control centers. In some studies, mice given injections of leptin lost their appetites and, consequently, lost weight. The human response to leptin varies dramatically, and the relationship between plasma leptin levels and obesity in humans is not yet clear or confirmed. According to one study, mutations in the leptin gene are indeed responsible for obesity in both mice and humans, but these mutations are quite rare outside of the laboratory setting. Another study shows that leptin is a signal to the hypothalamus of peripheral fat deposits, but further studies are being conducted to determine if obese individuals have trouble with leptin access into the brain. Other researchers have found that lean, physically active men have lower levels of leptin than heavier, sedentary men (ages 47 to 83).

Leptin research continues since solid findings could help in the treatment and prevention of obesity and diseases and health problems linked to obesity, such as hypertension, stroke, and type 2 diabetes (diabetes mellitus).

The *prevalence* of obesity (in this case defined as having body fat in excess of 25% for males or 30% in females) varies remarkably across ethnic groups and cultures, and across age groups. In the United States, obesity is consistently less common among African-American men than among white men across the entire age range; is consistently more common among African-American women than among white women; and tends to be more com-

mon among women of Eastern European and Italian ancestry than among those of British ancestry. Socioeconomic factors affect the prevalence of obesity, but men and women are affected differently: It is more common among all women in lower socioeconomic groups, but men in lower socioeconomic groups are leaner than average. Overall, approximately 40 million Americans are obese.

Some researchers and clinicians see similarities among certain patterns of overeating and other excessive behaviors such as drinking too much ALCOHOL, compulsive GAMBLING, engaging in "too much" sexual activity, and even exercising compulsively. Although there may be such similarities, the semantics attached to problems of overeating and OBESITY are formidable.

Not all persons whose weight is above average are obese (they may have excess muscle mass); not all who are obese eat excessively; not all who eat excessively become obese; and some individuals who have clinically recognized disorders centered on eating and body weight, such as BULIMIA, may or may not be obese.

(SEE ALSO: *Bulimia Nervosa; Overeating and Other Excessive Behaviors*)

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OPERATION INTERCEPT Described by government sources as the largest peacetime search-and-seizure operation in U.S. history, Operation Intercept was launched along the United States—Mexico border in September 1969. This unilateral program was instituted, ostensibly, to halt the flow of MARIJUANA, HEROIN, and other dangerous drugs from MEXICO into the United States. However, Intercept's true goal was not to interdict narcotics but to publicize the war on crime promoted by President Richard M. Nixon, who had taken office the previous January, and to force Mexican compliance with Washington's antidrug campaign. Fashioned by well-meaning but short-sighted law-enforcement officers, who all but totally neglected the State Department and knowledgeable border-state residents, Operation Intercept constituted a classic example of international pressure politics and became a serious incident between Mexico and the United States.

On September 16, 1968, presidential candidate Nixon had pledged to an Anaheim, California, audience that, if elected, he would move against the source of drugs and accelerate the development of tools and weapons to deter NARCOTICS in transit. As president, he came face-to-face with the reality of a staggering national drug abuse problem and accelerating drug-related street crime. With the director of his own BUREAU OF NARCOTICS AND DANGEROUS DRUGS contending that the United States had "failed miserably" in controlling narcotics abuse, Nixon chose to couple a highly publicized law-and-order campaign at home with an international offensive against foreign sources of heroin and marijuana. Attorney General John Mitchell was chosen to implement the program, and in April 1969 he assembled a multiagency task force to attack the importation into, and illegal sale and use of illicit drugs in, the United States.

Establishing a linear relationship between marijuana, deteriorating health, heroin usage, and increased crime, the task force turned its attention to the border problem. Mexico was correctly deemed

the primary source of high-potency marijuana entering the United States. Officials noted further that (1) a significant percentage of the heroin was of Mexican origin, (2) substantial quantities of European heroin were being smuggled across the southern frontier, (3) Mexico served as an in-transit point for South American COCAINE, and (4) considerable amounts of AMPHETAMINES and BARBITURATES entered the United States surreptitiously from Mexico. In the midst of so much smuggling, Mexico's resources and efforts remained inadequate. Something had to be done to elicit a concerted, sustained antidrug program from Mexico City. That something was Operation Intercept.

On Sunday afternoon, September 21, 1969, at exactly 2:30 P.M. Pacific standard time, "the biggest, broadest-based enforcement task ever mounted" was launched. Noting that the Mexican government had been kept "fully informed" of the operation, a U.S. Treasury Department news release termed Intercept a "coordinated effort" encompassing the law-enforcement resources of several branches of the federal government. Involving intensified land, sea, and air surveillance along the entire 1,945-mile U.S.—Mexico border, the effort would continue "for an indefinite period," as everything and everyone, no matter their nationality or status, was thoroughly and painstakingly searched.

More than 4.5 million individuals and their belongings were ultimately inspected. Vehicles, their component parts, personal baggage, purses, books, lunch boxes, jackets, toys, and in some cases even blouses and hairdos were searched. The daily routine of life in Mexican border cities was radically altered, as traffic backed up for miles, car radiators boiled over, and tempers, both private and diplomatic, flared. No person or object—including diplomatic and consular officials, their children, possessions, and even their diplomatic cargo—was spared during Intercept's 20-day existence. In the process, the maneuver encompassed some 2,000 personnel, intensified inspections, heightened air and sea surveillance, and the expenditure of some 30 million dollars.

Analyzed solely on the basis of drugs confiscated, Intercept surely was not worth the cost and effort it entailed. Seizures, however, were of minor importance. The primary objective was to "bring the Mexicans around, get them really moving against cultivation and trafficking." In this regard, the operation must be judged a qualified success. Diplomatic outcries notwithstanding, Intercept did play an undeniably important role in energizing Mexico's moribund antidrug program during the 1970s.

Viewed retrospectively, Operation Intercept's basic weakness was embodied in its title, for its purpose was not to interdict drugs at the border but to pressure Mexico through economic denial. Seeking a politically expedient solution to the highly complex problem of domestic drug abuse, the Nixon administration chose Mexico. Unfortunately, the White House failed to recall the salient fact that Mexico is a foreign country, and a friendly one at that.

Neglect of the State Department proved a serious blunder. Overlooked or overpowered by law-enforcement officials during Intercept's crucial formative stage, U.S. diplomats ultimately terminated the ill-advised project before it became an even greater diplomatic disaster. More important, if its supporters had managed to prolong the unilateral maneuver for an extended period, U.S. authorities probably would have never secured the level of cooperation they sorely needed to impair the cultivation of drugs in Mexico and the trafficking of drugs across the border.

Equally damaging was the failure of Intercept officials to gauge the impact of such a blockage on the U.S. border's economy. Highly dependent on Mexican shoppers, American border merchants reacted angrily and effectively through professional and civic groups. Pressure on the administration from border-state members of Congress was intense, and its impact increased as the project was prolonged. Along with diplomatic protests, this proved crucial to Intercept's demise.

Additionally, the operation was poorly timed; it came on the eve of *tapadismo*, the process through which Mexico chooses its next president, but before the Nixon administration's announcement of a Latin American policy. Furthermore, Mexico played host during the Intercept period to a regional meeting of the United Nations Commission on Narcotic Drugs and the thirty-eighth annual assembly of INTERPOL, thereby compounding its embarrassment over the blockade's indignities.

Yet despite its numerous shortcomings, Operation Intercept was not entirely void of accomplishments. Because of the tremendous publicity it en-

gendered, the program made Mexican officials keenly aware of a reality heretofore ignored or slighted—that nation's own burgeoning drug problem. Politicians and journalists became introspective and reluctantly admitted that the availability of domestically produced drugs posed a danger to the health of *nuestra juventud* (our youth) as well as providing an everyday pastime for "gringo jippies" (American hippies).

Intercept also helped spur a previously lagging Mexican campaign against the cultivation, manufacture, and shipment of illicit drugs of all kinds. Since the fall of 1969, the government of Mexico has budgeted ever increasing funds for *la campaña permanente* (the permanent campaign) and is presently conducting (mid-1990s), with U.S. assistance, the world's most comprehensive eradication program against opium poppies and marijuana plants. As a corollary to this effort, cooperation between Mexican and American narcotics officials improved dramatically during the 1970s, only to tail off during the 1980s. Thus, while Intercept proved a short-term diplomatic blunder, it indirectly and somewhat ironically became a long-term catalyst to an accelerated Mexican antidrug campaign and a springboard to more effective international cooperation.

(SEE ALSO: *Border Management; Crime and Drugs; Crop Control; Drug Interdiction; International Drug Supply Systems; Transit Countries for Illicit Drugs; U.S. Customs Service*)

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OPERATION PAR *See* Treatment Programs/Centers/Organizations: An Historical Perspective

OPIATES/OPIOIDS The *opiates* are central nervous system depressants that are found in OPIUM or are derived from a substance found in opium, which is the juice of the opium poppy (*Papaver Somniferum*). The *opioids* include the opiates, along with totally synthetic agents, and naturally occurring peptides that bind to one or more opioid receptors found in a number of animal species. In general usage, both terms are often used interchangeably—but opioids is the larger grouping.

The effects of opium have been known for several thousand years. For most of this time it was not clear which of the ingredients in opium provided its analgesic (painkilling) and other therapeutic properties. Regardless of their benefits, health care providers are often afraid to prescribe them for fear of psychological dependence and sale to illegal markets (Carver, 2000). Still, the medical community has been increasing the use of opioid analgesics (Increasing Use, 2000).

MORPHINE and CODEINE, two of the most abundant constituents of opium, were the first pure opiates isolated—morphine in 1806 and codeine in 1832. Chemical modifications were soon attempted in an effort to eliminate their problematic side effects. One of the first attempts (in the 1890s) produced 3, 6-diacetylmorphine, which is commonly known as heroin. This agent did not eliminate the problems of tolerance, dependence, or abuse. Since then, extensive studies of the important components of morphine's structure have led to the development of a number of different classes of organic compounds. In 1939 and 1940, the first synthetics were discovered. The recent discovery of the opioid peptides have provided even more diversity in drug design.

AGONISTS, ANTAGONISTS, AND PARTIAL AGONISTS

Some drugs have very complex actions and many drugs act at specific RECEPTOR, locations on the surface of a cell. All of the drugs that belong to the class of drugs called opioids act at opioid receptors on the surface of cells. Usually these cells are neurons, but there are also opioid receptors on white blood cells. Once a drug binds to a receptor, it can either turn it on (AGONIST) or do nothing (ANTAGONIST). Even if a compound does nothing once it binds to the receptor, it still blocks the site and prevents an active compound from binding to the receptor. The situation is much like a key in a lock; some keys fit into the lock but will not turn, and as long as they remain in the lock they prevent the insertion of keys that would turn the lock. Finally, there are drugs known as *partial agonists*; these compounds bind to the receptor and turn it on but not nearly as well as pure agonists.

Again, using the key analogy, these partial agonists will turn in the lock, but only with some jiggling, lowering the efficiency in opening the door. Pharmacologically, partial agonists have limited effects at the receptor, termed a *ceiling* effect. This means that increasing the dose further will not give a greater response. To further complicate understanding of these drug actions, it is important to recognize that the opioid receptors (and many other types of receptors as well) are actually families of similar but subtly different receptor types. Some opioids are agonists at one receptor type and partial agonists or even antagonists at another receptor type. These drugs are termed mixed agonist/antagonists and they can have complex pharmacological profiles. For this reason it can be difficult for pharmacists to determine conversion amounts (for example, to methadone) (Magill-Lewis, 2000).

RECEPTORS

Morphine and drugs with similar actions work through specific recognition sites, termed *receptors*, located on the outside of cells (see Table 1). A number of general classes of opioid receptors have now been identified and it is likely that even more will be discovered. The major types of opioid receptors have been designated mu, kappa, and delta. From the clinical perspective, the mu opioid receptors are the most important. This class, comprised of two subtypes, mu_1 and mu_2, have high affinity for morphine and most of the clinically used agents. Both mu subtypes mediate analgesia, but through different mechanisms and locations within the brain and spinal cord. Mu receptors have been implicated in euphoria and mu agonists have often been abused. Equally important, activation of mu receptors depresses respiration and inhibits gastrointestinal transit. In addition to analgesia, euphoria, respiratory depression, and decreased activity in the stomach, mu agonist opioids produce

TABLE 1
Tentative Receptor Classification

Receptor	*Agonists*	*Analgesia*	*Other Action*
Mu	Morphine		
mu_1		Supraspinal*	Prolactin release
			Acetylcholine turnover
mu_2		Spinal	Respiratory depression
			Inhibition of gastrointestinal transit
			Guinea pig ileum bioassay
Kappa			
$kappa_1$	Dynorphin A	Spinal	Diuresis
			Sedation (?)
			Rabbit vas deferens bioassay
$kappa_2$	Bremazocine		Pharmacology unknown
$kappa_3$	Nalorphine	Supraspinal	
Delta	Enkephalins	Spinal	Mouse vas deferens bioassay
			Dopamine turnover

*The supraspinal system is far more sensitive than the spinal one.

some actions that are clinically useful, such as cough suppression. However, most of their actions are considered unwanted side effects; for example, they affect endocrine function, constrict pupils, induce sweating, and cause nausea and vomiting. All mu agonist opioids also induce increasing tolerance and physical dependence in the user.

Kappa opioid receptors were defined using ketocyclazocine, an experimental benzomorphan derivative, and subsequently with dynorphin A, an endogenous opioid, which is believed to be the natural ligand for at least one of the kappa receptor subtypes. Morphine has relatively poor affinity for kappa receptors, but other drugs, such as pentazocine and nalbuphine (analgesics in clinical use), interact with kappa receptors quite effectively. The importance of kappa mechanisms in their actions have only recently been appreciated. The pharmacology of kappa receptors in humans has not been extensively studied; however, animal studies indicate that the kappa receptors also can relieve pain through receptor mechanisms distinct for each of the subtypes. Many of the clinically used drugs active at kappa receptor are mixed agonists/antagonists. Although they are agonists at kappa receptors, they are antagonists or partial agonists at mu receptors. In contrast to mu agonists, which can produce mood elevations and euphoria, drugs that activate kappa agonists appear to produce weird feelings and dysphoria.

The discovery of the enkephalins—endogenous peptides with opioid properties—soon led to the identification of delta receptors. The clinical pharmacology of delta receptors is not well known, primarily because so few agents have been tested in humans. Again, animal testing indicates an important role of delta receptors in analgesia, which is supported by a few studies with humans. However, there are no pure delta agonists clinically available yet.

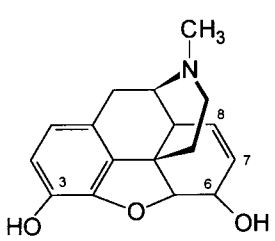
Although all the various receptor subtypes examined can relieve pain, each receptor represents a different mechanism of action. Their sites of action within the brain differ and, most importantly, agents highly selective for a specific subtype do not show cross-tolerance. While tolerance develops with continued activation of any of the various receptors, tolerance to one does not lead to tolerance to another. For example, tolerance to morphine does not diminish the response to a kappa or delta drug. Similarly, mu agonists produce a characteristic variety of physical dependence, and there is cross-dependence among mu agonists (that is, people dependent on heroin will not experience withdrawal if given methadone.) However, there is no cross-dependence between mu agonists and kappa agonists.

All the various subtypes produce a number of actions other than analgesia. Most of the nonanalgesic actions of opiates can be explained by considering the receptors to which they interact. An excellent example is mu_2 receptors, which mediate respiratory depression and the constipation seen with morphine. Drugs that are agonists at these receptors also produce these side effects while compounds lacking affinity for these receptors do not. The role of multiple receptors is important clinically, primarily since few drugs are specific for one receptor. Even morphine, which is highly selective for mu receptors, interacts with two mu subtypes, and at higher doses with delta receptors as well.

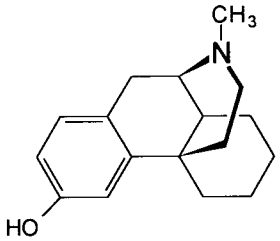
CLASSES OF OPIOIDS

Opioids can be divided into a series of classes based upon their chemical structures, illustrated by prototypic compounds from each group (see Figure 1). These include morphine and its close analogs, the morphinans, the benzomorphans, the phenylpiperidines, and methadone. The pharmacology of agents within each category can be quite varied and often can be predicted from their affinity for various opioid-receptor subtypes. Most of the clinically relevant drugs will interact with more than one receptor. Thus, their actions can be ascribed to the summation of a number of receptor actions.

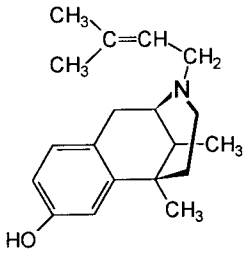
The importance of various regions of the morphine molecule has been well studied and a number of related compounds are widely used (see Figure 2). Early studies examined small changes in morphine's structure. One of the critical groups is the hydroxyl group at the 3-position on the molecule. Blockade of this position by adding chemical groups markedly reduces the ability of the drug to bind to opioid receptors. Although this may seem at odds with the analgesic activity of codeine, which lacks a free hydroxyl group at the 3-position, evidence indicates that codeine itself is not active and is metabolized to morphine, which is responsible for its actions. A similar situation exists for OXYMORPHONE and OXYCODONE.



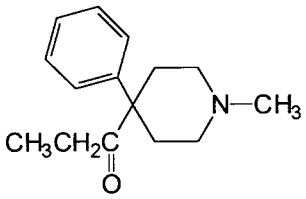
Morphine



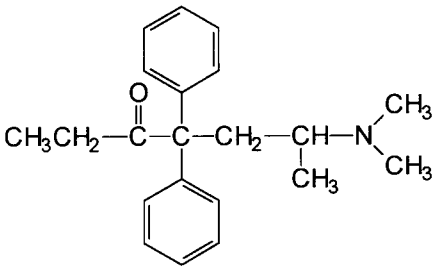
Levorphanol
(morphinan)



Pentazocine
(benzomorphan)



Meperidine
(phenylpiperidine)

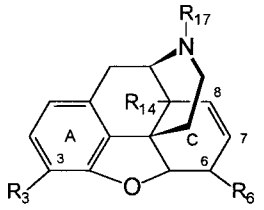


Methadone

Figure 1
The Classes of Opioid Compounds, Based on Structure

The morphine molecule has a single nitrogen atom. The substituent on the nitrogen in these series of opiates can have major effects on activity. Morphine and most of the mu agonists contain a methyl (CH_3-) group on the nitrogen, but a number of other compounds with different substituents have been developed. Replacing the methyl group with an allyl (-$CH_2CH=CH_2$2) or methylcyclopropyl (-$CH_2CHCH_2CH_2$) group does not have much effect upon the ability of the compound to bind to opioid receptors, but it markedly changes what happens when they do bind. For example, oxymorphone, with its methyl group on the nitrogen, is a clinically useful analgesic many times more potent than morphine. Replacing the methyl group with an allyl group produces NALOXONE. Naloxone is an antagonist, a drug that blocks or reverses the actions of other opiates. Clinically, naloxone is used as an antidote to opiate overdose. This shows how simple changes can profoundly influence the pharmacology of these agents.

Further investigations revealed that Ring C of morphine can be eliminated, enabling use of the benzomorphans—many of which are potent analgesics. The major drug in this group is pentazocine (Talwin). Even simpler structures produce potent



	POSITION ON MOLECULE					
Drug	**R_3**	**R_6**	**R_{14}**	**R_{17}**	**C_6–C_7**	**Action**
Morphine	OH	OH	H	CH_3	=	Agonist
Codeine	CH_3O	OH	H	CH_3	=	Agonist
Heroin	O ‖ CH_3CO	O ‖ CH_3CO	H	CH_3	=	Agonist
Oxymorphone	OH	=O	OH	CH_3	–	Agonist
Oxycodone	CH_3O	=O	OH	CH_3	–	Agonist
Hydromorphone	OH	=O	H	CH_3	–	Agonist
Naloxone	OH	=O	OH	$CH_2CH=CH_2$	–	Antagonist
Nalbuphine	OH	OH	OH	CH_2—◇	–	Ag/Antag
Nalorphine	OH	OH	H	$CH_2CH=CH_2$	–	Ag/Antag

Figure 2
The Morphine Molecule and Some Widely Used Related Compounds, Based on Region of the Molecule

analgesics, such as methadone. The phenylpiperidines comprise another large group of opioids. The first of these to be used clinically was meperidine, which was first prescribed in 1939 and which still is extensively used. Modifications of the phenylpiperidine structure led to a subgroup of drugs, with fentanyl as a prototype. Fentanyl is approximately 80-fold more potent than morphine, but its very short duration of action requires continual infusions. An advantage is that once the infusion is discontinued, the effects of the drug clear rapidly. This ability to quickly turn on or off the drug's actions, along with its great potency, has made this agent a valuable tool in anesthesia. Recently, this high potency has been exploited to develop skin patches which give a constant release of fentanyl into the body as the drug is absorbed through the skin. Other agents within this series, such as sufentanil and alfentanil, are even more potent than fentanyl. Two other members of this series, loperamide and diphenoxylate, have activity but very poor solubility. This property has led to

TABLE 2
Selected Opioid Peptides

[Leu^5]enkephalin	**Tyr-Gly-Gly-Phe-Leu**
[Met^5]enkephalin	**Tyr-Gly-Gly-Phe-Met**
Dynorphin A	**Tyr-Gly-Gly-Phe-Leu-**Arg-Arg-Ile-Arg-Pro-Lys-Leu-Lys-Trp-Asp-Asn-Gln
Dynorphin B	**Tyr-Gly-Gly-Phe-Leu-**Arg-Arg-Gln-Phe-Lys-Val-Val-Thr
α-Neoendorphin	**Tyr-Gly-Gly-Phe-Leu-**Arg-Lys-Tyr-Pro-Lys
β-Neoendorphin	**Tyr-Gly-Gly-Phe-Leu-**Arg-Lys-Tyr-Pro
β_h-Endorphin	**Tyr-Gly-Gly-Phe-Met-**Thr-Ser-Glu-Lys-Ser-Gln-Thr-Pro-Leu-Val-Thr-Leu-Phe-Lys-Asn-Ala-Ile-Ile-Lys-Asn-Ala-Tyr-Lys-Lys-Gly-Glu
Dermorphin	Tyr-D-Ala-Phe-Gly-Tyr-Pro-Ser-NH_2

their use as antidiarrheal agents since they cannot be made soluble and injected and are therefore less likely to be abused.

Together, these structure activity studies reveal that the basic requirements needed for opioid activity are quite simple. However, the wide variety of structures becomes even more intriguing since morphine and the other opioids act within the brain by mimicking naturally occurring peptides—the endogenous opioids. The enkephalins were the first such naturally occurring substances to be isolated and sequenced (Table 2). Initially, these results were somewhat confusing since the two enkephalins—both pentapeptides—contain the identical first four amino acids and differ only at the fifth. The complexity of these peptides became more clear with the subsequent isolation and characterization of β-endorphin, a 31 amino acid peptide derived from a larger protein, which also gives rise to active compounds, including ACTH and α-MSH. The first five amino acids in β-endorphin are identical to [met^5]enkephalin, but [met]enkephalin and β-endorphin derive from different gene products. There are also a series of compounds containing the sequence of [Leu^5]enkephalin, including dynorphin A, dynorphin B and α-neoendorphin. All these compounds (the ENKEPHALINS, ENDORPHINS, and dymorphine) have distinct genes and are expressed independently from one another. Thus, they comprise a family of similar, but discrete NEUROTRANSMITTERS.

The opioid peptides are only now becoming important clinically. A major difficulty in the use of peptides is the fact that they are broken down when taken by mouth, and thus, most have very limited oral activity. However, new derivatives specifically designed to be more stable have been developed, which will provide new leads. The enkephalins are potent at delta receptors, and many of their derivatives are delta-selective. Some of the more recent derivatives label delta receptors more than 10,000-fold more selectively than others. Yet other peptides are very much like morphine in terms of their pharmacology and receptor binding. Finally, peptides with opioid actions are now being identified in a variety of other tissues; for example, toad skin has dermorphin, a potent and stable opioid peptide.

(SEE ALSO: *Addiction: Concepts and Definitions; Opioid Complications and Withdrawal; Pain*)

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WILL OPIUM REAR ITS SMOKY HEAD YET AGAIN? (1999). *Psychopharmacology Update, 10*, 3.

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OPIOID COMPLICATIONS AND WITHDRAWAL Opioids are frequently used in medicine for pain relief. The most commonly used opioids include morphine sulfate (Duramorph, MS Contin, Roxanol); meperidine (Demerol); hydromorphone (Dilaudid); oxymorphone (Numorphan); methadone; codeine phosphate and codeine sulfate; oxycodone (Percocet, Percodan); and hydrocodone (Hycodan, Vicodin). These substances are also, however, among the most common drugs of abuse. When taken under medical supervision, opioid drugs have a low level of serious toxicity. The most common side effects are nausea, drowsiness, and constipation—but when self-administered, not under medical supervision, their use is associated with a high incidence of untoward actions and side effects, as well as with a high death rate when used alone or in combination with other drugs (including ALCOHOL).

Table 1 presents estimates of untoward actions of opioids, derived from data collected by the DRUG ABUSE WARNING NETWORK (DAWN), which appeared in the *Annual Emergency Room and Medical Examiner Data, 1992*. As can be seen,

opioids account for approximately 16 percent of emergency room and 64 percent of medical-examiner death reports. (Suspicious and accidental deaths are sent to the county medical examiner.) More than 76 percent of the medical-examiner opioid mentions involve death by opioid drugs in combination with either alcohol or COCAINE, whereas more than 20 percent occur in combination with other opioids. It is further estimated that about 67 percent of all such deaths were unintentional overdoses (ODs). Adverse results also occur in patients given opioids for therapeutic reasons, including, although uncommonly, serious respiratory depression.

RESPIRATORY DEPRESSION

It is generally believed that the most common life-threatening complication of opioid use, whether therapeutic or illicit, is respiratory depression (loss of the ability to breathe automatically). Probably the most important action of morphine-like drugs in producing respiratory depression is the lessening of the sensitivity and responsivity of the brain's medullary respiratory center to carbon dioxide (CO_2—the metabolic waste that circulates in the blood, derived from carbonic acid during animal respiration). Therefore, CO_2 becomes an inefficient respiratory stimulant, and automatic breathing ceases.

Administering a specific opioid ANTAGONIST such as NALOXONE to patients with severely depressed respiration frequently produces a dramatic increase in the rate of respiration and the volume of air taken in per breath. This occurs when a partial or completely resensitized respiratory center is confronted with high brain levels of CO_2. When the brain CO_2 levels are dissipated as a consequence of the evoked excessive rate and volume of breathing (hyperpnea), the minute volume (the volume of air breathed per minute) decreases. Yet when brain levels of the antagonist decrease, the respiratory depressant action of the opioid may assert itself again. Naloxone is a relatively short-acting antagonist. Patients who, for example, have received an overdose of long-acting opioids (e.g., METHADONE) have experienced a fatal respiratory depression following successful treatment with naloxone.

TOLERANCE AND PHYSICAL DEPENDENCE

Another group of complications associated with chronic use of opioids is the development of tolerance and dependence.

Tolerance. The most common concept of TOLERANCE to opioid drugs is that following chronic administration of a drug, its effects are diminished. Several mechanisms have been demonstrated to be involved in the development of tolerance to drugs, and these include (1) the induction of drug-metabolizing enzymes; (2) the development of coping strategies; (3) the exhaustion or depletion of NEUROTRANSMITTERS; and (4) an alteration in the number of active and inactive RECEPTORS. These mechanisms have, by and large, failed to provide adequate explanations for tolerance to opioid drugs. This may stem in part from the complexity of the results of chronic administration of opioids, the involvement of multiple mechanisms, and the influence of the dose, route, and frequency of drug administration.

Opioids, for example, alter the functioning of some body homeostats, and apparent tolerance is related to the establishment of new equilibrium conditions. This is clearly evident in respiratory depression, where opioids depress both the sensitivity and the reactivity of the brain-stem respiratory CO_2 homeostat, causing CO_2 to be a less effective respiratory stimulant. Yet when CO_2 accumulates because of depressed respiration, the increasingly higher concentrations will cause stimulation of respiration to the degree that the altered homeostat dictates. The ability of opioids to constrict pupils is dose-related, and patients receiving opioids frequently have miosis—near-maximally constricted pupils; hence, it is difficult to determine if tolerance develops to opioids' miotic effect. This has given rise to the commonly accepted view that tolerance does not develop to the miotic effects of opioids.

In former opioid addicts, morphine-like drugs produce dose-related feelings of enhanced self-image, of being more efficient and effective, and of well-being. These related subjective states form the essence of opioid-induced euphoria, which is produced in patients who are plagued by feelings of inadequacy. This can be quantitatively measured using the Morphine-Benzedrine Group scale of the Addiction Research Center Inventory.

Tests in many normal subjects (nonabusers) who are not suffering from pain indicate that opioids do not produce euphoria—but in sufficiently large doses instead produce feelings of apathy and ineffectiveness, which can be dispiriting (dysphoric). When opioids are administered chronically to addicts, the subjective effects they produce change from feelings of well-being to feelings of being withdrawn, tired, and weak. With regard to these effects of chronic opioid administration, they are not simply diminished but rather changed.

The development of tolerance can be a problem when opioids are used in the treatment of pain. Although some degree of tolerance to ANALGESIC effects is expected when opioid drugs are used repeatedly, in practice there is a great deal of variability among patients. Some patients with CANCER pain appear to derive satisfactory relief from the same dose of MORPHINE or similar drugs over a period of many months. For these patients, a need to increase the dose can be a signal that the disease is progressing. Other patients with terminal disease can develop remarkable tolerance. There are reports of patients who have been given the equivalent of 1000 milligrams of morphine per hour intravenously. This is an impressively large dose, since the usual starting therapeutic doses of morphine are 10 to 15 milligrams by injection every 4 to 6 hours, and doses of more than 60 milligrams by injection can cause potentially fatal respiratory depression in nontolerant individuals. It is not usually of much benefit to change to another opioid that acts at the same receptor. For example, morphine acts at the mu-opioid receptor. When tolerance develops to morphine, other opioids acting at mu receptors will be less effective, a phenomenon referred to as "cross-tolerance."

Physical Dependence/Withdrawal. Closely related to the phenomenon of tolerance is the phenomenon of physical dependence. Subjects given repeated doses of opioid agonists exhibit a syndrome when the drug is withheld or when the subject is administered an opioid antagonist. The resulting group of signs and symptoms is called the WITHDRAWAL or precipitated abstinence syndrome; subjects who exhibit an abstinence syndrome are termed *physically dependent* on the opioid. The degree of physical dependence and the intensity of the abstinence syndrome are related to the dose of the opioid agonist chronically ingested. In general, the intensity of all signs and symptoms covary together.

The abstinence syndrome includes restlessness, weakness, chills, body and joint pains, gastrointestinal cramps, anorexia (loss of appetite), nausea, feelings of inefficiency, and social withdrawal. Signs of abstinence include activation of the autonomic nervous system, lacrimation (tearing eyes), rhinorrhea (running nose), piloerection (gooseflesh), tachypnea (rapid breathing), mydriasis (dilated pupils), hypertension (high blood pressure), tachycardia (rapid heart beat), muscle spasms, twitching, restlessness, vomiting, and diarrhea. The waves of gooseflesh that occur during severe opioid withdrawal reminded some observers of the look of a plucked "cold turkey," a term that has come to be used not only for any abrupt discontinuation of a drug, but also for sudden cessation of any habit or pattern of behavior. The twitching and kicking movements of the lower extremities that can occur during opioid withdrawal have given the English language another widely used term, "kicking the habit," to denote the process of giving up any pattern of behavior or drug use.

The time of onset of opioid abstinence depends on the length of activity for the dependence-producing opioid. The abstinence syndrome of subjects dependent on morphine or HEROIN is well developed within 24 hours after the last dose of the opioid, peaks after 48 hours of abstinence, and gradually subsides thereafter. Signs of abstinence in patients dependent on METHADONE begin to emerge 24 to 48 hours after the last dose and may not peak for 2 weeks.

After this early abstinence syndrome subsides, a protracted abstinence syndrome emerges. The protracted abstinence syndrome becomes manifest 5 to 10 weeks after acute or early withdrawal in humans. It differs from the early abstinence syndrome in some ways but not in others. In subjects who were dependent on morphine or methadone, protracted abstinence is characterized by the following signs: a modest hypotension (low blood pressure), bradycardia (low heart rate), hypothermia (lower than normal body temperature), miosis (small, constricted pupils), and tachypnea. Other signs of protracted abstinence may include an inability to concentrate and a decrease in fine-motor control. Symptoms associated with protracted abstinence in patients who were dependent on methadone in-

clude feelings of tiredness and weakness, withdrawal from society, inefficiency, decreased popularity and competitiveness, and loss of self-control. Patients withdrawn from methadone have also exhibited a significant elevation of the Sc (schizophrenia) scale of the MINNESOTA MULTIPHASIC PERSONALITY INVENTORY (MMPI). This elevation of the Sc scale may be related to feelings of social withdrawal that patients in protracted abstinence experience. Protracted abstinence persists for at least 25 weeks after withdrawal. Protracted abstinence following addiction to morphine has also been demonstrated in rats and in dogs.

The patterns of abstinence and time course of symptoms described above are those seen when opioid drugs that have been used for weeks or months are discontinued. However, opioid withdrawal can also be observed when a drug-dependent person is given an opioid antagonist (a drug such as naloxone that competes with opioid agonists for the opioid receptor). In a matter of minutes, this will produce a precipitated abstinence syndrome that can be severe, with vomiting, cramps, and diarrhea. This precipitated abstinence is usually brief, however, because as soon as the antagonist is metabolized (usually less than an hour for naloxone), the opioids still in the body can again attach to the opioid receptors and suppress the abstinence syndrome.

The biological mechanisms that are responsible for the development of opioid physical dependence are set into motion with the very first doses of an opioid drug. If volunteer subjects are given standard doses of morphine (15 to 30 mg) and then, after an interval varying from 6 to 24 hours, they are given naloxone, they report nausea and other feelings of dysphoria and exhibit yawning, dilated pupils, tearing, sweating, and runny nose. Changes in endocrine levels are also seen that are in the same direction, although not as extreme, as those seen when chronically administered opioids are abruptly discontinued.

TREATMENT OF OPIOID WITHDRAWAL (DETOXIFICATION)

The opioid withdrawal syndrome varies in severity depending on the amount of opioid used and the duration of use. For the average user of illicit opioids, withdrawal is rarely severe because the amount of drug used typically is not high. The withdrawal syndrome from such a level of use can be uncomfortable, but it is not life-threatening in otherwise healthy individuals. However, death can occur if severe withdrawal is left untreated in individuals who are weakened by other medical conditions.

The process of treating someone who is physically dependent so that acute withdrawal symptoms are controlled and the state of physical dependence is ended is usually referred to as detoxification. For opioid drugs, this process can be managed on an ambulatory (outpatient) basis or in a hospital or other residential (inpatient) setting. The most common approach to easing the severity of opioid withdrawal is to slowly lower the dose of opioid over a period of days or weeks. However, in the United States, if the drug has been heroin, a substitution technique is used instead. Since virtually all opioids that are abused act as AGONISTS at the mu-opioid RECEPTOR, any mu agonist could be a suitable substitute, but the only ones approved for this purpose in the United States are methadone and LAAM (L-ALPHA ACETYLMETHADOL). These medical agents are effective when taken by mouth. Methadone can completely suppress the opioid abstinence syndrome. This capacity of one opioid to prevent the manifestations of physical dependence from another is called cross-dependence.

Outpatient detoxification using methadone typically involves using doses of 20 to 40 milligrams per day for a few days and then gradually reducing the dose over several weeks. Because so many patients return to illicit drug use as the dose of methadone approaches zero, government regulations controlling methadone permit a long period (up to 180 days) of slow dose reduction.

When detoxification takes place in a hospital or other residential setting, where the patient is presumably not as likely to be exposed to environmental cues that elicit CRAVING for opioids, dose reductions of methadone can be more rapid (e.g., over 8 to 10 days), although the intensiy of discomfort will be higher.

Other opioid agonists and partial agonists that have been used satisfactorily to facilitate detoxification include BUPRENORPHINE, (Buprenex) a partial mu agonist, and LAAM (Levomethadyl acetate). The opioid withdrawal syndrome can also be modified and reduced in severity by using agents that do not act at the mu receptors, but instead act on some of the physiological systems that exhibit

hyperactivity as part of the syndrome. The use of CLONIDINE (Catapres) is an example.

The opiate antagonist NALTREXONE (Trexan) can be used to detoxify patients rapidly and to help detoxified addicts stay off opioids. Naltrexone binds more strongly than heroin to the specific brain receptors to which heroin binds. The withdrawal is usually more severe than that which comes from simply stopping the heroin, but it also has the effect of detoxifying more quickly. Thus, a combination treatment of clonidine to suppress the intensity of withdrawal symptoms and naltrexone to accelerate the pace of withdrawal has been used for rapid detoxification.

Because opioid withdrawal is time-limited and rarely life-threatening, many nonmedical treatments have also been used, including ACUPUNCTURE and herbal medicines. Another nonmedical treatment that has been used in addicts is transcutaneous electrical nerve stimulation (TENS). It is thought that both acupuncture and TENS may be helpful because they stimulate the parts of the central nervous system that release natural opioids. At present, further research is needed because opioid addicts are very suggestible and may feel better after acupuncture or TENS because of the placebo effect.

NAUSEA AND VOMITING

Nausea and vomiting are common side effects associated with the use of opioid analgesics. These effects are experienced following administration of opioids orally, by injection, or by injection into the spinal canal (epidurally)—they are worsened by movement and the resulting stimulation of the vestibular (inner ear organ responsible for balance). The site and mechanism responsible for these actions of opioids is presumed to be a special area in the brain stem or medulla, the chemoreceptive trigger zone of the area postrema.

CONSTIPATION

Constipation, an often undesirable effect of opioids, is sometimes a useful effect for which opioids can be prescribed. It is undesirable when opioids are used for the relief of pain and in opioid-dependence maintenance therapy.

The oldest of the therapeutic actions of opiates is their antidiarrheal and constipating effects. It is now known that the extrinsic innervation (nerves leading from the central nervous system to the gut) and the intrinsic innervation (nerves within the wall) of the gastrointestinal (GI) tract are complex and vary from species to species. A variety of naturally occurring neurones with diverse neurotransmitters have been identified, including neurones and their process that contain opioid peptides: the enkephalins, B-endorphin, dynorphins, and other ligands derived from pro-opiomelanocortin. Further mu and delta opioid receptors have been identified in the GI tract. The vagus nerve also has fibers that contain enkephalins, and the central nervous system has opioid mechanisms that modulate GI movement (motility).

Several influences must play a role in the constipating effects of opiate agonists—these include increased segmental activity, decreased propulsive activity, and decreased secretory activity. Naloxone, even when administered in high doses for a long period of time in antagonist therapy of opioid abusers, does not produce an overt stimulation of the GI tract resulting in diarrhea. When opioid antagonists are administered to opioid-dependent subjects, however, GI cramps and diarrhea develop as classic opioid withdrawal signs.

PRURITUS

The ability of morphine-like drugs to produce the sensation of itching (pruritus) is well known, and it is a discomforting complication when opioids are administered for therapeutic reasons. Further, many morphine-like drugs (e.g., codeine) release histamine from white blood cells that store it (mast cells and basophils). When morphine is administered intravenously, wheals (hives—raised red lumps) may appear at the site of the injection and along the course of the vein. The wheals may be associated with the sensation of itching. Occasionally, large doses of morphine may produce generalized itching. Rarely does morphine produce pulmonary edema (fluid in the air sacs of the lung), bronchoconstriction (narrowing of the air tubes in the lungs), or wheezing. With the advent of the use of intrathecal and epidural morphine (injection of morphine into spinal fluid or around the lining of the spinal canal) in pain management, the incidence of morphine-induced pruritus has become greater. Under this circumstance, the distribution of itching may be segmental (limited to the part of the spinal

cord involved). Itching remains an elusive phenomenon and is harder to define and investigate than pain. It is thought that it may be mediated by a subgroup of nociceptive (pain-carrying) C fibers. Further, morphine's histamine-releasing property has been implicated in its ability to produce itching, as histamine does in allergic reactions.

CONVULSIONS

Although most opiates produce convulsions when administered in very large doses, convulsions are most frequently observed when excessively large doses of MEPERIDINE (Demerol) or *d*-propoxyphene (Darvon) are administered. Emergent meperidine seizures are characterized by tremors and twitching, which may evolve into tonic-clonic (epileptic) convulsions. Focal and tonic-clonic seizures have been observed in patients overdosed with *d*-propoxyphene. The mechanisms whereby opioid drugs produce convulsive phenomena are not well understood and may involve several mechanisms, including (1) direct and indirect dysinhibition of glycine and GABA-mediated inhibition and (2) excitatory actions that are probably mediated by yet-to-be-classified receptors. The convulsant effects of *d*-propoxyphene can be readily antagonized by naloxone; however, meperidine's convulsant effects may be more resistant. Meperidine probably has a convulsant effect in its own right when administered in very large doses acutely, yet convulsant phenomena seen following the administration of multiple doses of meperidine are produced by the accumulation of a metabolite, normeperidine.

DYSPHORIA, DELUSIONS, AND HALLUCINATIONS

It is rare for morphine-like analgesics to produce psychotic reactions. In patients with severe pain and discomfort and in opiate addicts, single doses of morphine-like drugs most commonly produce feelings of well-being. In normal subjects with no pain or with only modest levels of discomfort, morphine produces feelings of apathy and enervation, which are somewhat dysphoric. The drug *d*-propoxyphene (Darvon) has been reported to produce bizarre reactions—delusions and hallucinations—particularly when taken chronically in large doses and when used to suppress opioid abstinence. Some agonists-antagonists (e.g., pentazocine [Talwin], nalorphine, and cyclazocine) produce feelings of apathetic sedation, perceptual distortions, anxiety, delusion, and hallucinations.

STREET DRUGS

The complications described in the preceding sections are most commonly associated with pure, unadulterated opioids. When street drugs are used, which are typically diluted by the seller with quinine, lactose, or other powdered materials—and injected by the user in an unhygienic manner, in doses that vary significantly—the range of complications widens. These are described fully in the entry on neurological complications, but among the complications of heroin use reported in the medical literature are strokes, inflammation of cerebral (brain) blood vessels, toxic amblyopia, bacterial meningitis, aneurysms and brain abscesses, disorders of peripheral nerves, impairment of segments of the spinal cord, and widespread injury to muscle tissue (rhabdomyolysis)—which by releasing muscle protein can denote damage to the kidneys.

OTHER MEDICAL COMPLICATIONS

Medical complications of opioid addiction may result from unsanitary administration of the drug, from overdosing, from intoxicated behavior (e.g., unsafe sex), or from the chemical properties of opioids themselves.

Lungs. Opioid addiction may lead to pneumonia, aspiration pneumonitis, lung abscess, or septic emboli in the lungs. It also decreases the vital capacity and diffusion capacity of lung tissue. Opioid addicts who also smoke tobacco are at increased risk of lung infections.

Liver. Opioid addicts frequently develop viral hepatitis (types A, B, and C). In addition, addicts who are also heavy drinkers have a high incidence of cirrhosis and other disorders of liver function.

Immune System. Hypergammaglobulinemia (an abnormally high level of gamma globulin in the blood) develops in about 90 percent of opioid addicts. As of 1999, it is unclear whether this change in the immune system is caused by infections or by daily injections of foreign substances. It diminishes in addicts on methadone maintenance. In addition to hypergammaglobulinemia, opioid addicts are at

a very high risk of contracting HIV infection from shared needles.

Muscles and Bones. Osteomyelitis (inflammation of bone and the bone marrow caused by bacterial infection) is a common complication of opioid addiction. Drug abuser's elbow is a complication in which the muscles of the lower arm are damaged by repeated needle punctures and tears.

Skin and Lymphatic System. Opioid addicts frequently develop skin abscesses and ulcerated areas from injecting heroin under the skin ("skin popping"). Using contaminated needles may result in cellulitis, lymphangitis, lymphadenitis, and phlebitis (inflammation of a major vein).

Pregnancy and Lactation. Infants of opioid-addicted mothers are born physically dependent on the drug, because both heroin and methadone cross the placental barrier. They may also acquire HIV infection or hepatitis from an infected mother. Pregnant addicts should be encouraged to enter a methadone maintenance program rather than attempt complete withdrawal, because withdrawal in the last trimester of pregnancy may cause early labor. Mothers on methadone maintenance can nurse infants without harm to the child, because breast milk will not contain large amounts of the methadone.

(SEE ALSO: *Addiction: Concepts and Definitions*)

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OPIOID DEPENDENCE: COURSE OF THE DISORDER OVER TIME Opioid dependence is the modern diagnostic term for narcotic addiction, but the older term is still often used. This entry, however, uses the modern term. The term *opioid* refers to natural and synthetic substances that have morphine-like effects. The term *opiate* is generally used in a more restricted sense to refer to MORPHINE, HEROIN, CODEINE, and similar drugs derived from OPIUM. OPIOID dependence is defined as a cluster of symptoms related to continued use of an opioid drug. One of the prominent features of the disorder is the inability to stop using the drug. Persons with repeated periods of opioid dependence are often called narcotic addicts. Because they are not always dependent (that is, addicted), the term *opioid users* seems more suitable and therefore is used here. During the late nineteenth and early twentieth centuries the principal opioid drugs used were LAUDANUM (a solution of opium in alcohol, taken orally) and morphine (usually injected by needle). During the latter half of the twentieth century, heroin has been the principal drug of opioid users. It is usually taken by intravenous injection, but sometimes by insufflation, that is, by sniffing it into the nasal cavities.

The course of opioid dependence is affected by multiple interacting conditions in the person and in the environment. The combined conditions create thresholds for the onset, continuation, and relapse after remission of opioid dependence. Different methods of investigation (for example, pharmacological, psychological, sociological, psychiatric) have led to different theoretical conceptions of the causal conditions and processes in opioid dependence. These conceptions, however, tend to be compatible and supplementary rather than contradictory. In the following description of the course of opioid dependence, the principal conditions thought to affect its onset and course will be identified.

In the United States, legal and medical conditions affecting opioid use and dependence have changed since the nineteenth century. In the nineteenth century many persons regularly used laudanum or morphine that they obtained legally from physicians, retail drug stores, or other sources. Physicians often prescribed or recommended these drugs for treatment of chronic physical PAIN or psychological distress. Although daily use of an opioid drug with consequent dependence on it probably impaired the social performance of many persons, reports exist of persons—including some with distinguished careers—who acceptably filled social roles during years of opioid drug dependence. Though some antisocial persons used opioid drugs, such use itself did not lead to criminal behavior.

In the twentieth century, opioid dependence became closely associated with criminal behavior. Enactment and enforcement of federal and state laws to control the production and distribution of opioid drugs (mostly called narcotic drugs in the laws) became prominent features of the twentieth-century environment of opioid use. Physicians could no longer prescribe opioid drugs to maintain dependence, and opioid users now had to obtain their drugs from illicit sources. Furthermore, because the illicit opioid drugs were expensive, users often engaged in illegal moneymaking activities—especially theft, burglary, fraud, prostitution, and illicit drug traffic—to pay for their drugs. In addition, twentieth-century opioid users have often had histories of delinquent behavior that preceded their opioid use.

WHO IS SUSCEPTIBLE?

At the turn of the century, when opioid drugs were legally and easily available to all adults, only a few persons became dependent on them. Although the exact scale of opioid dependence at that time is not known, it probably did not exceed 2 percent of the adult population. An interview survey conducted in the 1970s of a national sample of young men in the United States revealed that 5.9 percent had used heroin at some time in their lives, but only 1.7 percent ever considered themselves dependent on this drug (O'Donnell et al., 1976). Other studies indicate that normal people free from physical pain tend to react to the effects of opioid drugs with indifference or dislike. With rare exceptions, patients who receive opioid drugs to relieve pain after surgery make no effort to continue drug use after they become free from pain. It is now well-known that opioid dependence develops in only a small proportion of those exposed to the effects of the drugs.

The characteristics of persons susceptible to opioid dependence have not been clearly defined, but clinical and other studies point to three personality

problems that probably increase susceptibility. First, chronic emotional distress, such as DEPRESSION, tension, ANXIETY, anger, or mixtures of these, is relieved by opioid drugs, and this relief probably prompts repeated use of the drug. Second, impaired capacity to regulate emotional distress increases the urgency of the need for relief. Third, an antisocial attitude makes it easy for the person to perform the illegal actions needed for regular illicit opioid use. The notion that opioid drugs are used to relieve emotional distress is called the self-medication hypothesis. The origins of the personality problems that increase susceptibility probably lie partly in genetic inheritance and partly in adverse psychosocial experience. Modern opioid users often come from dysfunctional parental families.

Environmental conditions in the deteriorated areas of large cities in the United States place young persons living there at special risk for opioid dependence. Most of the retail illicit drug traffic and much of the opioid use takes place in these areas. Young persons are consequently exposed to available heroin and heroin-using role models and associated criminal behavior. Since these areas are heavily populated by minority groups—primarily African Americans, Puerto Ricans, and Mexican Americans—these groups are at special risk. The experience of POVERTY and adverse discrimination may contribute to emotional distress in members of these groups and thereby increase their susceptibility to opioid dependence. Apart from environmental conditions, ethnic status as such does not seem to affect susceptibility. Men seem to develop opioid dependence more often than women do; the ratio of men to women in treatment programs is about three to one.

ONSET OF OPIOID DEPENDENCE

Opioid use is usually preceded by use of tobacco, ALCOHOL, and MARIJUANA. Before their first opioid use, most users dropped out of school and began to associate with opioid users. Heroin is nearly always the drug of choice. With few exceptions, it is first used within a few years of the user's twentieth birthday. Users report that they were not coerced or urged to use heroin by either their associates or drug dealers. In a typical sequence a person becomes aware of drug use by his friends or relatives, becomes curious about its effects, and asks for the first injection. As already noted, most persons exposed to the effects of heroin do not become regular users.

Susceptible persons rarely become compulsive daily users immediately after first use. A variable period of occasional use—once a month or more often, but not daily—usually ensues. Curiosity fades as a motivation; the effects of the drug are what prompt repeated use. The drug users call these effects the "high." The high is not described as exhilaration or excitement but rather as relaxation and mood elevation. Descriptions of the high offered by many drug users suggest that it amounts to relief of the chronic emotional distress mentioned before as a factor in susceptibility. Susceptible persons increase the frequency of use until it reaches once or several times daily. From first use to daily use typically takes about one year, but it may take much longer. In a study of opioid users in San Antonio, one man reported that he first used heroin at the age of sixteen. He did not like it and did not use it again for fifteen years. At that time he felt depressed following the death of a friend and decided to try heroin again. This time the heroin made him feel better, and he quickly became a daily user (Maddux & Desmond, 1981).

With daily or nearly daily use, the user develops physiological DEPENDENCE on the drug. This means that when the drug use is reduced or stopped, the user develops distressing symptoms called the WITHDRAWAL illness. The threat or the onset of withdrawal symptoms provides additional strong motivation to continue daily use of the drug.

In the progression from initial use to daily use, heroin users learn how to inject heroin intravenously, how to acquire the drug and injection equipment, and with some exceptions, how to conduct illegal moneymaking activities to pay for the heroin. Those who began a delinquent career before their initial use of heroin were already oriented toward criminal activity. In some cases, heroin users or dealers provide a regular supply of heroin to their spouses or live-in companions; the latter thus do not have to engage in regular illegal activity to pay for their drug. Another exception to the pattern of illegal moneymaking activity is linked to opioid dependence among physicians and other health professionals. Health professionals rarely purchase heroin from street retailers. They have access to meperidine or other opioids available in pharmacies and hospital supplies, and they use these drugs instead of heroin.

Probably the most serious and disabling feature of opioid dependence is the inability to put a stop to it, also called loss of control of the drug use. After drug use has become daily and physiological dependence has developed, many opioid users try to stop using it and find themselves unable to do so. This inability to stop is a subjective mental state reported by drug users. It probably starts as a mild impairment of control and progresses to complete or nearly complete loss of control.

EARLY TERMINATION OF OPIOID DEPENDENCE

Continued daily use with loss of control depends partly on the availability of the drug and other environmental conditions. American soldiers serving in VIETNAM during the Vietnam War were exposed to an environment in which heroin was easily available and heroin use was common. An interview survey of a sample of returning veterans revealed that 35 percent had tried heroin while in Vietnam and 19 percent (about half of those who tried it) considered themselves addicted to it. During a three-year period after return to the United States, however, only 12 percent of those addicted in Vietnam became readdicted in the United States. These represented about 2 percent of the entire sample interviewed (Robins et al., 1980). Other studies of early termination of opioid dependence in the United States have identified various life events as probable causative factors in the termination. Among these are change of residence, marriage, a drug-related arrest, and death of a friend from overdose. Many persons who terminate their opioid dependence do so without treatment.

CHRONICITY, REMISSION, AND RELAPSE

With continued daily use and physiological dependence, the user's bond to the drug becomes stronger. Drug use, drug seeking, and illegal activity become the dominant activities of the user's life. Psychosocial development is retarded. Those who become dependent during adolescence often fail to complete high school and never develop regular work habits or job skills. With continued dependence, opioid users become impaired marital partners or parents.

Daily use does not continue indefinitely. In some cases, as noted, an important life change leads to cessation of use. In other cases, pressure from family or friends or other sources prompts entry into a treatment program. In still others, arrest, conviction, and incarceration interrupt the daily use. Sometimes conviction leads to probation with treatment as a requirement of the probation. After treatment or incarceration, the majority of chronic users resume opioid use within six months. The common long-term pattern consists of initial use followed by irregular sequences and varied durations of occasional use, daily use, treatment, abstinence, and incarceration. Remissions enduring for three years or longer followed by relapse are not unusual. Variations in the course of opioid dependence are illustrated in the following case summaries.

> An employed man first used heroin at the age of twenty-six and after two months of occasional use began daily use. He continued working but engaged in the illicit heroin traffic to pay for his heroin. Two years after first use, he was arrested and convicted for sale of heroin. In lieu of prison, he was sent to a federal hospital for treatment. Released on parole at age twenty-nine, he remained abstinent for ten years, when he was last interviewed at age thirty-nine. He abstained from heroin, he said, because he did not want to return to "that miserable life."
>
> Shortly after release from an institution for delinquents, a boy had his first injection of heroin at the age of sixteen. He became a daily user in about three months. During the next thirteen years he had two brief periods (each of about five months' duration) of abstinence from heroin. He used heroin occasionally or daily during the remaining time, except for four years in prison. He was murdered by gunshot at the age of twenty-nine.
>
> After dropping out of school, a fourteen-year-old boy learned to make money by selling marijuana and heroin. He tried heroin at age sixteen, liked it, and promptly became a daily user. He used heroin daily for the next twenty years, except for relatively brief periods when he was in prisons and hospitals. Then, at age thirty-six, he was sent to prison for two years. During this period in prison, he felt some change in himself while participating in a THERAPEUTIC COMMUNITY program. After release, he abstained from heroin for the next

eight years. He obtained employment as a counselor in a drug-abuse treatment program. He was aged forty-six when last interviewed.

Modern TREATMENT of opioid dependence includes drug withdrawal done as an inpatient or outpatient procedure, residential treatment, therapeutic community, drug-free outpatient treatment, the use of opioid ANTAGONISTS, and METHADONE MAINTENANCE. Prompt abstinence from opioid drugs is the goal of the first five of these types of treatments. Methadone maintenance, in contrast, consists of continued substitution of methadone, itself an opioid drug, for the illicit opioid. In addition to these forms of treatment, self-help groups such as NARCOTICS ANONYMOUS are available as well as special religious programs for drug users.

Opioid users who enter treatment aimed at prompt abstinence reveal mixed motivations for the treatment. They would like to become free of the burden of their drug dependence, but they do not want to give up the effects of the drug. Most leave treatment before completing it. Relapse after treatment is common, but the severity of the dependence is usually reduced, short periods of abstinence are often achieved, and for a small proportion of users, enduring cures of opioid dependence are attained. Methadone maintenance aims for social rehabilitation, with opioid abstinence as a possible distant goal. It has become a major mode of treatment for chronic opioid users and benefits many of them by helping them reduce or stop illicit opioid use and stop their criminal activity. This treatment, however, only infrequently leads to enduring abstinence.

USE OF MULTIPLE SUBSTANCES

In the early twentieth century, many alcoholics were converted from ALCOHOLISM to opioid dependence. If the opioid dependence was terminated, alcohol dependence often replaced it. In the later twentieth century, the patterns of use of other psychoactive substances during the course of opioid dependence have become more complex. Heroin users often substitute alcohol when they become abstinent from opioids, but, in addition, many use alcohol regularly while using heroin daily. They also use TOBACCO, marijuana, and cocaine. In a recent interview study of opioid users in California, 75 percent reported current use of tobacco, 20 percent reported being drunk on alcohol in the previous seven days, 38 percent reported use of marijuana in the previous thirty days, and 18 percent reported use of cocaine in the previous thirty days (Hser, Anglin & Powers, 1993).

WHY DOES OPIOID DEPENDENCE BECOME INTRACTABLE TO TREATMENT?

This important question can be answered only partially and tentatively. The conditions that contribute to the onset of opiod dependence also support the tendency to continued use. These, as previously noted, include chronic emotional distress, drug-using models, an available opioid drug, and withdrawal symptoms. Two other effects of the drug dependence probably contribute to relapse after treatment or incarceration. First, mild withdrawal symptoms such as muscular aching, insomnia, and irritability often persist for six months or longer after the last dose. These symptoms (called protracted withdrawal) are promptly relieved by an opioid drug, and they probably contribute to relapse after treatment. Second, the opioid user becomes conditioned to environmental conditions associated with withdrawal symptoms, so that after a period of abstinence, exposure to a conditioned stimulus will evoke withdrawal symptoms. This conditioned withdrawal probably contributes to relapse.

Three other changes in the mental state of the user probably also contribute to the intractable quality of the disorder, but these have not been as well defined and studied. First, over time the drug-using habit tends to become automatic, requiring no conscious decision to use or abstain. Second, the drug-seeking and the associated criminal behavior seem to become a part of an established lifestyle, and the user becomes enmeshed in a social network that includes illicit drug users and criminals. Third, with repeated relapses after treatment or incarceration, the opioid user comes to a self-perception as an addict with a diminishing capacity for change. This complex of learned attitudes and behaviors amounts to a personality change, which is probably accompanied by change in the brain. Such change may not become permanent, but it tends to endure.

LONG-TERM OUTCOMES

In follow-up studies extending from five to more than twenty years after admission to treatment, the percentages of users reported abstinent from opioid drugs have varied from 9 percent to 21 percent (Maddux & Desmond, 1992). Some of this variation was due to different ways of counting abstinence. In some studies the users were counted as abstinent only if they remained so during the entire period from treatment to follow-up, whereas in others the users were counted as abstinent if they were found so at the time of follow-up. Despite these differences, the studies collectively indicate that only a minority of opioid users are found to be abstinent on long-term follow-up.

Although only small to medium percentages were found to be abstinent, it should not be assumed that the remainder of people were using opioid drugs. Some were dead, some were in prison, and some were in treatment. The death rate of opioid users is about three times the expected rate. Overdose, homicide, suicide, accidents, and liver disease account for many of the deaths. In the 1980s the acquired immunodeficiency syndrome appeared as an additional hazard for drug injectors. A follow-up of opioid users in San Antonio revealed the following different statuses twenty years after first use: 16 percent were abstinent, 29 percent were using heroin, 30 percent were in prison or other institutions, 8 percent were maintained on methadone, and the remaining 17 percent were dead or their status was unknown (Maddux & Desmond, 1981).

WHAT CAN BE DONE?

Since policies and programs to reduce drug abuse are described elsewhere in this encyclopedia, only a brief comment will be offered here. Two broad approaches—supply reduction and demand reduction—have been put in place in the United States. Supply reduction consists of the enactment and enforcement of drug control laws. Although the supply-reduction effort has undoubtedly reduced the supply of illicit opioid drugs, it has failed by far to eliminate them from the environment of susceptible persons.

Demand reduction consists of treatment and prevention. Treatment of opioid dependence produces short-term abstinence and reduces the pool of daily users in the community, but it achieves few enduring cures. Publicly supported treatment programs in the United States are insufficiently financed to provide prompt treatment to all who seek it. A few pilot projects have been developed for reaching out to young persons at risk for opioid dependence and providing special services for them, but more research is needed on this type of preventive effort. Finally, opioid use in the United States seems embedded in a complex matrix of family dysfunction, poverty, undereducation, unemployment, and crime. Anything that reduces these problems would likely reduce illicit opioid use. Easy solutions seem unlikely.

(SEE ALSO: *Addiction: Concepts and Definitions; Britain, Drug Use In; Causes of Substance Abuse; Coerced Treatment; Conduct Disorder and Drug Use; Crime and Drugs; Opioid Complications and Withdrawal; Opioids and Opioid Control: History; Vulnerability; Wikler's Pharmalogic Theory of Drug Addiction*)

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OPIOIDS AND OPIOID CONTROL: HISTORY Throughout recorded history and in most parts of the world, OPIATES have occupied a central place in medicinals. They have been used popularly against a wide range of ills and to produce calm or well-being. Opiates are renowned for their powerful ability to relieve PAIN. They also have been used for their PSYCHOACTIVE properties and, within the last 100 years, have come to symbolize the problems with attempts to control drug use through legislation and enforcement. (Technically, opiates are a subset of the OPIOIDS, which also include synthetic agents and naturally occurring peptides that bind to opioid RECEPTORS found in certain animal species.)

The OPIUM poppy (*Papaver somniferum*) grows easily in semiarid parts of the Middle East and southern Asia, including dry or steep locales where other crops are difficult to cultivate. For thousands of years, farmers in these regions have grown the poppy as an important staple crop. For traditional poppy farmers, opium is a cash crop that supplements an agricultural livelihood. The entire plant is used: Poppy seeds are baked into breads, or oil for cooking or fuel is extracted from them and the body of the plant is fed to cattle. The labor-intensive aspect of collecting the sap for sale means that whole families are pressed into service at harvest time. The desire for opium in other parts of the world has long made it an important commodity in international trade networks.

References to opium appear in inscriptions and texts of ancient Sumer, Egypt, and Greece. The Greek physician Galen, in the second century C.E., noted that opium cakes were widely sold in Rome. This observation highlights an important difference between drug use before the twentieth century and contemporary drug use. Currently, drug use is divided into medical and nonmedical (or recreational) uses. Nonmedical use for opiates is banned in most countries, and persistent demand fuels a large and vigorous illicit trade. Medical uses are defined exclusively by physicians, and consumption of these drugs is allowed only in the context of treatment by a physician.

The sharp separation between medical and nonmedical uses of drugs is comparatively new in human history, although attempts to control drug use legislatively are not. In the past, physicians constituted only a small group of specially trained professionals who found their clientele primarily among the rich and powerful. A wide range of healers provided different kinds of health care; for example, in Europe from the Middle Ages to about the mid-nineteenth century, apothecaries prepared and sold drugs to anyone seeking treatment. Apothecaries consulted with the patient, helping diagnose an ailment and suggesting a remedy, but they charged a fee only for the sale of the drug.

Opium became an important European drug in the sixteenth century. During the Middle Ages, the severing of ties between Europe and the Middle East meant that large amounts of opium were not shipped to Europe. In the Middle East, however, the ancient Roman and Greek texts remained important sources of knowledge, and medical, as well as scientific and mathematical, theories were developed and debated among scholars like the Arab physician Avicenna. In these Moslem countries, where alcohol was absolutely forbidden, both opium and cannabis were widely used.

During the European Renaissance, renewed ties with the Middle East brought the ancient texts and their Arab interpretations to the attention of Euro-

pean scholars. Galen, who had systematized humoral theory in his writings, was recognized as an important authority in sixteenth- and seventeenth-century Europe. Galen's views were challenged by the sixteenth-century Swiss physician Paracelsus, who favored chemical remedies (such as mercury) to herbal ones. Paracelsus valued opium highly. He devised a mixture of alcohol, opium, and other ingredients that he called "laudanum" (from the Latin for "praise") to suggest its superiority.

Thomas Sydenham, the influential English physician, wrote in 1680: "Among the remedies which it has pleased Almighty God to give to man to relieve his sufferings, none is so universal and so efficacious as opium." This valuation of opium (and later of its derivatives) has been repeated by physicians in the centuries since as ongoing testimony to the drug's central role in medical treatment.

The medical use of opium grew more widespread in eighteenth-century England; for example, the relief of pain at the time of death was seen as an important adjunct to preparing the patient for death in a blessed state of peace. England was an important commercial power in this period, and new kinds of goods from distant parts of the world became increasingly plentiful. Opium was a valuable commodity, and, as such, it was handled commercially like any other. Individuals seeking to treat themselves for aches or ailments, or wanting to relieve drudgery or sleeplessness or persistent coughs, could buy pellets of opium from various merchants, innkeepers, or apothecaries. This pattern persisted through most of the nineteenth century, although by the late eighteenth century a particular effect of chronic opium consumption was described: If a habitual user stopped taking the drug, a clearly recognizable syndrome of symptoms ensued. These included runny nose, tearing, sweating, aches, muscle tremor, vomiting, and diarrhea. These problems were seen as an expected difficulty connected with taking medicines; they were not portrayed as a unique and devastating kind of problem that threatened the social fabric.

In the United States, also, opiates were freely sold. In the first half of the nineteenth century, neither medications nor medical practice were regulated. During the presidency of Andrew Jackson, antimonopolistic sentiment had led many states to repeal licensing requirements for physicians, on the grounds that such licenses created artificial elites. Many people saw no physician at all; they treated themselves or their family members with homemade or purchased remedies. Taking charge of one's own medical care also reflected the kind of broadened democratic spirit that characterized the Jacksonian age. In home treatment, opiates were valued for their wide-ranging effects, including quick and dramatic improvement in how one felt. Physicians also administered opium generously as part of the heroic brand of therapy favored in the nineteenth century. Based on humoral theory, "heroic therapy" sought to provide clear evidence of its effects on body fluids by promoting fluid discharges. Emetics and cathartics were the hallmarks of such practice, but the ability of opiates to produce sweat in addition to their other valuable effects made them a component of heroic therapy.

For individuals who appeared chronically weak, perhaps as the result of lingering fever, opium improved spirits and energy and was considered by many medical practitioners to have a STIMULANT effect (although it is now classed as a DEPRESSANT). Individuals who took the drug to relieve vague feelings of unease, or in the absence of serious medical conditions, were said to take the drug for its stimulant properties.

Rapid industrialization caused profound social shifts in England in the first half of the nineteenth century. People whose families had worked on the land for generations became part of the first large-scale factory work force. Working conditions were brutal; men, women, and children worked 14 hour days, 6 days a week. Working women often had to bring young children to the factory with them. For working people, opium was an easily available source of relief for many complaints of both adults and children.

Early in the nineteenth century, Thomas De Quincey and Samuel Taylor Coleridge wrote about opium-induced reveries. Although their works were widely read, their opium use was treated more as a curiosity than a cause for alarm. The earliest concerns about excessive or indiscriminate opiate use centered on adulteration or on deaths due to accidental OVERDOSE. These were voiced by a new group of professionals, public health workers. Extensive surveys of health conditions in England in the 1840s both highlighted problems and created opportunities for government and professional workers to expand their professional arenas. At the

same time, the old three-rank system of health-care givers, in which physicians treated the well-to-do while surgeons and apothecaries met the health needs of those of more modest means, was giving way. Surgeons and physicians joined a unified healing profession, whereas pharmacists prepared and sold drugs without providing diagnostic or therapeutic advice. As physicians worked to increase their professional authority, they sought to gain control over the use of drugs, defining them as medicines that only the medically trained could use or prescribe with safety. Toward the middle of the nineteenth century, a few physicians expressed concern about opium use for its "stimulant" effects. These voices foreshadowed an alarm about nonmedical use of opiates that would transform how this behavior was viewed. In the meantime, the 1868 Pharmacy Act called for precise labeling of any preparation containing opium.

The incidence of addiction also worried some observers, and this phenomenon became increasingly visible in part as a result of new pharmacological discoveries and changing medical technology. In 1806, Frederich Sertürner of Hannover, Germany, announced that he had isolated the chief active component of opium. He named this new drug MORPHINE, after Morpheus, the Greek god of dreams. Morphine was the first drug compound to be isolated from the plant that contained it, and as such it marked the first step in the development of scientific pharmacology. Drug effects could not be precisely described and measured until individual compounds were isolated. The isolation of CODEINE followed in 1832. In time, the systematic modification of the molecular structure of such compounds would be an important source of new medications and the basis of the modern pharmaceutical industry.

In the 1840s, the invention of the hypodermic syringe provided a new means of administering drugs. Morphine was among the first drugs to be administered by syringe, and the immediate introduction of the dose into the bloodstream provided stronger and faster drug effects than by swallowing and digesting the drug.

During the American Civil War (1861–1865), the combination of the more potent morphine, the hypodermic syringe, and wartime conditions contributed to widespread hypodermic morphine use. Large numbers of wounded soldiers and relatively few physicians meant that many soldiers were given syringes and supplies of morphine to treat their own pain. Many soldiers inevitably became addicted. Following the war, some of these soldiers phased out opiate use as their wounds healed, while others continued their pattern of morphine use for years. In the postwar period of industrial and commercial expansion, a wide variety of preparations containing opium were sold through vigorous advertising in an unregulated market. Physicians prescribed opiates, including morphine and codeine, for a wide variety of conditions. Many preparations were advertised specifically for women's health problems or for children bothered by colic or teething pain.

After 1850, Chinese laborers were brought to the American West to work on railroad building and other forms of gang work. As they moved away from these forms of labor, some Chinese took up placer mining in the Sierra Nevada or settled in Pacific coast cities like San Francisco. There, as white laborers sought to exclude them from the labor market, many opened and operated small businesses. The Chinese brought with them the practice of smoking opium to induce a 2 to 3 hour state of dreamy relaxation. Prejudice against Chinese people was based largely on fears that they would displace white laborers by accepting wages that white people considered to be below subsistence level; this prejudice focused on Chinese customs such as opium smoking. The U.S. Congress passed several laws in the 1880s to reduce the importation of opium intended for smoking into the United States.

In 1898, the Bayer company of Germany began marketing the newly trademarked drug Heroin, produced by modifying the morphine molecule. At first, HEROIN was valued for its apparent ability to cure morphine addiction; a dose of heroin quickly relieved all symptoms associated with morphine withdrawal. Within a few years, heroin's addictiveness was recognized and physicians stopped prescribing it, despite its effectiveness in relieving pain and coughing.

For many who became addicted through self-medication, addiction was a source of shame of which they could not free themselves. They sought treatment in privately run clinics that promised anonymity and offered little more than a place to rest while they went through withdrawal; or they purchased purported cures that, in fact, merely contained more opiates. Others continued to take

opium or morphine and managed their jobs or other responsibilities as long as their drug supply remained uninterrupted. The initial response to rising rates of addiction was to blame unscrupulous medicine merchants and physicians who administered opiates too readily.

In the United States, the concerns about adulteration, overdose, and addiction associated with an unregulated drug market became acute around the turn of the twentieth century. In the context of Progressive Era reform, the 1906 Pure Food and Drug Act required that any medication containing opiates state their presence and the amounts on the label.

In both the United States and England, what is now called recreational use of drugs emerged around the 1890s. People began taking opiates for pleasure, or to provide a novel experience, in a social setting with no medical overtones. Rising alarm about drug use as a particularly dangerous kind of social problem dates from this period, which also saw the rising political power of the Temperance Movement and its efforts to enact a total prohibition on the use of alcohol. Unfamiliar drug-use practices provided an additional focus for social anxieties in a time of rapid economic change. A Protestant middleclass ethos helped burgeoning new groups of professionals and business people adjust to new kinds of economic opportunity in an industrial age. Behaviors that challenged that ethos with pleasure seeking, new modes of entertainment, and unfamiliar druguse practices proved disturbing.

In the 1890s in England and the United States, small numbers of artists and bohemians, seeking to challenge what they saw as restrictive Victorian artistic and social standards, visited Chinese opium dens where they learned to smoke opium. For some Chinese in London or Liverpool, opium smoking provided a means of relaxation from a life of hard work in an alien land. As the existence of opium dens became more widely known, however, images of ghostlike, numb pipe smokers began to appear in popular literature. The middle- and upper-class pleasure seekers who smoked opium prompted a compassionate response, but British working-class use of opium was viewed as an indication of laziness, poor child-rearing habits, or loose morals.

In the United States, the 1880s and 1890s brought waves of new immigrants from southern and eastern Europe—and they brought new customs to the American cities they settled. By the early 1900s, sniffing heroin, for example, had become a practice of some young adults in urban neighborhoods crowded with large immigrant families or for some single adults making their way alone in a new industrial setting.

In this drawing from the Illustrated London News, *July 1857, workers at Hong Kong harbor transfer bales of opium from one ship to another for export to the West.* (© CORBIS)

Rising concern about opiate use in this period was only partly a reaction to incidence of opiate addiction, which, with alcoholism, was classed as a psychiatric condition called inebriety. In the late nineteenth century, many troubling conditions were redefined as diseases, especially as forms of psychopathology, and opiate addiction was among them, although many physicians even decades later saw addiction as resulting from a moral failing.

Worldwide missionary activity also resulted in concerns about opiate addiction. Christian missionaries in China and the Philippines, for example, believed that opiate addiction among the local populations helped explain what they perceived as economic backwardness. Like some temperance advocates in the United States, reformers concerned about addiction portrayed it as a form of slavery that followed a collapse of moral will. In such a framework, opiate addiction appeared as a scourge to be eradicated. Between 1911 and 1914 reformers met at The Hague to urge worldwide control of opiate supplies so as to prevent any nonmedical use of the drugs. Some countries joined in signing and ratifying a treaty that marked the first attempt to develop a coordinated international system for controlling worldwide opiate supplies. The U.S. representatives to these meetings were

embarrassed by the lack of any U.S. legislation for controlling access to opiates. A lobbying effort to bring U.S. legislation into line with the goals of The Hague resolutions led to passage of the Harrison Narcotics Act in 1914, the first U.S. law enacted to control who could buy a drug. The act banned sale of opiates and cocaine except for use by physicians or through a doctor's prescription. The American Medical Association (AMA), sensitive to charges that physicians' overprescribing of opiates was the chief cause of addiction, supported the legislation.

Following implementation of the HARRISON NARCOTICS ACT in 1915, health authorities in several American cities were worried that the sudden lack of opiate supplies for addicted individuals would create great personal stress and a possible public crisis. They opened clinics that were intended to dispense opiates to addicts so that they would not go suddenly into withdrawal when legal supplies were cut off. In many cases, the mission of a clinic was unclear: Were patients expected to reduce their doses gradually and wean themselves off opiates, or would some be permitted to continue to maintain their addiction by means of opiates supplied through the clinics? The U.S. Treasury Department, charged with enforcing the Harrison Act, moved vigorously to enforce it by charging certain physicians with the excessive prescription of opiates and by arguing in court cases that the act specifically disallowed addiction maintenance. In 1919, the Supreme Court ruled that the Harrison Act meant that physicians could not prescribe opiates to addicts except as part of a shortterm program of detoxification. Again, the AMA agreed. Armed with this legal support, the Treasury Department continued its enforcement activities against the maintenance clinics, and by the mid–1920s all had been closed.

The Harrison Act was envisioned by its proponents as part of a planned worldwide system of treaties in which each country that imported opiates would allow only the amounts needed for medical treatment to cross its borders. Opium-producing countries and the European countries where, in this period, most of the world's opium was refined into drugs like morphine or codeine would also cooperate to limit supplies of the drug. This approach to drug control has characterized the drug policies of most countries ever since.

Meanwhile, morphine and heroin use became part of a new urban social scene that included new kinds of entertainment. Concerns about opiate addiction shifted from compassion for innocent victims of improper medication to alarm about new centers of vice in urban neighborhoods. Inner cities became populated with groups whose social and political behaviors worried some business leaders, middle-class reformers, and workers who felt their jobs were threatened.

The passage of the Harrison Act was followed by the creation of federal enforcement bodies to prohibit unauthorized entry of opiates into the country, and to arrest and convict unauthorized sellers and possessors of opiates. In the 1920s, psychiatric theory held that chronic addicts suffered from personality deficits that caused them to feel inordinate pleasure from opiates and thus become mired in addiction. Opiate addiction was now viewed as both a medical and a criminal problem. The creation of the Federal Bureau of Narcotics in 1930, and the appointment of HARRY J. ANSLINGER as its head, moved drug enforcement out of the Prohibition Unit that oversaw enforcement of the Volstead Act. Following repeal of alcohol prohibition in 1933, the Federal Bureau of Narcotics continued to carry out the enforcement of the prohibition of opiates and cocaine. Anslinger was a skillful administrator with a background in diplomatic service. He oversaw American participation in the activities of the League of Nations' Opium Advisory Committee, which furthered the work on international control of opium supplies that had been initiated through the Hague Opium Treaty. On the domestic front, Anslinger managed an efficient team of nationwide enforcement officials. Believing that harsh and early punishment would be effective deterrents, he supported increasingly severe punishments for drug offenders, including mandatory minimum sentences for first offenders. For decades, the "drug problem" remained in the background of public consciousness as a kind of exotic problem associated with a city world of jazz, marijuana, and beatniks, but the threat carried enough symbolic weight to cause penalties for drug trafficking and possession to be stiffened in 1951 and again in 1956. Anslinger remained the U.S. government's chief drug-enforcewment official until his retirement in 1962, when, in both medical and legal circles, a new generation of observers were urging less punitive responses to drug offenses and greater emphasis on medical approaches to treating addicts.

The British approach to controlling opiate use in the twentieth century proceeded along a policy basis that was different from that of the American approach, despite some similarities in legislation. The Dangerous Drugs Act of 1920, like the American Harrison Act, restricted the use of opiates to legitimate medical needs. However, the British government did not seek to define the limits of those medical needs. The government-appointed ROLLESTON Committee, which met in 1924, recommended that addiction be regarded as an illness to be treated by physicians. Reacting in part to perceived difficulties in enforcing America's prohibitions of both alcohol and opiates in that period, the Rolleston committee members sought to avoid stimulating an illicit market by banning opiates. Rather, they favored allowing individual physicians to prescribe opiates to selected addicts—that is, they recommended a policy of addiction maintenance. British policy was also conditioned by the demographics of opiate use in Britain, which differed from patterns in the United States. In Britain, opiate use continued to be associated with affluent bohemianism and those addicted through legally prescribed medication, and the powerful stigma against addicts that characterized the American scene did not develop to the same degree in Britain. In such an atmosphere, nonpunitive policies appeared appropriate.

In the 1960s, startling new patterns of drug use brought the issue to mainstream consciousness in the United States and throughout Western Europe. Since the nineteenth century, the leaders of American reform efforts aiming to curb drug use had typically couched their rhetoric as concern about use patterns among specific population groups—foreigners (as in opium use by Chinese people) or the working class. Now, illicit drugs were typically being used by young, white, and middle-class persons.

Events of the 1960s prompted a generation of young people raised during the prosperous 1950s to question the ideals of the relatively calm and affluent world that they knew. These events included the ongoing civil-rights movement, the assassinations of President John F. Kennedy, Martin Luther King, Jr., and presidential candidate Robert F. Kennedy, and the escalating war in VIETNAM. As they questioned and challenged the establishment, young people disregarded old prohibitionist messages about illicit drugs; at the same time that they sought to forge new values, they also hoped they could eliminate the superficial and hypocritical aspects in American life. MARIJUANA and psychedelic drugs most closely symbolized the new spirit, but young people buying drugs on the illicit market and sharing lore about highs also encountered amphetamines and opiates.

For the young men who went to Vietnam to fight the war, the ready availability of heroin provided one possible avenue of escape from the horrors some of them experienced and witnessed daily (although boredom was often reported as a common motive for use). Southeast Asia remained an important source for the world heroin market, even more so as the trade from Turkey through southern France became hampered by enforcement activity. It was relatively easy for many returning veterans to stop using heroin once they returned to the United States. The men came back, however, after fighting a losing war to a United States deeply divided over the conflict. Receiving little welcome, many veterans had difficulty in readjusting to civilian life; for some of these, continued drug use remained part of a web of problems made up of chronic medical conditions or difficulties in finding work, although opiate use specifically was remarkably uncommon.

In 1972, President Richard M. Nixon was reelected on a platform that included bringing an end to the war and responding to growing American fears about crime. He united these concerns by increasing enforcement resources directed against drug use. In 1971, Nixon had proposed the most significant federal drug-policy initiatives since the passage of the Harrison Anti-Narcotic Act of 1914. He announced the creation of the Special Action Office for Drug Abuse Prevention (SAODAP). This office, administratively located in the White House and headed by Jerome H. Jaffe, M.D., led an expanded federal funding for drug treatment and special programs to identify and treat addicted soldiers returning from Vietnam. Jaffe had been director of an innovative program in Illinois that offered a range of treatment services, including methadone maintenance. The previous U.S. policy toward opiate addiction, which placed emphasis on law enforcement, was for a time replaced by one that emphasized concern for treatment and prevention in addition to control of the drug supply. Beginning in 1963 in New York, Vincent Dole and Marie Nyswander had demonstrated that longtime heroin

users, stabilized on daily doses of oral methadone and supported with a range of rehabilitative services, showed reduced criminal activity and improved functioning in social and employment areas. Nixon came to believe that methadone maintenance would provide a cost-effective means of reducing the money-seeking crimes committed by street addicts. Previously viewed as an experimental treatment, methadone maintenance, though subjected to special regulations, was made an accepted element in the treatment of opiate addiction. In the same legislation that created the Special Action Office, Congress included language that authorized the formation of the National Institute on Drug Abuse to coordinate federal funding of treatment services and research on drug abuse.

Meanwhile, in the 1970s, under federal leadership, treatment programs were expanded and new ones created in cities across the United States. Increasingly, those running the programs encountered patients who did not fit the model of the criminally involved longtime heroin addict. Younger patients, more women, and those using a variety of drugs reflected changing U.S. drug-use patterns. As these patterns were recognized, opiates ceased to dominate images of drug abuse in both the popular mind and in policy circles. Rather, opiates became just one group among many that were traded on the illicit market and used for philosophical, lifestyle, political, recreational, and even habitual reasons.

The CONTROLLED SUBSTANCES ACT of 1970, also passed at Nixon's initiative, reformulated how drugs were assigned legal status. The act created five schedules for categorizing psychoactive drugs, ranging from those considered to have no medical use and high risk of abuse to those having important medical use and only a mild risk of abuse potential.

In Britain, as in the United States, drug users in the 1960s and 70s experimented with a growing range of drugs besides opiates. New patterns of chronic drug use, new, flamboyant behaviors symbolized by the lives of celebrities and rock stars, and a sharp escalation in the absolute numbers of heroin addicts prompted some divisions in Britain's medical community about the wisdom of continuing Britain's nonpunitive maintenance policy toward opiate addiction. Some physicians became unwilling to treat addicts, whereas others remained committed to a purely medical approach to addiction with maintenance as an important component of the policy. In 1968 new laws were passed that limited the role of the general physician in the prescribing of heroin and that established a system of clinics supervised by specialists.

The early 1980s advent of ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) has added a new dimension of concern about drug use by injection, the preferred mode of administration of many heroin users. Because sharing used syringes can transfer the human immunodeficiency virus (HIV) from one person to another, drug use by injection has been named a high-risk behavior for its transmission.

In the late 1990s, heroin addiction once again is escalating and has moved from center city shooting galleries and dope houses (places people gather to use drugs) to more middle-class neighborhoods. There has also been a change in the ways people use heroin. Indeed, these changes in use patterns and user groups are comparable to those last seen during the Vietnam-era epidemic of the late 1960s and 70s. The so-called new heroin users are younger, smokers and snorters.

The new millennia heroin user is much *less* likely to start out injecting heroin. Snorting and smoking heroin is not, however, without inherent health risks. Heroin snorters risk neurologic complications, respiratory infections, and problems associated with other forms of heroin use, such as dependence, withdrawal, and vulnerability to future injection drug use and its associated diseases. Heroin smokers share these same health risks plus the added problem of respiratory infections through "shotgunning" or inhaling smoke and then exhaling it into another individual's mouth. This practice has the potential for the efficient transmission of respiratory pathogens, particularly tuberculosis.

Most of these young heroin users move on to injection drug use at some point in their drug using careers. In the absence of an effective treatment or vaccine, efforts to control the spread of HIV and hepatitis C (HCV) infections depend on reducing risk behaviors. Public health interventions have taken the form of prevention campaigns employing the media, educational groups or seminars, and street outreach workers. However, we also know that knowledge of health risks is not enough to help injection drug users to change their behaviors.

The availability of drug-using paraphenalia and the problems associated with finding clean and sterile equipment play a major role in disease transmission. One response has been to reduce the sharing of paraphernalia through the creation of needle exchange programs that distribute sterile needles and syringes, as well as other drug-using equipment. Assessments of the impact of such programs, in Australia, Europe, and in the United States, suggest that syringe exchanges play an important and significant role in reducing the rates of sharing for drug-using equipment.

All modes of heroin ingestion increase heroin users' vulnerability to hepatitis infection through the sharing of drug-using equipment (e.g., needles, straws, pipes, receptacles to cook or mix drugs). The spread of HIV/AIDS, hepatitis, tuberculosis, and other pathogens and infections among youthful drug-using populations poses not only serious public health threats, but also potentially large increases in public and private health-care costs.

Opiates remain important medication for the treatment of pain, cough, and diarrhea. Recent discoveries that opiates achieve their effects by mimicking compounds occurring naturally in the body (e.g., ENDORPHINS and ENKEPHALINS) have spurred exciting neuroscience resesearch about how the brain works. After millennia of use, then, opiates continue to be one of the most interesting classes of drugs.

(SEE ALSO: *Asia, Drug Use in; Britain, Drug Use in; Chinese Americans, Alcohol and Drug Use among; Dover's Powder; Shanghai Opium Conference; Terry and Pellens Study*)

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REVISED BY SHEIGLA MURPHY

OPIUM The milky juice derived from the unripe seed capsules of the poppy plant *Papaver somniferum* is called opium. This material, which dries to a brownish gum contains a large number of alkaloid compounds. These ALKALOIDS can be categorized into two major groups—the benzylisoquinolines and the phenanthrenes. The phenanthrene group includes the OPIOIDS, the most important of which is MORPHINE, which constitutes approximately 10 percent of opium. CODEINE is present in far smaller quantities, at 0.5 percent, and thebaine is only 0.2 percent. Both morphine and codeine can be extracted from opium and each crystallized to yield pure compounds. Virtually all morphine is derived from opium, since to synthesize it is complex and expensive. Although morphine and codeine have been used extensively in the clinical treatment of PAIN, thebaine is equally important—it is the starting material for the synthesis of many semi-synthetic opioid analgesics (painkillers). Of these, the most widely used include oxymorphone, oxycodone, and naloxone. Thebaine, itself, has no opioidlike effects.

Opium has a long history of use and abuse. It was initially used for the treatment of diarrhea and then for the relief of pain. Today, opium still has a number of medicinal uses, primarily as tincture of opium, a concentrated alcoholic extract of opium. Although this is occasionally used for extreme diarrhea, most physicians prescribe paregoric, a camphorated opium-tincture preparation containing approximately 0.4 milligrams per milliliter of morphine in 45 percent alcohol. The concentration of morphine in paregoric is far smaller than in opium tincture, so doses are adjusted accordingly. Doses that effectively treat diarrhea typically do not cause

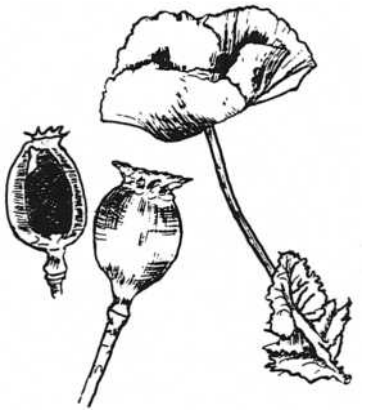

Figure 1
Opium Poppy and Pod

euphoria or analgesia—however, excessive doses can be abused and can lead to dependence.

History. The plant grows wild in the Middle East—especially in the Turkish plateau region—and has been known and used since antiquity. Opium was introduced into India by Arab traders of the thirteenth century. By the seventeenth century, along with the spread of TOBACCO use, the Chinese had devised a method of smoking opium—using small sticky balls of opium gum in opium pipes. It is said that by 1900, about 25 percent of the Chinese smoked opium, although it was banned by the emperor. This high level of use was the result of the British East India Company's practice, beginning in the mid-eighteenth century, of shipping opium to China from their conquered lands around Bengal (1750)—one of the major opium-producing areas of the subcontinent. Export of opium to China helped balance the company's trade deficit, caused by tea purchases. After 1780, opium was produced as a monopoly by the company.

China was at that time basically closed to all kinds of outside trade, except for certain port cities, where special concessions were granted by the emperor. Indian opium was auctioned to British traders in Calcutta, who carried it to Southeast Asia and China, often by way of shippers and smugglers off the South China coast and the islands there, including Hong Kong. British concern for the security of their opium trade led to the colonization of Malaysia and Singapore and, eventually, to the Opium War of 1840–1843, with China, where the emperor's troops were outmaneuvered. The series of treaties that ended that conflict "opened" China to trade with the West and to European political and economic domination.

Suppression of the trade began with the concerns of Protestant missionaries and physicians in China—which outweighed the concerns of the emperor for keeping his people producing for him, not enslaved by opium dreams. International bodies were formed in the late nineteenth and early twentieth centuries to restrict the opium trade, but the British refused to move toward any kind of regulation until 1905. The international conferences and conventions of 1909, 1915, and 1930 led to the restriction and prohibition of traffic in opium and opium derivatives—morphine, codeine, and heroin.

In the United States, opium abuse is not anywhere near the problem of HEROIN abuse, as of the early 1990s. Opium smoking and opium eating are the two major forms of abuse. Some immigrants to the United States have brought these customs with them, but on a small scale. When smoked, opium is prepared by heating it over a flame until a small ball of roasted opium gum is formed. The ball is then pushed into a pipe, where it is held over either a flame or a coal and smoked. Opium eating is widely practiced in India and in other countries where the opium poppy is cultivated—Turkey, Afghanistan, Southeast Asia, and so on. It is used as a household remedy for pain and other ailments, much as it has been for hundreds of years. Approximately 50 percent of opium eaters in India, for example, use it for medicinal purposes, taken as a pill or as a solution.

While the legitimate opium trade had slowed by the 1930s, illegal production continued in several places. In Southeast Asia, colonial governments drew revenues from opium monopolies until 1942, when the invading Japanese suppressed it during World War II. With the victory of the Communists in China in 1949–50, steps were taken to eradicate the growing of opium and its use. By 1960, opium production was confined to a few isolated areas of Burma, Laos, and Thailand. During the VIETNAM War, various tribal peoples were encouraged to grow opium by a number of politically motivated groups, resulting in the establishment of the GOLDEN TRIANGLE as one of the major centers of illegal opium production.

(SEE ALSO: *Asia, Drug Use in; Shanghai Opium Conference*)

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OVERDOSE, DRUG (OD) Administration of a drug in a quantity that exceeds that which the body can metabolize or excrete before toxicity develops constitutes an overdose. Whether it is accidental or deliberate, drug overdose is a significant problem that is encountered by providers of emergency medical care. Accidental overdose is common among users of illegal substances of abuse, since little reliability can be placed on the potency, presence of adulterants, and even identity of the street substance. For example, HEROIN potency has been demonstrated to range from 3 to 90 percent. Overdoses and deaths from heroin are therefore common. The prevalence of comorbid disorders in substance-abusing populations, particularly DEPRESSION, has been found to be high. Thus, deliberate drug overdoses taken in the attempt to commit SUICIDE are frequently encountered in this population. Also, people with a psychiatric illness but no drug-abuse problem most often attempt suicide with a drug overdose. Substances frequently implicated in drug overdose involve nonnarcotic ANALGESICS (painkillers), BENZODIAZEPINES (tranquilizers), OPIATES, or ANTIDEPRESSANTS—often in combination with alcohol.

The treatment of a drug overdose begins by providing basic supportive care (i.e., ensuring that there is adequate ventilation and monitoring the heart), calling 911, an emergency medical service (EMS), or the Poison Control Center (see Appendix I in Volume 4). If little time has elapsed since ingestion, efforts may be made to prevent further absorption of the drug by such means as gastric lavage or by administration of activated charcoal. Other treatments include increasing the rate of excretion through forced diuresis or giving specific antidotes (e.g., NALOXONE for opiate overdose) when the substance is known or can be identified from the presenting clinical syndrome. Obtaining a careful drug history from the patient or accompanying individuals is of paramount importance in effectively treating and minimizing risks from a drug overdose, which often results in death.

(SEE ALSO: *Drug Abuse Warning Network; Drug Interactions and Alcohol*)

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KAREN PARKER

OVEREATING AND OTHER EXCESSIVE BEHAVIORS Overeating, a behavior not always limited to persons with BULIMIA, is grouped together with substance abuse and dependence in a superfamily of disorders designated as behavioral (non-substance-related) addictions. The term *impulse control disorders* has been used by some clinicians to describe these behaviors. In this context the notion of ADDICTION centers on the repetitiveness of the behavior and would include such behaviors as compulsive spending, compulsive gambling, pathological overeating (bulimia), hypersexuality, kleptomania (repetitive, compulsive stealing when there is no need), as well as miscellaneous obsessive-compulsive behaviors such as tics and hair-pulling (trichotillomania). Some researchers have pointed out similarities among these disorders and believe that there may be similar brain mechanisms involved in some of them. For example, it has been shown that DOPAMINE levels in certain areas of the brain (such as NUCLEUS ACCUMBENS) are elevated by the ingestion of reinforcing drugs including COCAINE, AMPHETAMINES, OPIOIDS, and, to some degree, NICOTINE. However, increased dopamine levels in these same brain circuits have been shown to occur when animals an-

ticipate food or sexual activity. Also, learning, conditioning, and reinforcement play important roles in these repetitive behavior disorders as well as in the more traditional chemical or substance-abuse and -dependence disorders. It has also been pointed out that treatments for nonchemical "addictive" disorders often follow principles used in substance-abuse disorders; for example, identifying trigger and high-risk situations, teaching alternative coping behaviors, and emphasizing relapse prevention. Self-help groups using AA principles have also been organized, such as Overeaters Anonymous or Gamblers Anonymous. Some pharmacologic agents appear to alter both drug ingestion and obsessive-compulsive behaviors that are not drug related. For example, SEROTONIN UPTAKE INHIBITORS, now used as ANTIDEPRESSANT medications, seem to help alcoholics decrease alcohol consumption and compulsive hair-pullers reduce that behavior.

Such broad definitions of addictive behaviors have disadvantages when they focus too much attention on the commonalities among the diverse behaviors while minimizing the differences and particularities. At a time when rapid progress is occurring in the understanding of the biological processes associated with substance dependence, focusing only on commonalities may obscure the value of therapeutic interventions aimed at specific disorders. For example, nicotine transdermal patches seem to have considerable value in treating tobacco dependence but are probably of no value for cocaine dependence or compulsive gambling.

The way society (or science) chooses to categorize behaviors—desirable or undesirable, repetitive or episodic—is determined in large measure by the objectives of developing the categorization. There are probably some circumstances where it is helpful to think about a broad category of problematic excessive behaviors encompassing everything from substance abuse to television watching. There is also the risk that in doing so we convey the notion that excessive drug use is no more serious or refractory to intervention than watching television or jogging. Certainly at the present time the social costs and medical consequences of the substance-use disorders are so great that we should be cautious about embracing any conceptual scheme that tends to trivialize or make these problems seem less serious than they are.

(SEE ALSO: *Addiction: Concepts and Definitions; Adjunctive Drug Taking; Causes of Substance Abuse: Learning; Obesity; Research, Animal Model: An Overview of Drug Abuse*)

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OVER-THE-COUNTER (OTC) MEDICATION This class of medication can be purchased without a prescription. Which medications require prescriptions and which do not varies widely from country to country. Common examples of OTC medications in the United States include ANALGESICS (aspirin, Tylenol®), cough and cold products (Sinutab®, Drixoral®), allergy medications (Benadryl®, Tavist), gastrointestinal products (Maalox®), antidiarrheals (Imodium®), and nicotine replacements (e.g., Nicorette® Gum, Nicoderm® Patch). Recently, a number of medications that previously were sold only by prescription have been made available over-the-counter. These include medications that block the production of gastric acid to relieve heartburn (Axid AR®, Tagamet HB 200®, Zantac 75®) and nicotine gum (Nicorette CQ®) and the nicotine patch (Nicotrol®, Nicoderm CQ®) for smoking cessation.

Prescription medications are labeled with patient-specific instructions determined by a physician whereas OTC products provide general information for use by consumers. OTC products *are drugs*, and as such they may cause side effects or adverse effects, or they may interact adversely with foods, ALCOHOL, or other medications. Some of the

A drugstore clerk in Deerfield, Illinois removes Tylenol capsules from the shelves after reports of tampering, February 18, 1986. (© Roger Ressmeyer/CORBIS)

more than 500,000 OTC products that are available have the potential to be misused or abused. Antihistamines, hypnotics, decongestants, analgesics, laxatives, and diet pills are often consumed in higher than recommended quantities; they have caused physical and/or psychological dependence. An epidemic of the early 1990s among adolescents has been "baby speed," the combining of OTC CAFFEINE pills with the decongestant pills pseudoephedrine. Handfuls cost only a few dollars and are responsible for overstimulating the heart and central nervous system, causing strokes and death.

An estimated 28 percent of adults in the United States use all kinds of OTC products, often responsibly but also in combination with prescription medications or alcohol. The high cost of visits to a physician and stays in a hospital has generated heightened interest in self-medication, which has increased opportunities for pharmacists to counsel patients. This situation is also contributing to the increased availability of medications as products are transferred from prescription to OTC status. The legislation that controls OTC products is quite recent. It was in 1951 that the United States first separated drugs into the two categories—prescription and OTC. A drug that is available only on prescription cannot be made available as an OTC product until its relative safety and efficacy have been reviewed by the U.S. Food and Drug Administration.

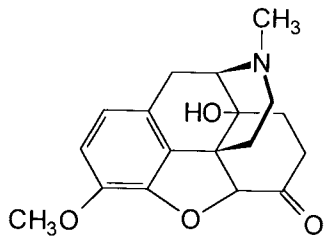

Figure 1
Oxycodone

(SEE ALSO: *Drug Interactions and Alcohol; Legal Regulation of Drugs and Alcohol*)

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MYROSLAVA ROMACH
KAREN PARKER

OXFORD HOUSE *See* Treatment Programs/Centers/Organizations: An Historical Perspective

OXYCODONE Oxycodone is one of the most widely used OPIOID ANALGESICS in the United States, and it is usually used in conjunction with the analgesics aspirin or acetominophen. The combinations have proven effective and are, in some ways, superior to oxycodone alone, since they permit a lower dose of the opioid—and are therefore less likely to produce constipation, drowsiness, and nausea. Oxycodone is a derivative of OXYMORPHONE, the relationship being the same as that between CODEINE and MORPHINE. Like codeine, oxycodone is metabolized to oxymorphone, which is assumed to be responsible for its activity. Pharmacologically, the actions of oxycodone and oxymorphone are quite similar to those of morphine, so toxicity and ADDICTION can occur.

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GAVRIL W. PASTERNAK

OXYMORPHONE Oxymorphone is a potent semisynthetic OPIOID ANALGESIC derived from thebaine, one of the twenty ALKALOIDS occurring naturally in OPIUM. It is approximately fivefold more potent than MORPHINE and has very similar actions and side effects. It is used to treat moderate to severe PAIN. Oral formulations are not available in the United States, but it is available by injection or by rectal suppository. Like morphine, continued use of oxymorphone leads to TOLERANCE AND PHYSICAL DEPENDENCE. It is interesting that oxymorphone shares the same basic chemical structure as the ANTAGONISTS NALOXONE and NALTREXONE, the only difference being the substituent on the nitrogen. Neither naloxone nor naltrexone have analgesic activity; in contrast to oxymorphone, they are instead capable of blocking opiate actions.

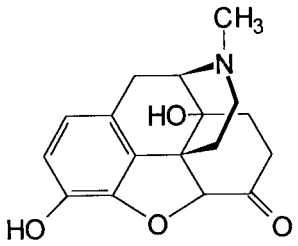

Figure 1
Oxymorphone

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GAVRIL W. PASTERNAK

P-Q

PAIN: BEHAVIORAL METHODS FOR MEASURING ANALGESIC EFFECTS OF DRUGS Pain is a sensation produced by potentially harmful stimuli, such as intense heat, stretching, cutting, or chemical irritation. The ways in which information about these stimuli is carried to the brain and the interpretation that results are very complex. Pain sometimes occurs in the absence of a harmful stimulus, such as in phantom-limb pain (where the limb has long been missing). In other instances, pain is not even felt, although harmful stimuli are present. Thus pain is both a sensation and a response to that sensation. The response to pain can vary depending on the individual and the circumstances. Given this complexity, it is not surprising that pain can be modified in many ways—by a variety of drugs, by hypnosis, and by stimulation such as acupuncture.

PAIN TRANSMISSION

The transmission of pain involves two systems—an ascending and a descending neural system. Ascending neural systems carry information about potentially harmful stimuli from peripheral nerves to the spinal cord and from there to the brain, where information about the emotional and psychological aspects of painful stimuli is incorporated. In addition, the perception of painful stimuli is altered by descending neural systems, which send information from the brain back to the spinal cord. Pain transmission can be altered at any point in this loop. Drugs such as aspirin (an analgesic) relieve pain by reducing pain sensitivity in the periphery. Local anesthetics such as lidocaine (Xylocaine) and procaine (Novocaine) relieve pain by blocking nerve conduction in specific areas. Morphine and other opioids (narcotics) alter pain transmission by interfering with the processing of painful stimuli in the spinal cord and the brain.

MORPHINE AND OTHER OPIOIDS IN HUMAN PATIENTS

Among all the drugs that relieve pain, opium and its derivative morphine, are certainly the best known. When morphine is given to patients who are experiencing severe pain, they often say the pain is less intense or that it no longer exists. Other patients say the pain is still present, but it just does not bother them. Thus, morphine affects both the sensation of pain and the patient's response to the painful stimulus. It is generally believed that morphine acts in both the spinal cord and the brain. In the spinal cord, morphine inhibits the flow of information about painful stimuli from the spinal cord to the brain. In the brain, morphine alters pain perception by modifying activity in the descending pain-control system. In addition to relieving pain, morphine-like drugs produce a sense of pleasure (or euphoria) in some patients. Morphine and other opioids are the most effective drugs known for the relief of pain. Although their usefulness is sometimes limited by the fact that they can produce DE-

PENDENCE, this is generally not a problem in clinical settings.

NONOPIOID ANALGESICS

Although the opioids are considered the most effective drugs for the treatment of pain, THC (delta^9-TETRAHYDROCANNABINOL), the active constituent of MARIJUANA, has some pain-relieving properties, but it is not as effective as morphine in this respect. Very large doses of drugs such as ALCOHOL and the BARBITURATES also appear to relieve pain; however, these effects do not represent true analgesia, since they only occur at doses of alcohol and the barbiturates that produce a loss of consciousness. Thus, the organism's lack of response to painful stimuli is simply an inability to respond.

STUDIES IN LABORATORY ANIMALS

To determine whether a newly-developed compound has pain-relieving properties, scientists use behavioral procedures developed in laboratory animals. In general, these procedures measure the time it takes an organism to respond to a painful stimulus, first when no drug is present and then after a drug is given. Morphine and other opioids consistently alter this and other measures of pain perception. For example, morphine increases the time it takes an animal to remove its tail from a warm water bath, as illustrated in Figure 1. It takes about 2 seconds for the monkey to remove its tail from a warm water bath if morphine is not given. A small amount of morphine increases tail-removal time to about 8 seconds; larger amounts of morphine increase the time to as much as 20 seconds. Modification of pain perception also depends on the intensity of the painful stimulus. If the water in the bath is very hot, only very large amounts of morphine will increase the time it takes animals to withdraw their tail, whereas a lesser amount of morphine will increase response time at lower temperatures. Similarly, some drugs such as BUPRENORPHINE are most effective in relieving pain when the pain is mild. Since buprenorphine also produces less dependence than morphine, it may be a very useful drug for treating mild forms of pain. By combining data about the pain-relieving effects of a drug with data about its likelihood to produce dependence, information is obtained about the usefulness of a new drug in a clinical setting.

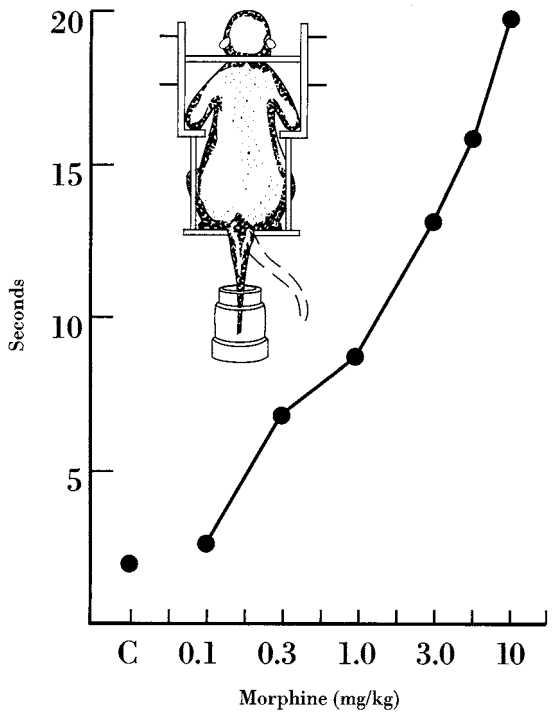

Figure 1
Pain Perception

(SEE ALSO: *Addiction: Concepts and Definitions; Pain: Drugs Used for; Opiates/Opioids*)

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LINDA DYKSTRA

PAIN, DRUGS USED FOR Pain is a sensation unique to an individual. Its perception depends on the injury involved and the situation or context. A bruise obtained in a football game may not be appreciated at the time of the injury, yet in other circumstances the pain from a minor injury, such as stubbing a toe, may be overwhelming. The extent of the injury does not predict the amount of pain experienced; it is this wide variability that makes the treatment of pain difficult.

Within the brain, there are two systems that can appreciate the sensation of pain. One deals with the objective component and tells the exact location of the injury and what type of injury it is. The other is more diffuse and comprises the "hurt." Many people have experienced both types of pain. Touching a hot object or stubbing a toe is quickly followed by the appreciation that an injury has occurred, followed an instant later by the pain. It is this second pain that contains the "suffering," the "hurt," and the elimination of this second pain is the goal of ANALGESIC therapy.

Physicians have divided pains into three general categories. The first, and most common, is termed somatic pain. This results from tissue injury, such as a broken leg, metastases in the bone from cancer, muscle pulls, or ligament sprains. The second is termed visceral pain, which results from activation of pain fibers in internal organs, typically in the abdomen or chest. This category includes discomfort associated with gall bladder disease, peptic ulcers, or pancreatitis, to name a few. Unlike somatic pain, visceral pain is poorly localized. The most difficult pain to understand and to treat is deafferentation, or neuropathic pain, which is a consequence of injury to nerves. It is difficult for patients to describe these sensations, but they often use terms such as "burning," "shooting," or "electriclike." This type of pain is commonly seen in cancer patients where tumors invade nerve bundles. It also is seen with mild damage to nerves. The most common class of injury is the peripheral neuropathies. This collection of disorders results from a wide variety of causes; it affects nerves as they course through the body. The longest nerves are most sensitive to injury, which explains why this type of pain is most likely to develop in the feet. Diabetes is one of the most common causes. A special type of pain also falling in this classification is postherpetic neuralgia, a burning and/or shooting pain associated with *Herpes zoster*, known as shingles.

When considering pain, it is important to classify it as either acute (short-term) or chronic (long-term). The duration of many kinds of pain can be anticipated. The acute pain associated with surgery is usually limited in duration and, over the period of several days, decreases markedly. In contrast, the chronic pain associated with disseminated cancer can often be severe and persistent, actually increasing over time. Acute pain is associated with a number of very specific symptoms that are usually recognized by others—making it relatively easy to be believed. Patients may be pale and sweaty, the heart may be beating rapidly, and they may be grimacing. Chronic pain is different; it is usually defined as pain that persists for six months or longer. Many of the signs we see acutely wear off during this time, despite the continued pain, leading some observers to conclude that the pain is minimal or even absent; this conclusion is incorrect and often leads to undertreatment and therefore unnecessary suffering. Despite the sophistication of modern medicine, the most accurate estimate of pain remains simply to ask the patient. Chronic pain may seem to have no cause, at times, may be difficult to evaluate or treat, and often requires specialists. Special pain clinics exist for such cases.

Pain medicines (analgesics) are often broken into three major groups. The first group comprises the most commonly used drugs—aspirin, acetaminophen, and related compounds; these drugs are effective for mild to moderate pain. The second group include the OPIOIDS (OPIATES). Some opioids are used for moderate pain while others are typically employed for more severe pain. Thirdly, there are a number of drugs used either for specific pain syndromes or in conjunction with the first two groups. The agents in this last group are termed adjuvant drugs.

The choice of analgesic is based on both the type of pain and its intensity. Most pain is treated in a standardized fashion. Initial therapy often utilizes aspirin, ibuprofen, or acetaminophen. These agents are available without prescription and can be very effective for mild to moderate types of pain. They have a number of properties that make them excellent analgesics. Their effectiveness against a wide variety of different types of pain and their oral dosage greatly enhance their utility. Unfortunately, these agents exhibit relatively low ceiling effects. This means that the maximal degree of analgesia that can be obtained by a drug can be limited, regardless of the dose. These drugs also reduce fevers and help with the muscle aches commonly associated with viral diseases, such as colds and influenza.

Typically, these agents act at the site of injury, leading to their classification as peripherally acting drugs as opposed to centrally acting drugs, such as the opiates, which work within the brain and spinal cord. These nonsteroidal anti-inflammatory drugs

TABLE 1
Commonly Used Analgesics

	Average Oral Dose (in milligrams)	*Frequency (in hours)*	*Comment*
Nonnarcotics			
Aspirin	650	4–6	No prescription needed
Acetaminophen (Tylenol)	650	4–6	No prescription needed
Ibuprofen (Motrin)	200–400	4–6	No prescription needed
Fenoprofen (Nalfon)	200–400	4–6	Nonsteroidal anti-inflammatory
Diflunisal (Dolobid)	500–1000	8–12	Nonsteroidal anti-inflammatory
Naproxen (Naprosyn)	250–500	8–12	Nonsteroidal anti-inflammatory
Piroxicam (Feldene)	20–40	8–12	Nonsteroidal anti-inflammatory
Narcotics (opioids)			
Codeine	32–65	4–6	Often used in combination with aspirin or acetominophen (Tylenol#3)
Oxycodone	5–10	3–5	Often used in combination with aspirin (Percodan) or acetaminophen (Percocet)
Propoxyphene HCl	65–130	4–6	Used alone (Darvon) or in combination with aspirin and caffeine (Darvon Compound)
Propoxyphene napsylate	65–130	4–6	Used alone (Darvon-N) or with acetaminophen (Darvocet)
Morphine	30–60	4–6	Also available in slow-release formulations
Meperidine (Demerol)	50–100	3–5	Not very effective orally
Pentazocine (Talwin)	50–100	4–6	Partial agonist with tendency to produce unpleasant, subjective feelings; can precipitate withdrawal in dependent people
Hydromorphone (Dilaudid)	4–8	4–6	Very potent analgesic
Methadone (Dolophine)	5–20	8	Very effective analgesic; also used in maintenance programs
Levorphanol (Levo-Dromoran)	2–4	4–6	Potent analgesic used for cancer pain
Oxymorphone (Numorphan)	1–2	4–6	Only available by injection
Nalbuphine (Nubain)	10	4–6	Only available by injection
Butorphanol (Stadol)	2	4–6	Available by injection or by nasal inhalation; partial agonist; avoid using in dependent patients

(NSAIDs) and aspirin work directly on the mechanisms of inflammation, which explains their effectiveness against arthritis. Ibuprofen became the first nonsteroidal drug approved for sale without a prescription, based on its long use and excellent safety record. Over the years, a number of additional drugs have been developed, many with analgesic potencies approaching those of morphine (Table 1). All of these require prescriptions and carry risks greater than the drugs available over the counter. Side effects include a tendency to irritate the stomach and to interfere with the actions of platelets, a blood cell important in clotting; therefore, aspirin and the nonsteroidal anti-inflamma-

tory drugs should be avoided in patients with ulcer disease, since the drugs can cause bleeding. Acetaminophen does not irritate the stomach and does not interfere with platelets—however, it has its own potential problems. Although it is one of the safest drugs available when used as directed, overdoses with acetaminophen can be very dangerous. Overdoses are associated with major damage to the liver, which can be life-threatening. Care must be taken to use only the recommended doses of acetaminophen.

As an alternative to NSAIDs, a new class of drugs, COX-2 (cyclooxygenase-2) inhibitors, are being used to treat and manage arthritis pain and inflammation. Three COX-2 inhibitors have been developed: celecoxib (brand name Celebrex), rofecoxib (brand name Vioxx), and meloxicam (brand name Mobic). Meloxicam is the most recent of the three, having been approved by the Food and Drug Administration (FDA) in April 2000, while Celebrex, the first to be marketed, became the fastest-selling drug in history.

COX-2 inhibitors, when compared to NSAIDs, are better for the intestinal and stomach linings (Kubetin, 2000). On the other hand, like NSAIDs, they have been found to cause renal (kidney) side-effects, such as reductions in filter rates (McCarthy, 2000).

Opioids work within the brain and spinal cord to relieve the second pain—the hurt—described above. In this regard, they are amazing, since they take away pain without interfering with other sensations, unlike local anesthetics. It is this ability to selectively act on the hurt that makes them so valuable. A number of opioids are used for moderate pain (see Table 1). Of these, CODEINE is the most widely used, both alone and in combination with the nonopioids described above, followed by OXYCODONE. Both are usually used in combination with either aspirin or acetaminophen. The peripheral and central analgesics's work complement

TABLE 2
Comparison of Analgesics

	Action	*Potential Side Effects*	*Special Comments*
Nonnarcotic			
Aspirin	Relieves pain, reduces fever and inflammation, inhibits blood clotting	Gastrointestinal irritation, allergic reactions; reduced blood clotting may harm mother or fetus if taken during pregnancy; has been linked to Reye's syndrome in children	Especially effective for pain from inoperable cancer and dental surgery; inhibiting effect on bloodclotting can reduce incidence of heart disease
Acetaminophen	Relieves pain, reduces fever	Liver and kidney damage	Effective for mild-to-moderate pain but not for inflammation
Ibuprofen	Relieves pain, reduces inflammation and fever	Gastrointestinal irritation, allergic reactions	Especially effective for menstrual cramps as well as pain and inflammation in muscles and joints
Narcotic			
Codeine	Reduces pain, suppresses cough	Drowsiness, nausea, moderate physical and psychological dependence, constipation, respiratory depression	Effective for mild-to-moderate pain; especially effective against cough
Meperidine	Reduces pain and anxiety	Drowsiness, respiratory depression, high physical and psychological dependence	Effective for moderate-to-severe pain
Methadone	Reduces pain, alleviates heroin withdrawal symptoms	Drowsiness, respiratory depression, high physical and psychological dependence	Effective for severe pain

SOURCE: U.S. Department of Health and Human Services, Washington, D.C.

each other. If they work well together, they also bring with them the side effects of all the ingredients. Thus, both codeine and oxycodone produce constipation and sedation, along with occasional nausea, while the aspirin or acetaminophen have the problems noted above. Propoxyphene is another opioid used for mild to moderate pain. Like the others, it is most often used in combination with aspirin or acetaminophen. Standard doses are not much more effective than aspirin or acetaminophen alone, but at sufficiently high doses propoxyphene is an effective painkiller.

Pentazocine is a relatively unusual analgesic. It is an opioid indicated for moderate pain, but unlike morphine and codeine, which act primarily through mu receptors, pentazocine works in part through kappa receptors. Caution must be used when taking this agent along with other opioids, since it is a mixed AGONIST/ANTAGONIST and can precipitate WITHDRAWAL symptoms in dependent people. Many opioid addicts report an "allergy" to pentazocine when being treated by physicians, to avoid the possibility of withdrawal.

For more severe pain, a number of highly potent opioids are available (see Table 1). They include MORPHINE, hydromorphone, levorphanol, MEPERIDINE, and METHADONE. All are available orally. Morphine is now available in special slow-release formulations, which permit dosing as infrequently as every twelve hours. This is much more convenient for patients, particularly at night, when they no longer have to awaken to take their medicines. Special care must be taken when using these long-acting analgesics. Slow-release morphine, like methadone, may take days to reach stable levels in the blood. Thus, it can be difficult to adjust dosages without "overshooting"—which, if severe, can lead to OVERDOSES that may be life-threatening.

In hospitals, many patients receive opiates by injection or intravenously. Doses need to be adjusted to compensate for differing distributions and metabolism, but these changes are relatively straightforward for physicians working in the area of pain. Special devices are also available that permit patients to dose themselves, as needed, within specified guidelines. This approach is termed *patient controlled analgesia* (PCA). Even more sophisticated routes of administration are available. Some medications can be injected deep in the back, adjacent to the spinal canal (epidurally) where they can act primarily on the spinal cord. Localizing the medication to the spinal cord can minimize the side effects produced in the brain, such as nausea and respiratory depression.

The chronic use of opioids leads to a lessening of potency, which is termed *tolerance*. To overcome this, it may be necessary to increase the dose to maintain a constant effect. Furthermore, all patients taking sufficient quantities of drug for an extended time will become physically dependent—that is, they will experience some withdrawal if the drug is stopped. Very few patients taking opioids for medicinal purposes will ever become addicted, as the term is now used by psychiatrists. This distinction between the standard physiological responses of TOLERANCE/DEPENDENCE and ADDICTION is important, because fear of addiction should not interfere with the appropriate medical therapy of pain.

(SEE ALSO: *Abuse Liability of Drugs; Addiction: Concepts and Definitions; Controlled Substances Act; Opioids and Opioid Control; Pain: Behavioral Methods for Measuring Analgesic Effects of Drugs for; Tolerance and Physical Dependence*)

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REVISED BY REBECCA MARLOW-FERGUSON

PAPAVER SOMNIFERUM Poppy plants, of the genus *Papaver*, are long-stalked flowers of varying colors encompassing approximately 140 species. Of the many types of poppy plants, *Papaver somniferum* is known as the OPIUM poppy. It has white or blue-purple flowers and is widely cultivated in Asia, India, and Turkey, which supply much of the world's opium. Cultivation requires a tropical or subtropical climate without excessive rainfall. In the Northern Hemisphere, the plant flowers in late spring, after which the petals fall in a short time. This is followed by the rapid growth of the capsules (the plant's ovaries) for about two weeks. Incisions are carefully made in the capsule to obtain the milky juice, which is then dried as a gum that yields opium. The yield of opium can vary widely, but is typically about five pounds (2.25 kilograms) per acre.

The opium serves as a source of MORPHINE, CODEINE, and thebaine and is widely used in the production of important painkillers (ANALGESICS).

Typically, morphine comprises 10 percent of opium and most of the morphine used in medicine is obtained by purifying opium.

Illicit uses of opium are also widespread. In many parts of the world, opium is still smoked or eaten. Morphine extracted from opium may in turn be converted to HEROIN in clandestine laboratories. Heroin is the major opioid used illicitly in the United States. To prevent the collection and sale of opium for illicit conversion to heroin, new ways of processing the poppy plant have been developed. The most widely used consists of mowing the poppy fields before the pods are ripe enough to yield opium. The mowed stems, immature pods, and plant matter, referred to collectively as poppy "straw," are then shipped in bulk to large processing centers where the active ALKALOIDS are extracted under careful supervision.

Other species of *Papaver* also contain alkaloids that can be converted into potent opioids. For example, *Papaver bractiatum* contains high concentrations of thebaine, which can be used to produce compounds several hundred times more potent than morphine.

(SEE ALSO: *Asia, Drug Use in; Crop-Control Policies; Golden Triangle as Drug Source; International Drug Supply Systems; Pain, Drugs Used for; Opioids and Opioid Control*)

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GAVRIL W. PASTERNAK

PARAPHERNALIA LAWS AGAINST Drug paraphernalia are articles that facilitate or enable the use of illicit drugs, such as hypodermic syringes for HEROIN or pipes for smoking MARIJUANA. Laws prohibiting the possession and use of paraphernalia have been adopted in every state of the United States despite significant constitutional objections to them.

The first drug-paraphernalia laws, prohibitions against possessing opium pipes, were enacted by western states in the late nineteenth century as part of broad statutory efforts to suppress opium smoking by CHINESE immigrants. During the first third of the twentieth century, some states, in conjunction with a legislative attempt to criminalize the nonmedical use of OPIATES and COCAINE, also prohibited the possession of hypodermic syringes without a medical prescription. By 1972, when the NATIONAL COMMISSION ON MARIJUANA AND DRUG ABUSE conducted a survey of state drug laws, about twenty states had adopted some type of drug-paraphernalia prohibition.

Commercialization of drug paraphernalia, especially through so-called head shops, in the early 1970s triggered a new generation of paraphernalia prohibitions, many of which criminalized the sale as well as possession of these articles. Such laws attempted to enforce comprehensive bans on drug-related devices or articles intended for use with illicit drugs.

The drug-paraphernalia industry responded to the enactment of these laws by challenging their constitutionality on vagueness and overbreadth

Since the late nineteenth century, the federal and state governments have enacted laws to regulate the possession and sale of drug paraphernalia, like the crack pipes pictured here. (© Bettmann/CORBIS)

grounds. In most cases, courts struck down the laws as unconstitutionally vague: first, because they applied to objects that had lawful as well as unlawful uses, these laws failed to provide fair notice of prohibited conduct; and second, the lack of explicit standards left police with discretion to enforce these laws in an arbitrary and discriminatory manner.

In 1979, the U.S. Drug Enforcement Administration (DEA) stepped into the fray. In an attempt to assist states and localities in drafting laws that might withstand constitutional scrutiny and at the same time effectively combat the drug-paraphernalia trade, the DEA drafted a Model Drug Paraphernalia Act (MDPA). Unlike prior state laws, the MDPA explicitly requires prosecutors to prove that the defendant knew the alleged paraphernalia would be used with illegal drugs. The addition of the so-called intent requirement was designed to alleviate the fair-warning concern associated with the earlier generation of statutes. In addition, the MDPA attempts to provide a more specific definition of drug paraphernalia by listing objects included within the category and by providing factors that judges should consider in determining whether an object falls within the definition. Finally, the act prohibits placement of an advertisement when one knows, or "reasonably should know," that it is intended to promote the sale of objects "designed or intended for use as drug paraphernalia."

A majority of states have adopted the MDPA or an equivalent statute, but its constitutionality has yet to be ruled on by the U.S. Supreme Court. In 1982, however, the Court upheld a local ordinance that required businesses to obtain a license in order to sell articles designed to be used with illegal drugs. Although this law did not involve a criminal statute prohibiting sale or possession of paraphernalia, most lower courts have subsequently upheld criminal laws modeled after the MDPA against vagueness and overbreadth challenges.

In the wake of the HIV/AIDS epidemic, another feature of traditional drug-paraphernalia laws has become controversial. In an effort to reduce the risk of transmission of the HUMAN IMMUNODEFICIENCY VIRUS (HIV) and other blood-borne diseases among needle-sharing illicit drug users, state and local public-health authorities have sought to establish clean-needle exchange programs, usually through hospitals and clinics. To implement these programs, lawmakers have had to repeal the paraphernalia laws or prosecutors have agreed not to enforce them in this context. Many states and local governments have refused to support needle-exchange programs, and the federal government has not funded them due to concerns that dispensing needles will encourage illicit drug use. However, the National Academy of Sciences has concluded that these programs reduce the risk of HIV transmission and has found no evidence that they encourage drug use.

In general, drug-paraphernalia laws represent a type of drug legislation aimed mainly at declaring and symbolizing society's intolerance of illicit drug use. Like other symbolic uses of criminal law, however, these laws are subject to highly discretionary enforcement and can have unintended costs or ramifications.

(SEE ALSO: *Legal Regulation of Drugs and Alcohol; Needle and Syringe Exchanges and HIV/AIDS; Parents Movement; Prevention Movement; Substance Abuse and AIDS*)

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RICHARD J. BONNIE

PARASITIC DISEASES AND DRUGS OF ABUSE A long historical awareness exists regarding the association between heavy ALCOHOL use and an increased risk or severity of symptoms caused by infectious diseases. In the United States, this awareness can be traced back to the medical literature of the late 1700s. It continues to evolve in ongoing research. Historically, most infectious diseases were viral and bacterial and caused death, such as tuberculosis. Some intestinal diseases have been also noted, especially cholera. This is an acute infectious disease of epedemic proportion caused by *Vibrio cholerae* (a gram-negative bacillus) that produces a soluble toxin in the intestinal tract, with profuse watery diarrhea, extreme loss of fluid and electrolytes, and a state of dehydration and collapse with death often following.

Modern research in immunosuppressed humans and animals has isolated a protozoan parasite, *Cryptosporidium parvum*, that affects the gastrointestinal tract. In immunocompetent hosts the disease is self-limiting and recovery is accompanied by resistance to reinfection. *Cryptosporidium* is, however, common in patients with acquired immunodeficiency syndrome (AIDS). It has been noted in 16 to 50 percent of cases, but is rarely manifested in HIV-positive people before loss of CD4 cells. Research with alcohol and COCAINE in AIDS-compromised animals has indicated lessened resistance to *Cryptosporidium*. This is true as well with similar AIDS-compromised animals having colonies of trophozoites (a vegetative protozoan) of Giardia muris infecting the small intestine. The reason parasite infections in addition to some cancers and certain other diseases are more common in heavy or chronic alcohol users relates significantly to suppression of host defenses. Alcohol use lowers production of antibodies. Cocaine suppresses the functioning of T-lymphocytes, critical to the activation of immune defenses. Some infections, particularly parasitic ones, require a substantial lowering of natural immunity and resistance to be able to grow for more than a few days. Alcohol and drugs of abuse are strong suppressors of resistance mechanisms. Thus, their adverse effects are even more pronounced in the elderly, AIDS patients, transplant recipients, and others with damage to their immune system. In addition, alcohol and drugs of abuse lower intakes and tissue levels of antioxidant vitamins and nutrients, important for optimum functioning of host defense systems. Supplementation with antioxidant vitamins helps overcome some of the damage due to AIDS, age, and drug abuse.

(SEE ALSO: *Complications; Alcohol; Immunology; Substance Abuse and AIDS*)

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RONALD R. WATSON

PAREGORIC A camphorated OPIUM tincture; tinctures of opium are alcoholic extracts of opium, widely used in the treatment of diarrhea. Paregoric contains powdered opium, anise oil, benzoic acid, camphor, glycerin, and diluted alcohol. With only 0.4 milligrams per milliliter of MORPHINE in 45 percent alcohol, it is more dilute than opium tincture—and the taste of the camphorated formula is generally disliked, helping to minimize excessive use or abuse.

Although paregoric is not indicated for bacterial or parasitic causes of diarrhea, it can be very helpful for other causes. Taken orally, it effectively slows down the gastrointestinal transit of wastes and enhances resorption of fluid from the intestine. Doses that effectively treat diarrhea typically do not cause euphoria or analgesia; however, excessive doses can be abused and can lead to DEPENDENCE.

(SEE ALSO: *Dover's Powder: Laudanum; Opiates/Opioids*)

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GAVRIL W. PASTERNAK

THE PARENT MOVEMENT The parent drug-prevention movement emerged in the latter half of the 1970s in response to the greatest escalation in drug use by children and adolescents in the history of the world. It originated with a number of people, who founded several different national organizations to lead the parent movement.

In August 1976, an Atlanta mother, Marsha Keith Mannat Schuchard, Ph.D., and her husband, Emory University professor Ronald Schuchard, Ph.D., discovered at their eldest daughter's thirteenth birthday party that she and most of her friends were using drugs that evening. In response, the family organized the nation's first parent-peer group. Such groups consist of parents whose children are each others friends. The parents come together to establish age-appropriate social and behavioral guidelines they agree to adhere to in order to protect their children and help them avoid unhealthy and destructive behaviors during adolescence. In a very short time, the young people whose parents formed this first parent-peer group stopped using drugs and returned to the productive behaviors in which they'd been engaged before they entered the drug culture. Dr. Schuchard later wrote about this experience in *Parents, Peers and Pot*, a book the National Institute on Drug Abuse published and distributed free to the more than one million people who requested it during the 1980s.

In the fall of 1977 a group of concerned Atlanta citizens formed National Families in Action. Founders included Keith Schuchard and Sue Rusche. Mrs. Rusche later became the organization's executive director. This organization called attention to the social and environmental factors that seemed to promote the use of illicit drugs. Its purpose is twofold: 1) to replace commercial and societal messages that glamorize drug use with accurate information based on scientific research about drug effects, and 2) to help people put this information to use by organizing community-based parent drug-prevention groups. At the time of its founding, National Families in Action responded to the explosion in all communities of head shops, which appeared to target children and teenagers as potential customers. Drug users called themselves "heads"—"acid heads," "pot heads," "coke heads," etc. Head shops were places that sold books and magazines that taught people how to use drugs, and toys and gadgets to assist and enhance drug taking. The materials head shops sold were called drug paraphernalia. In January 1978, National Families in Action succeeded in getting the Georgia Legislature to pass the nation's first laws banning the sale of drug paraphernalia.

At about the same time, Otto and Connie Moulton, of Danvers, Massachusetts, founded Committees of Correspondence. Their goal was to alert citizens about the activities of drug-culture and drug-policy organizations that advocate for the decriminalization and legalization of marijuana, cocaine, heroin, PCP, and other illicit drugs. They began sending out packets they called "Otto Bombs," detailing information about the local, state, and federal lobbying activities of drug-legalization organizations such as the National Organization for the Reform of Marijuana Laws (NORML), whose board and advisory board at the time consisted of many drug-paraphernalia manufacturers and publishers. Patterned after the original Committees of Correspondence, founded by our forefathers to uphold the rights of colonists before and during the Revolutionary War, the modern-day version seeks to uphold the rights of citizens to be drug-free. A periodic newsletter presents information from researchers and doctors that refutes medical and scientific claims made by legalization proponents. Committees of Correspondence also

tracks the lobbying efforts of other organizations that advocate legalizing drugs, including the Drug Abuse Council in the 1970s and the Drug Policy Foundation in the 1990s.

In April 1978, Thomas "Buddy" Gleaton, Ed.D., invited Keith Schuchard and Sue Rusche to address the Fourth Annual Southeast Regional Drug Conference. Gleaton held the conference for drug-education professionals at Georgia State University, where he taught. He also invited officials from various federal agencies. Many accepted, particularly from the National Institute on Drug Abuse. The Parents' Resource Institute for Drug Education (PRIDE) was founded in the summer of 1978, following this conference.

Publicity generated by the passage of Georgia's drug paraphernalia laws, by the Fourth Southeast Drug Conference and, later, by the publication of *Parents, Peers and Pot*, brought requests for help from parents throughout the United States. These parents wanted to form parent groups to ban drug paraphernalia sales in their cities, towns, and states, and to prevent substance abuse among their children in their families and in their communities. For the next several years, leaders from National Families in Action, PRIDE, Committees of Correspondence, and other national organizations, along with leaders of emerging groups from various states, traveled across the nation helping parents form prevention groups. A contract the National Institute on Drug Abuse awarded to Pyramid made much of this work possible. Pyramid hired parent-group leaders as consultants and paid their expenses to travel to communities that sought their help in organizing groups.

One of the first groups to form outside Georgia was Naples (Florida) Informed Parents, led by Pat and Bill Barton. The Florida leaders joined those from Georgia and Massachusetts to help parents in other states form similar groups. By 1979, hundreds and perhaps thousands of parent groups had organized across the nation. In January 1979, Senator Charles Mathias (D-MD) held Congressional hearings on the harmful effects of marijuana, and invited many parent-group leaders, along with scientists, to Washington to testify. The parent leaders took advantage of this opportunity to be together for the first time and discussed the need to form a Washington-based organization that could represent their interests with both Congress and the federal agencies that were making and implementing national drug policy. They agreed to meet at the Fifth Annual Southeast Regional Drug Conference, now known as the PRIDE conference, in Atlanta in the spring of 1979. There, they founded the National Federation of Parents for Drug-Free Youth. Pat and Bill Barton were elected as the group's co-presidents and a Maryland parent group leader, Joyce Nalepka, later became the Federation's executive director.

The summer of 1979 was an election year, and parent groups worked hard to get drug-abuse-prevention policy on the agendas of Presidential candidates. After the election, the National Federation of Parents for Drug-Free Youth led a massive letter-writing campaign to President-elect Ronald Reagan, asking him to bring Carlton Turner, Ph.D., to the White House as his drug-policy advisor. Dr. Turner, of the University of Mississippi, was responsible for growing all marijuana used in scientific research throughout the world. He had devoted much time to educating parents at various conferences about the pharmacological effects of marijuana on the brain and body, and had earned their trust. President Reagan acted on the parent federation's appeals and selected Dr. Turner as his drug advisor.

Shortly after the inauguration, Dr. Turner helped the federation arrange for parent-group leaders to brief Mrs. Reagan on the prevention movement and enlist her support for their cause. She not only responded positively, but served informally as the national spokesperson for the parent drug-prevention movement. A few years later, President Reagan appointed parent-group leader Ian Macdonald, M.D., a pediatrician from Florida, to serve as Administrator of the Alcohol, Drug Abuse and Mental Health Administration (ADAMHA), the federal agency in the Department of Health and Human Services that was responsible for substance abuse and mental health research and services. One of Dr. Macdonald's legacies is the Center for Substance Abuse Prevention (then called the Office for Substance Abuse Prevention, or OSAP), which he created as an office during his tenure at ADAMHA. Congress formally authorized OSAP as a center, changed its name to CSAP, and funded it in the Anti-Drug Abuse Act of 1986.

Through this kind of concerted effort, parents were able to place key policy-makers in the federal government to emphasize and implement their goals: To prevent the use of illegal drugs (and alco-

hol and tobacco among those underage) before it starts, to help drug users quit, and to find treatment for those who are addicted and cannot quit by themselves. The parent movement was the first leg of the national drug-prevention effort that was active in the year 2000. It is generally credited with developing and carrying out strategies that reversed the drug policies of the 1970s, which seemed to increase drug use throughout that decade.

These strategies included outlawing head shops and the sale of drug paraphernalia, stopping the decriminalization/legalization of marijuana (and other drugs), and insisting that drug-education materials contain "no-use" messages, based on accurate scientific information about the effects of drugs on health and on local, state, and federal laws and international treaties. As a result of effectively implementing such strategies, both Robert DuPont, M.D., and William Pollin, M.D., the first two directors of the National Institute on Drug Abuse, credit the parent movement with being responsible for reversing the 1970s escalation in drug use by children, adolescents, and young adults, and for initiating the reduction in regular drug use that took place among all ages between 1979 and 1992.

Beginning in the late 1980s, the Center for Substance Abuse Prevention made demonstration grants available to support local, grass-roots, drug-prevention efforts targeting high-risk youth, primarily in African-American, Hispanic, Asian-American, and Native American Indian communities. Many new parent and family-based groups emerged to join the parent drug-prevention effort as a result. So did national groups representing each of these populations, including African-American Parents for Drug Prevention, based in Cincinnati, Ohio; the National Hispano/Latino Community Prevention Network, based near Albuquerque, New Mexico; National Asian Pacific American Families Against Substance Abuse, in Los Angeles, California; and the National Association for Native American Children of Alcoholics in Seattle, Washington. These groups joined with National Families in Action, PRIDE, and the National Federation of Parents for Drug-Free Youth to form The Parent Collaboration to inspire today's parents to form volunteer parent groups to prevent drug use among their children. An additional group, the Drug Free America Foundation based in St. Petersburg, Florida, works with the collaboration. Unfortunately, economic pressures that drive contemporary parents to work and to devote an average of 50 to 60 hours to their workweeks, mean there is simply no time for parents to volunteer a sustained drug-prevention effort, as the previous generation of parents were able to do. Furthermore, Congress eliminated high-risk youth grants from the Center for Substance Abuse Prevention, and funds were simply not available to enable parents to work full-time, or even part-time, at preventing drug abuse in their families and communities.

As funding to support minority parent- and family-based drug-prevention groups disappeared, a well-funded effort to legalize drugs re-emerged in the 1990s. This effort has contributed to re-establishing conditions that are similar to those that appeared to drive drug use up among young people in the 1970s. Legalization proponents reject abstinence-based drug-education as "unrealistic," and advocate instead for educational materials that teach children how to use drugs "safely." Proponents also are leading efforts to sponsor state ballot initiatives that attack or weaken laws forbidding drug use, drug dealing, and drug trafficking and, at the same time, are launching lobbying efforts to legalize drugs. With the growing popularity of the Internet, the sale of drug paraphernalia, and even of illicit drugs, along with an amazing array of misinformation about drug effects, dominates online drug sites. As these conditions intensify, so does drug use and drug abuse. In the state that has passed the most measures to soften or eliminate its drug laws, Oregon, more citizens now abuse illicit drugs than alcohol, according to a survey commissioned by the Health Division of the Oregon Department of Human Services. Of even more concern nationwide, the 13-year-long, two-thirds decline in regular drug use among adolescents (and the 500 percent drop in daily marijuana use—from 11 to 2 percent among high school seniors) ended in 1992, and drug use doubled among teens throughout the decade. While some government surveys show adolescent drug use is now leveling off, others show drug use continues to rise among teens and young adults.

As drug use rises once again among America's children, America's parents are unavailable to work for drug prevention. Most must work to earn money to provide for their families. They cannot afford to do the long sustained work of drug prevention without pay. No funding mechanisms exist to give parents the opportunity to "switch jobs"

and work full time, or even part time, to prevent children from entering a culture whose lure intensifies each year. Until this changes, the outlook for reducing drug use among the nation's children, adolescents, and young adults remains bleak.

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SUE RUSCHE

PARTNERSHIP FOR A DRUG-FREE AMERICA The Partnership for a Drug-Free America is a nonprofit coalition of the United States communications industries; its mission is to help reduce demand for illegal drugs by using the media to change the attitudes that affect drug trial and experimental (nonaddicted) use. The key officers of the organization are James E. Burke, chairman; Thomas A. Hedrick, Jr., president; Richard D. Bonnette, executive director; and Robert L. Caruso, chief financial officer.

The partnership was founded by Richard T. O'Reilly in early 1986 as a project of the American Association of Advertising Agencies. It was based on the idea by Philip Joanou, chairman of Dailey & Associates in Los Angeles, that the disciplines of marketing could be used effectively and efficiently over time to help "unsell" illegal drugs. The hypothesis was that prevention could be viewed as trying to affect individual decisions to buy or use illegal drugs in the same way that individual decisions to buy or use legal products and services are affected—except in reverse. Rather than using media messages on the *benefits* of a product, the partnership set out to reduce drug trial by building awareness of the *risks* and danger of using illegal drugs.

The Partnership's early strategy was based on a concept developed by Dr. Mitchell S. Rosenthal, president of the PHOENIX HOUSE treatment programs in New York. He theorized that the epidemic levels of drug use and addiction in the early 1980s was caused by a process of "normalization"—to both the use and users of illegal drugs—since the mid-1960s. According to Dr. Rosenthal, we could not achieve significant progress in "the war on drugs" until we reversed that process and "denormalized" individual and subcultural attitudes toward illegal drugs.

The three primary functions of the partnership are (1) to understand consumer attitudes that affect the trial and use of illegal drugs; (2) to develop messages targeted to specific demographic groups; and (3) to deliver those messages to the public through all forms of the media, but primarily public-service announcements. These functions, managed by a small full-time staff, have been accomplished through the volunteer efforts of research firms, advertising agencies, production groups, and the media. As of the end of 1992, more than 300 antidrug print and broadcast messages had been delivered, at no cost to the partnership and valued at more than 50 million dollars. Since the program's launch in March 1987, the media have donated more than 1.5 billion dollars in advertising time and space.

The partnership's prevention messages are targeted primarily to preteens and young teens, inner-city youth, and also parents, peers, and siblings, who are viewed as the key influencer groups. The focus of the messages is on building perceptions of risk and social disapproval, promoting resistance skills, and reinforcing a consistent tone of social denormalization in regard to illegal drugs. Overall media efforts are directed at achieving the goal of 1 million dollars a day in donated time and space. This results in the delivery of approximately one antidrug message per household per day. All major national media are visited personally by partnership staff to monitor the program. State and local media programs are also developed and supported through staff and volunteer efforts.

The organization's tracking research is funded by the NATIONAL INSTITUTE ON DRUG ABUSE

(NIDA) and directed by the Gordon S. Black Corporation. The annual Partnership Attitude Tracking Survey (PATS) uses a centrally located sampling to evaluate attitudes toward illegal drugs among more than 8,500 preteens, teens, and adults. This research, along with other major NIDA studies and especially the HIGH SCHOOL SENIOR SURVEY done by the Institute for Social Research, suggests that since 1986 attitudes to illegal drugs have been changing. Furthermore, the surveys indicate that the partnership's messages have been a major source of information (among others) that helped effect these changing attitudes.

It is difficult to establish a scientifically conclusive cause-and-effect relationship between the partnership's efforts and U.S. trends in drug-use behavior. Many components are necessary—particularly community efforts—to reduce demand for illegal drugs, and it is unlikely that any one component is sufficient to the task. It is also imperative to note the importance of timing in this media effort, since the media are most effective in accelerating trends that are already in place. The media play a large role in American society and therefore in the lives of the children growing up in that society. The Partnership is mounting a very significant communications effort to influence the way Americans think about illegal drugs.

(SEE ALSO: *Advertising; Prevention: Shaping Mass Media Messages to Vulnerable Groups; Prevention Movement; Prevention Programs*)

THOMAS A. HEDRICK, JR.

PASSIVE SMOKING *See* Nicotine; Tobacco: Medical Complications

PATENT MEDICINES *See* Over-the-Counter Medication

PCP *See* Phencyclidine (PCP)

PEER PRESSURE *See* Adolescents and Drug Use; Causes of Substance Abuse

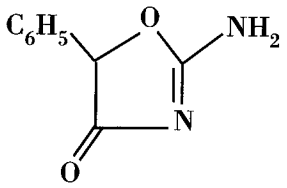

Figure 1
Pemoline

PEMOLINE Although not structurally similar to the AMPHETAMINES, pemoline has similar PSYCHOMOTOR STIMULANT effects but only minimal effects on the cardiovascular system. Pemoline is often used therapeutically (despite being less effective than amphetamine or METHYLPHENIDATE) in the treatment of ATTENTION DEFICIT/hyperactivity disorder (ADHD)—a syndrome that first becomes evident during childhood and is characterized by excessive activity and difficulty in maintaining attention. Pemoline has the advantage of a long half-life, which means that dosing can be once daily, but clinical improvement can be delayed by three to four weeks after initiation of pemoline therapy. In addiction, the likelihood for abuse of pemoline appears substantially less than that of the amphetamines.

MARIAN W. FISCHMAN

PERCODAN *See* Oxycodone

PERSONALITY AS A RISK FACTOR FOR DRUG ABUSE The term *personality* refers to those relatively enduring aspects of attitudes, feelings, responses, and behaviors that permit us to recognize a particular person whom we have known over time. It is, in a way, a fingerprint of an individual's psychological makeup—the framework of how the individual thinks and acts. Psychiatrists believe that this framework arises out of childhood, powerfully shaped by the actions of parenting and the other social and environmental factors on a complex set of genetic and other biological givens. It is then further molded throughout one's development to achieve more or less lasting form in adolescence and early adulthood.

In the nineteenth century, we said that some people had willpower or a strong character; now we might refer to their good coping skills or to their ego

strength—different ways of describing global measures of effective functioning. Current terms for more specific descriptors of personality might include the poles of introversion—extroversion, or approach—avoidance, as well as others.

There is a long tradition linking personality, or character, to alcohol and other substance use and abuse. In the popular imagination, the old usage of "alcoholic" or "drug fiend" conveyed images of weakness, untrustworthiness, and/or viciousness; more sophisticated imagery, "oral character," conveyed ideas of dependency and neediness—analogies to the greedy infant at the breast. Unfortunately, such simple postulates break down in the presence of the complexities of the real world: Not all substance abusers are frightening "drug fiends"; neither are they necessarily dependent, needy, demanding "oral characters."

The explanation for substance abuse is not found purely in the drug. Most adults are able to drink socially without becoming alcoholics; some of us are repeatedly exposed to opiates (e.g., after surgery) without becoming addicts. Clearly, the impact of personality on alcohol and other drug use depends on a variety of factors—the social context, the specific drug, and the stage of involvement with the drug. Is the individual brought up as a rich kid in the suburbs or poor in an inner-city ghetto? Is the person black or white? Do drugs and drug users surround the individual, and are they seen as normative, or are they considered dangerous, rare, and deviant? Is the drug a relatively weak reinforcer such as marijuana, or is it a powerful stimulant such as cocaine? Is the individual experimenting in the early stages of use, struggling with long-term dependency, or dominated by the pangs of withdrawal and craving? Although a number of predictor factors for substance abuse are known, such as age, sex, religiosity, and parental drug use, we do not know why only *some* of those at risk become drug dependent. Personality is another likely predictor of who will try a particular drug, who will continue to use it or abuse it, the success of the struggle with abstaining, and so forth.

As the preceding indicated, early thinking was that excessive drinking (alcohol) and smoking (tobacco) were linked to early childhood experiences of suckling and satiation, of hunger satisfied by taking something in through the mouth that resulted in blissful sleep. That this may, at least at times, be true was seen in one patient who had first been addicted to alcohol and then to a series of barbiturates and other sedatives; he said plaintively, "Doc, I could become addicted to orange juice if it gave me a dreamless sleep." Unfortunately, just as the thumb fails to provide milk, most drugs do not ultimately provide the desired end—the continuing sense of pleasure and/or relief. It was assumed that individuals who had had difficulty in the earliest stages of development might be particularly prone to some kinds of addiction—to depressive drugs, such as alcohol, sedatives, or opiates, which provide dreamy reverie states or sleep—and that difficulty in later stages of development might predispose to use of activating drugs, such as the stimulant amphetamines or cocaine.

Ongoing clinical experience and changing theories led investigators to focus additionally on aggression and on regulation of feelings. For example, many addicts appear to have difficulty distinguishing anxiety and anger, and they experience strong feelings as overwhelming, leading to loss of control. The drug may substitute, both pharmacologically and symbolically, for the parent—to "magically" help the individual maintain control. It has also been noted that many addicts appear not to have learned from their parents how to recognize, evaluate, and appropriately respond to danger. Many, or all, of these additional factors may operate at once: Individuals may be trying to satisfy primitive impulses and needs; there may be a defect in the recognition and control of feeling states; and they may be struggling to adapt to a stressful environment. A particular drug may, for a particular individual, transiently resolve these issues. Heroin may satiate, dampen, and control aggression—and provide relief from environmental pressures—for the moment. Amphetamines or cocaine may provide orgasmic pleasure, in the form of a "rush, as well as provide a sense of control and omnipotence. A patient who was dependent on amphetamines was panicked at the thought of dental anesthesia: "I can't stand the idea of not being in control, of being put to sleep. It's why I take the pills, to stay awake, to know what's happening."

Many individuals who misuse drugs will misuse many different kinds of drugs—the polydrug abusers. There are also people who, even after extensive experimentation with a variety of drugs, will choose to use and/or abuse a single drug or class of drugs—such as opiates, sedatives, or stimulants. It has been suggested that such individuals are driven

to seek a particular drug experience, since the various drugs indeed have differing physiological and psychological effects.

Some studies lend support to this notion of particular personality contributions to drug preference. For example, opiates tend to bolster withdrawal (from others) and repression (not acknowledging reality) by inducing a state of decreased motor activity, underresponsiveness to external situations, and reduction of perceptual intake. Such a state is conducive to reinforcing fantasies of omnipotence, magical wish-fulfillment, and self-sufficiency, but both sexual drive and aggression are diminished. In addition, there is evidence that opiate addicts are, in general, more severely impaired in terms of their ability to function in the ordinary world; they are less able to cope with the activities of daily living. In contrast, amphetamines elevate scores on autonomous functioning and sense of confidence; there is a feeling of heightened perceptual and motor abilities accompanied by a strengthened sense of potency and self-regard. These effects appear to serve the user's need to feel active and potent in the face of an environment perceived as hostile and threatening—and also to deny underlying fears of passivity.

It is important to remember that all of us have some quirks, that we do not always handle all kinds of stress equally well, that we all have some weaknesses in our personalities, some defects in our characters. These may predispose some of us to drug use and to particular drug choice. Others have significant defects in development, disordered adaptations to the real world in which we are expected to function; they may choose a particular drug or drugs to help them adapt to their difficulties—to make up, in a sense, for what is lacking within them. They are in effect choosing and self-administering their own medicine. This has been referred to as the *self-medication* theory of drug abuse. Certainly, drugs are capable of dramatically reversing painful emotional states; they can mute or free us from unmanageable feelings and provide some with the feeling that "It's the only time I've ever felt normal." Unfortunately, these effects are short-lived; side effects and the complications of physical dependance, tolerance, and withdrawal become prominent and even dominate the chronic user, who has become a substance abuser.

Be cautioned: These studies were done on people who had already been using illicit drugs for many years—who had been immersed in the "drug world" of copping (getting the drug), fearing detection and detention, and living with the altered state of consciousness induced by their drug of choice. These studies and others like them can tell us only of a correlation, not a causal relationship, where personality style or defect results in or leads to drug use/ abuse. There are, however, some longitudinal studies that have followed schoolchildren for enough years to have seen some of them enter the drug world. In general, they show remarkable agreement in the descriptions of those children who become seriously involved with drugs. They are the opposite of the stereotype of the Eagle Scout (who is "thrifty, loyal, brave, clean, and reverent"); instead, they may be characterized as impulsive, with difficulty tolerating feelings and delaying gratification, and as possessing an antisocial personality style given to breaking rules, oppositional behavior, risk taking, and sensation seeking. These personality characteristics are present before immersion in the drug culture and are altered as the individual moves from initial use to continuing use, to the transition from use to abuse, to cessation or control of abuse—and, all too often, to relapse.

Be further cautioned: These findings may have been true at the time of the studies but may prove to be specific to that moment of history and no longer true. Zinberg (1984) has pointed out that the setting in which one takes a drug, and therefore the meaning of the drug-induced experience, is continually changing:

> Chronic users [of marijuana], those that began using prior to 1965 were observed to be more anxious, more antisocial, and more likely to be dysfunctional than were the naive subjects who were just beginning to use marijuana in 1968. . . . By the late 1960s, drug use was being experienced as a more normative choice . . . in the early 1970s, controlled marijuana users could not possibly have been described as individuals driven to drug use by deep-seated, self-destructive, unconscious motives [p. 174].

An alternative view that has been suggested is that a series of otherwise accidental environmental reinforcers may so interact as to result in drug use in the absence, or the limited availability, of otherwise more necessary and pleasurable commodities. Experiments have shown just such development of "excessive behavior" in both animals and humans

during conditions of deprivation—of not enough water or food—but they have not yet demonstrated such a role in the induction to drug use.

Despite these cautions, it appears that PERSONALITY is a contributor that predisposes some to substance use and abuse. Different personalities are likely to make differing contributions to drug use, depending on the particular drug, the historical moment, the social surround, and the other determinants of use. Although it is still difficult to demonstrate more than generalities about the personality of addictive behaviors, the construct of addictive personality(ies) may be "theoretically necessary, logically defensible, and empirically supportable" (Sadava, 1978). Without such a construct—which includes the characteristic response patterns of the individual, the symbolic meaning of the experience to the individual (while recognizing that this may be retrospective rationalization), as well as the specifics of the particular drug's pharmacology—it will be difficult to explain the variation in drug use among individuals with apparently comparable life experiences.

(SEE ALSO: *Adjunctive Drug Taking; Causes of Substance Abuse; Conduct Disorder and Drug Use; Coping and Drug Use; Families and Drug Use; Vulnerability As Cause of Substance Abuse*)

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PERSONALITY DISORDER The concept of *personality* refers to the set of relatively stable and characteristic behaviors that individuals display in perceiving and responding to the environment, along with a particular way of thinking about themselves. These patterns of behavior and self-perception are called personality traits. They are manifested in a variety of social interactions in day-to-day living, and their diversity is extensive. When these traits become exaggerated, inflexible, and maladaptive, they begin to impair social functioning and can cause subjective distress. Different constellations of maladaptive traits are clinically diagnosed as personality disorders. Frequently, individuals identified as having a personality disorder do not see themselves as others see them, do not recognize the annoyance their behavior engenders in those around them, and hence do not seek to change their behaviors unless there are significant social repercussions. The characteristic traits of a personality disorder typify the individual's long-term functioning and are generally recognizable by adolescence.

In psychiatry, clusters of certain personality traits are recognized in the DIAGNOSTIC AND STATISTICAL MANUAL *of Mental Disorders- 3rd ed.-revised* as constituting particular personality disorders. There is some overlap in the trails of some of the following identifiable personality disorders.

Paranoid
: suspicious, mistrustful, hypervigilant, easily offended, unfeeling toward others

Schizotypal
: odd and eccentric behavior, speech, and manner of thinking; withdrawn and isolated

Narcissistic
: exaggerated sense of self-importance, feelings of entitlement to special favors, exploitation of others, lack of empathy, response of rage to criticism, disregard for social conventions

Histrionic
: dramatic, emotional, erratic, with displays of seductive behavior; attention-seeking

Antisocial
: antisocial behavior in many areas of life: lying, theft, violence, substance abuse, sexual promiscuity, spouse and child abuse, inconsistent work, legal conflicts; impulsivity and lack of remorse for antisocial acts

Borderline
: unstable mood, behavior, relationships, and sell-image; impulsive, self-destructive acts (e.g., suicide attempts, substance abuse); chronic feelings of emptiness, intolerance for being alone

Avoidant
: timid, extreme sensitivity to real or imagined rejection, socially withdrawn, poor self-esteem

Dependent
: avoidance of taking responsibility for their lives and a striving to get others to look after them; passive, submissive, with low self-esteem, and discomfort when alone

Obsessive-compulsive
: perfectionist, orderly, inflexible, indecisive, constricted emotions, obstinate, overly conscientious

Passive-aggressive
: resistance to demands for adequate social and occupational performance indirectly through procrastination, inefficiency, stubbornness, forgetfulness; frequent fault-finding with others

The origins of personality disorders are not well understood, but they clearly can be thought of as reflecting the contributions of genetic, constitutional (temperament), environmental (upbringing, relationships), sociocultural and maturational (psychological development) factors. The need for, and modalities of, treatment of personality disorders varies and can include psychotherapeutic and pharmacologic interventions.

(SEE ALSO: *Attention Deficit Disorder; Causes of Substance Abuse: Psychological (Psychoanalytic) Perspective; Comorbidity and Vulnerability; Conduct Disorder and Drug Use; Epidemiology of Drug Abuse; Personality As a Risk Factor, for Drug Abuse; Vulnerability As Cause of Substance Abuse*)

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PET SCANNER *See* Imaging Techniques

PEYOTE Peyote (or peyotl) is the common name for the cactus *Lophophra williamsii* or *Anhalonium lewinii*, which is found in the southwestern United States and northern Mexico. Although

there are many compounds found in the cactus, some of which may be PSYCHOACTIVE, the principal HALLUCINOGENIC substance found in peyote is MESCALINE. As the other psychoactive substances may make some contribution to the PSYCHEDELIC experience, there may be some slight difference in the behavioral effects produced by taking peyote and pure mescaline, but the overall effects of peyote are very similar to those produced by mescaline.

Peyote, one of the oldest psychedelic agents known, was used by the Aztecs of pre-Columbian Mexico who considered it magical and divine. Its use spread to other Native American groups who used it to treat various illnesses, as a vehicle to communicate with the spirits, and in highly structured tribal religious rituals. For these rituals, the dried tops of the cactus—the buttons—are chewed or made into a tea. Since peyote may cause some initial nausea and vomiting, the participant may prepare for the ceremony by fasting prior to eating the buttons. Peyote is usually taken as part of a formalized group experience and over an extended period of time; the peyote ceremonies may take place at night and around a communal fire to increase the hallucinogenic effects and visions.

(SEE ALSO: *Ayahuasca: Dimethyltryptamine; Psilocybin*)

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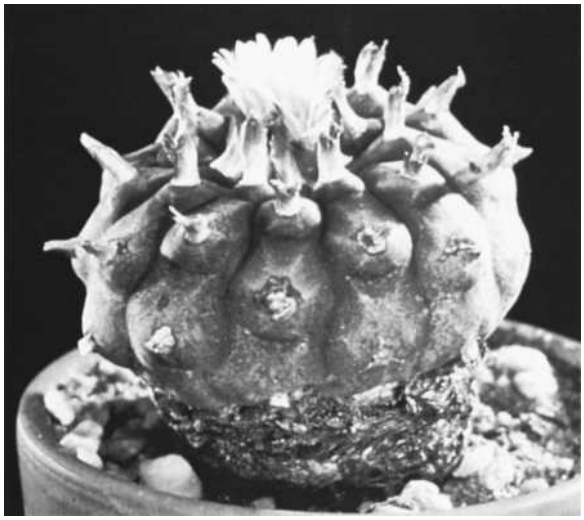
PHARMACODYNAMICS The study of the mechanism of drug actions is called pharmacodynamics. Most (but not all) drugs exert their action by binding to specific RECEPTORS. This binding may initiate changes that lead to the characteristic effects of the drug on body functions.

A central question in drug therapy (medication) is the proper dose of the drug that produces a desired action without many harmful side effects. To clarify this problem, pharmacologists analyze the relationship between dose and response. Most dose-response curves are sigmoidal (shaped like an S).



The peyote cactus, from which is derived the hallucinogenic mescaline. (Drug Enforcement Administration)

The log-dose-response can be viewed as having four parameters: potency, slope, maximal efficacy, and variability. Potency describes the strength of drug effects. It is usually employed to calculate relative strengths among drugs of the same class. Slope is the central part of the curve that is approximately straight. It is used to analyze drug concentration (dose) from the observed corresponding responses. Maximal efficacy, or simply "efficacy," is the greatest effect produced by the drug. This is one of the major characteristics of a drug. Efficacy and potency of a drug are not necessarily correlated, and the two characteristics should not be confused.

Many drugs, including drugs of abuse, produce TOLERANCE—when it becomes necessary to take progressively larger doses to achieve the same drug effect. In some cases, the brain and other tissues on which a drug acts undergo adaptive changes (neuroadaptations) that tend to offset the drug effect. When a drug that produces neuroadaptation is withdrawn, the brain and other tissues have to readapt, because they are no longer balanced by drug effect. The adaptation produces a variety of signs and symptoms called withdrawal syndrome. The severity of this syndrome depends on the degree of adaptive changes in the nervous system—which, in turn, depends on the dose and the duration of exposure to the drug. The particular characteristics of the withdrawal syndrome depend on the pharmacological effects of the drug(s) and typically

are opposite to the drug effects. For example, MORPHINE constricts the pupil; the morphine withdrawal symptom includes pupil dilation.

Most drugs of abuse produce pleasant effects in humans. For example, some people use AMPHETAMINES or other stimulants (e.g., COCAINE) to achieve a sense of well-being and euphoria. Some people use DEPRESSANTS—ALCOHOL, OPIOIDS, or TRANQUILIZERS—to relax. Still others use either stimulants or depressants to relieve boredom or reduce anxiety or pain. The common feature is that people use drugs because somehow the drug is rewarding to the user, either by producing a feeling of well-being (e.g., euphoria, elation) or by taking away a negative feeling (e.g., anxiety).

(SEE ALSO: *Addiction: Concepts and Definitions; Drug Interaction and the Brain; Drug Metabolism; ED50; LD50*)

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PHARMACOKINETICS: GENERAL

Pharmacokinetics describes quantitatively the various steps of drug disposition in the body including absorbtion of drugs, distribution of the drugs to various organs, and their elimination by excretion and biotransformation. The rates of these processes are important in characterizing the fate of a medication in the body.

The actual percentage of a drug contained in a drug product that enters the circulation unchanged after its administration, combined with the rate of entry into the body, determines the *bioavailability* of a drug.

Once absorbed, most drugs are carried from their site of action and elimination by the circulating blood. Some drugs simply dissolve in serum water, but many others are carried bound to proteins, especially albumin. Plasma *protein binding* influences the fate of drugs in the body, since only the free (unbound) drug reaches the site of drug action. This interaction with binding sites is reversible.

The intensity of drug action is most frequently related to the concentration of the drug at the site of action. The duration of drug effect is related to the persistence of its presence at this site. The time to reach maximum drug concentrations (or peak effects) is usually referred to as t_{max}.

Whenever the fate of a drug in the body is described by pharmacokinetic parameters, a model of the body is assumed. The fundamental principles of pharmacokinetics are based on the most elementary model. The body is considered a single compartment. Distribution of the drug is considered uniform. The "volume" in which the drug is distributed is referred to as the *volume of distribution* (Vd). It is typically expressed in liters per kilogram (L/kg).

Elimination of the drug is assumed to be exponential. The *rate of elimination* of a drug is usually described by its *half-life* ($t_{1/2}$), which is the time required for 50 percent elimination of the drug. This is typically expressed in hours (h). Another way to express drug elimination is the *clearance*, which represents the volume of drug cleared from the body per unit of time. This is usually expressed in milliliters per minute per kilogram (ml/min/kg) but can also be expressed in liters per hour per kilogram (L/h/kg).

An effect of a single dose of a drug may be characterized by its latency, the time needed for drug concentrations to reach maximum levels (t_{max}). Magnitude of peak effects and duration of action dosage and rates of absorption and elimination are influenced by these parameters. As dosage increases, latency is reduced and peak effect increased without change in the time of peak effect. Reduced elimination (long half-life, reduced clearance) results in an expected prolongation of drug effects and, in some cases, drug accumulation. Using more complex models than a single compartment model, physicians use pharmacokinetic data not only to characterize the fate of a drug in the body but also to calculate doses and frequency of drug administration for each particular patient. This is important because there are wide variations among individuals in the absorption, distribution, and elimination of drugs.

Tables 1 through 4 are a summary of the available data on the kinetic properties of alcohol and other abused drugs. Some of the drugs of abuse included in this summary are illicit drugs (e.g., COCAINE) while others are effective pharmacological agents that have the potential to be abused (e.g., OPIOIDS).

Although some of the drugs included in the tables have been used for centuries (e.g., ALCOHOL, CAFFEINE), knowledge of their kinetics and metabolism is very recent and, in some cases, still incom-

TABLE 1
Pharmacokinetic Parameters of Opioids

Drug	*Dosage/Route (mg)*	*Bioavailability (F) (%)*	*Protein Binding (%)*	t_{max} *(h)*	*Mean* $t_{1/2}$ *(h) (range)*	*Vd (L/kg)*	*Cl (ml/min/kg)*
Butorphanol	2/IV	100 (IM)	80	0.75	3–4	5	385
Codeine	60 oral/IV	40–80 (oral)	7–53	1	3 (2.3–9.3)	2–6	15
Dextromethorphan		>50 (oral)	30–50	—	2–3 (estimated)	3–5	—
Heroin (3,6 diacelylmorphine) (see morphine)	4–16/IV	79 (oral)	—	—	3.0 minutes	—	31
Buprenorphine	0.3/IV	40–90 (oral)	—	—	2–3	1–3	900–1,200 (ml/min)
Pentazocine	—	47 (oral)	65	—	4.5	7	17
Morphine	0.01/mg/kg	15–64 (oral) 100 (IM) 48 (rectal) 2 (epidural)	35	<1	2–4	3–4	12–21
Methadone	15–80	92 (oral)	40	<1	25 (13–47)	3.8	1.4
Meperidine	50–100/IM	50–60 (oral)	50–60	—	3–4	3–5	—
Propoxyphene	130	40–90 (oral)	—	1–2	2–15	—	—
Nalbuphine	—	16 (oral)	—	1–2	2–3	3–4	22
Naltrexone	—	5–40 (oral)	20	—	2–3	19	48

Vd = Volume of distribution Cl = Clearance IV = Intravenous IM = Intramuscular

TABLE 2
Pharmacokinetic Parameters of Stimulant Drugs

Drug	*Dosage/Route (mg)*	*Bioavailability (F) (%)*	*Protein Binding (%)*	t_{max} *(h)*	*Mean* $t_{1/2}$ *(h) (range)*	*Vd (L/kg)*	*Cl (ml/min/kg)*
Amphetamine	15–25/oral	—	23–26	1.25	14 (2–22)	6.1	0.2–0.6 (L/min)
Caffeine	1–5 mg/kg/oral	100 (oral)	15–40	0.5–1	5 (1–10)	0.6	1
Cocaine	30–100/IV;IN	28–51 (IN)	7	0.5–1.5	0.8 (0.3–1.5)	2	11
Nicotine	0.25–2 (mg/kg/min)/IV	30	5	—	2 (0.8–3.5)	1–2	18

Vd = Volume of distribution Cl = Clearance IV = Intravenous IN = Intranasal

TABLE 3
Pharmacokinetic Parameters of CNS Depressants

Drug	*Dosage/Route (mg)*	*Bioavailability (F) (%)*	*Protein Binding (%)*	t_{max} *(h)*	*Mean* $t_{1/2}$ *(h) (range)*	*Vd (L/kg)*	*Cl (ml/min/kg)*
Alcohol (ethanol)	—	80 (oral)	—	<1	0.25	0.5	124 mg/kg/h
Benzodiazepines:							
Alprazolam	0.5–30/oral	90 (oral)	70	0.7–1.6	12 (6–18)	0.7–1.5	0.7–1.3
Bromazepam	0.25–3/oral	—	70	1	10–15	—	—
Chlordiazepoxide	20–50/oral IV, IM	100 (oral) PO or (IM)	95	0.5–3	10 (6–28)	0.3	0.5
Clobazam	10–20/oral	Good (oral)	90	1.3–1.7	25 (16–49)	0.9–1.8	0.36–0.63
Clonazepam	—	98	86	1–2	23 (20–80)	3.2	1.55
Clorazepate (see Desmethyldiazepam)	—	—	—	—	2.0	0.33	1.8
Desalkylflurazepam	—	—	—	1	75 (40–200)	22	4.5
Desmethyldiazepam	—	99	97	1–2	51 (51–120)	0.78	0.14
Diazepam	1–40/oral IM, IV	100 (oral) 50–60 (IM, rectal)	96	0.5–2	31 (14–61)	1 (0.9–3.0)	0.38–0.51
Flurazepam (see Desalkylflurazepam)	15–90/oral	—	97	—	—	—	—
Halazepam (see Desmethyldiazepam)	—	—	—	—	—	—	—
Lorazepam	2–4/oral	93 (oral) 90 (IM)	90	1.5	13 (8–25)	0.8–1.6	1 (0.8–1.3)
Midazolam	5–15/oral IV, IM	44 (oral)	95	0.3–0.7	2 1.4–5	0.8–17	6
Nitrazepam	15–30/oral	78 (oral)	87	2	26 (16–48)	1.2–2.7	0.86
Oxazepam	15–45/oral	97 (oral)	98	3 (0.5–8)	7 (5.1–13)	0.5–2.0	0.6–2.9
Prazepam (see Desmethyldiazepam)							
Temazepam	10–30/oral	>80 (oral)	98	0.8–4.7	12 (7–17)	1.3–1.5	1.0–3.4
Triazolam	0.25–1.0/oral	44 (oral) 53 s.l.	90	1.6	2.5 (2–5)	1.1	3.7–8.8

Vd = Volume of distribution Cl = Clearance IV = Intravenous IM = Intramuscular s.l. = Sublingual

plete. This is due partly to their complex metabolism and partly to the difficulties of studying drugs of abuse in humans.

The tables show the route of administration, the type of subjects used in the study, the doses used, and the most important kinetic parameters such as protein binding, half-life, volume of distribution, and clearance.

(SEE ALSO: *Drug Metabolism; Pharmacogenetics; Pharmacokinetics of Alcohol*)

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TABLE 4
Pharmacokinetic Parameters of Hallucinogens

Drug	*Dosage/Route (mg)*	*Bioavailability (F) (%)*	*Protein Binding (%)*	t_{max} *(h)*	*Mean* $t_{1/2}$ *(h) (range)*	*Vd (L/kg)*	*Cl (L/min)*
Marijuana (Δ^9-tetrahydrocannabinol)	0.5–30	8–24 (smoked) 4–12 (oral)	95–26	3–8 min	25 (19–57)	626(L)	0.2–1
Phencyclidine (PCP)	0.1–0.7/IV Inhaled	5–90	65	1.5	24 (7–51)	6.8	0.30 (0.14–0.77)

Vd = Volume of distribution Cl = Clearance IV = Intravenous

PHARMACOKINETICS: IMPLICATIONS FOR ABUSABLE SUBSTANCES

Pharmacokinetics is the study of the movements and rates of movement of drugs within the body, as the drugs are affected by uptake, distribution, binding, elimination, and biotransformation. An understanding of the biological basis of the clinical actions of abused drugs depends, in part, on knowledge of their neurochemical and neurorecptor actions that reinforce and sustain drug use (Hall, Talbert, & Ereshefsk, 1990). The pharmacokinetic properties of abusable substances represent a second important component of the database. The discipline of pharmacokinetics applies mathematical models to understand and predict the time course of drug amounts (doses) and their concentrations in various body fluids (Greenblatt, 1991, 1992; Greenblatt & Shader, 1985). Pharmacokinetic principles can be used to provide quantitative answers to questions involving the relationship of drug dosage and route of administration to the amount and time course of the drug present in systemic blood and at the receptor site of action.

Before an orally administered PSYCHOACTIVE DRUG can exert a pharmacological effect through its molecular recognition site in the brain, a number of events must take place (see Figure 1). The drug must reach the stomach and dissolve in gastric fluid. The stomach empties this solution into the proximal small bowel, which is the site of absorption of most medications. The drug must diffuse across the gastrointestinal mucosal barrier, reach the portal circulation, and be delivered to the hepatic (liver) circulation. (The liver detoxifies chemicals, including drugs.) Before reaching the systemic circulation, then, the absorbed drug must "survive" this initial exposure to the hepatic circulation—sometimes termed the "first-pass" through the liver (Greenblatt, 1993). After reaching the systemic blood, the drug is transported to the cerebral (brain) capillary circulation as well as to all other sites in the body that receive blood directly from the heart (cardiac output). The drug diffuses out of the

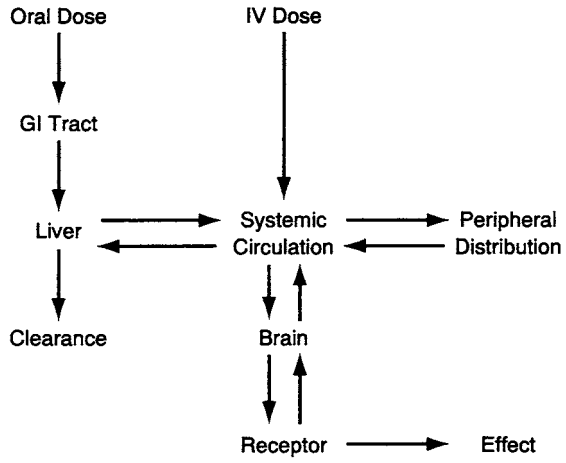

Figure 1
Schematic Representation of Physiological and Pharmacokinetic Events. These occur between administration of a centrally acting compound and the production of a pharmacological effect. If the medication is given orally, it must pass from the gastrointestinal (GI) tract to the portal circulation and to the liver before reaching the systemic circulation. Intravenous administration, however, yields direct access to the systemic circulation. Drugs of abuse may be taken by the intravenous route but are also taken by intranasal, intrabuccal, or inhalational routes, all of which will avoid the initial gastrointestinal-portal-hepatic exposure.

cerebral capillary circulation, crosses the lipoidal (fatty) blood-brain barrier, and reaches the extracellular water surrounding the neuroreceptor site of action. Only then is the drug available to interact with its specific molecular recognition site.

All of these processes take time, and some may serve as obstacles that delay or prevent the drug from reaching its site of action. Pharmacokinetic models incorporate the physiology of these processes, and can allow rational prediction of important clinical questions: How much drug reaches the brain? How fast does it get there? How long does it stay there?

DRUG ABSORPTION

The term *lag time* refers to the time elapsing between ingestion of an oral medication and its first appearance in the systemic circulation (see Figure 2). For most drugs, it generally falls between 5 and 45 minutes. For ethanol (drinking ALCOHOL, which is also called ethyl alcohol), however, the lag time may be very short, because the drug is already a liquid at the time it is ingested, and a significant component of absorption probably occurs across the gastric mucosa as well as in the proximal small bowel (Frezza et al., 1990). The physicochemical features of the drug contribute importantly to the time necessary for dissolution and therefore to the lag time. All else being equal, drugs in solution have shorter lag times than those administered in suspension form; they are, in addition, more rapidly absorbed than capsule preparations and, finally, tablet preparations. For any given solid dosage form, lag time and absorption rate are likely to be shorter if the drug particles are more finally subdivided. Sustained-release (time-release) drug formulations are deliberately prepared to have long lag times and slow absorption rates, thereby avoiding drug effects associated with the peak concentration.

Absorption rate refers to the time necessary for the drug to reach the systemic circulation once the absorption process actually begins. Pharmacokinetic models can be applied to assign a half-life value to the process of absorption. Values of absorption half-life tend, however, to be of low statistical stability, and it is increasingly common to characterize the absorption process using the observed peak plasma concentration (c_{max}) and time of peak concentration (t_{max}). The t_{max} is actually a

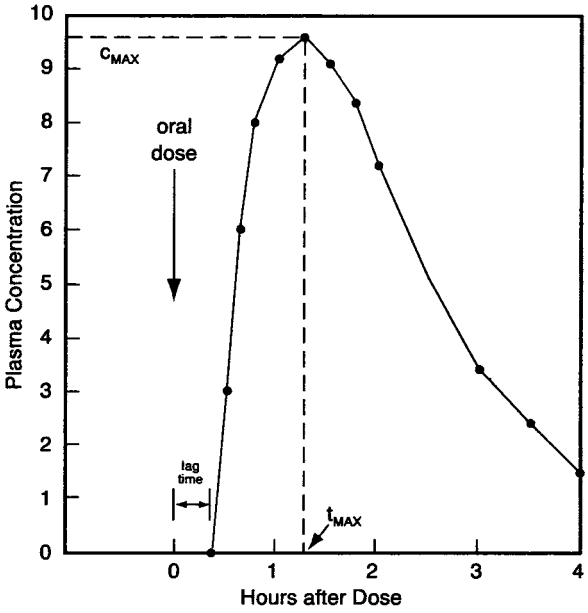

Figure 2
Schematic Plot of Plasma Concentration versus Time after Oral Dosage (given at time zero [arrow]). A lag time elapses between the time of administration and the beginning of appearance in the systemic circulation. Plasma levels then rise, reach a peak, and fall: c_{max} is the peak plasma concentration (9.6 units) and t_{max} is the time of peak concentration (1.25 hours after dosage).

composite of the lag time plus the time necessary to reach peak concentration once absorption starts (Figure 2). In general, fast absorption implies a high value of c_{max} and a short value of t_{max}; slow absorption implies a long t_{max} and a low c_{max}. Again, sustained-release drug preparations are deliberately formulated to produce long lag times and slow absorption, thereby delaying and reducing the c_{max} after an oral dose. Drug absorption tends to be slower when medications are taken during or just after a meal, rather than in the fasting state (before a meal, on an empty stomach).

For these reasons, the ethanol in alcoholic beverages is relatively rapidly absorbed after oral ingestion. The popular lore that alcohol bas a greater effect when taken on an empty stomach probably has a physiological basis, since peak concentrations will be higher and earlier when alcohol is taken in the fasting state. BENZODIAZEPINE derivatives (tranquilizers) clearly are not primary drugs of

abuse and are seldom subject to misuse by the great majority of patients; however, benzodiazepines may be taken for nontherapeutic purposes by some substance abusers (Woods, Katz, & Winger, 1987, 1992; Shader & Greenblatt, 1993). The preference of specific benzodiazepines by drug abusers appears to be closely related to their rate of absorption. That is, rapidly absorbed benzodiazepines, leading to relatively high values of c_{max} shortly after dosage, appear to be preferred by drug abusers. The benzodiazepine diazepam (Valium), for example, is much more rapidly absorbed than is oxazepam (Serax or Serenid). In controlled laboratory settings, diazepam is more easily recognized as a potentially abusable substance by experienced drug users, and it is also preferred by this group to oxazepam (Griffiths et al., 1984a, 1984b). This preference also appears to be supported by epidemiological studies of PRESCRIPTION DRUG misuse (Bergman & Griffiths, 1986).

Some orally administered medications reach the systemic (blood) circulation in small or even negligible amounts relative to the dose ingested. Incomplete absorption from the gastrointestinal tract sometimes explains this. However, oral medications may be poorly available to the systemic circulation even if they are well absorbed. This is explained by the phenomenon termed *presystemic extraction*, which results from the unique anatomy and physiology of the gastrointestinal circulation (Greenblatt, 1993). Orally administered medications are absorbed into the portal rather than systemic circulation (Figure 3), and portal blood drains directly into the liver. Many drugs that are avidly metabolized in the liver may therefore undergo substantial biotransformation before reaching systemic blood. Some drugs may also be metabolized by the gastrointestinal (GI) tract mucosa. First-pass hepatic metabolism together with GI tract metabolism is collectively termed presystemic extraction. COCAINE, for example, is not favored as a drug of abuse by the oral route, because of nearly complete presystemic extraction, allowing only small amounts of the intact drug to reach the systemic circulation (Jatlow, 1988; Jeffcoat et al., 1989).

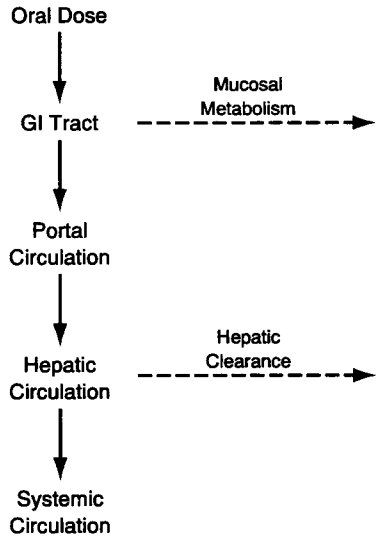

Figure 3
Possible Mechanisms of Presystemic Extraction. Orally administered medications may undergo metabolism as they pass through the gastrointestinal tract mucosa (dashed arrow), which contains significant amounts of Cytochrome P45-3A4. Mucosal metabolism of cyclosporine appears to occur in humans (Kolars et al., 1991). Metabolism may also occur as drug present in portal blood passes through the hepatic circulation (dashed arrow); this is termed "first-pass" metabolism. The net extent of presystemic extraction depends on the combination of mucosal metabolism and first-pass metabolism.

DRUG DISTRIBUTION

The process of distribution is an important determinant of pharmacokinetic properties, as well as the time course of action, of most centrally acting drugs, including those that are subject to abuse. Drugs reversibly distribute not only to their site of action in the brain but also to peripheral sites such as adipose (fat) tissue and muscle, where they are not pharmacologically active (Figure 1). Only a small fraction of the total amount of a psychotropic drug in the body goes to the brain. An even smaller fraction actually binds to the specific molecular recognition site (receptor). The extent of distribution of a psychotropic drug is determined in part by lipid (fat) solubility (how well a substance dissolves in oils and fats; lipophilicity), which is related to molecular structure and charge. Most psychotropic drugs are highly lipid-soluble. Drug distribution is also determined by some characteristics of the organism: the relative amounts of adipose and lean

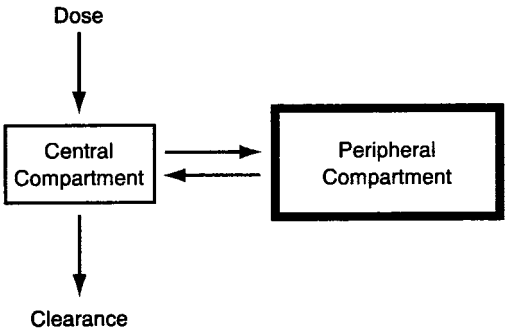

Figure 4
Schematic Diagram of the Two-Compartment Model. It is assumed that medications are administered into and cleared from the central compartment only, and that only the central compartment (which includes blood) is accessible to measurement. Reversible distribution occurs between central and peripheral compartments. For most psychotropic drugs, high lipid solubility favors distribution to the peripheral compartment, producing a large apparent pharmacokinetic volume of distribution.

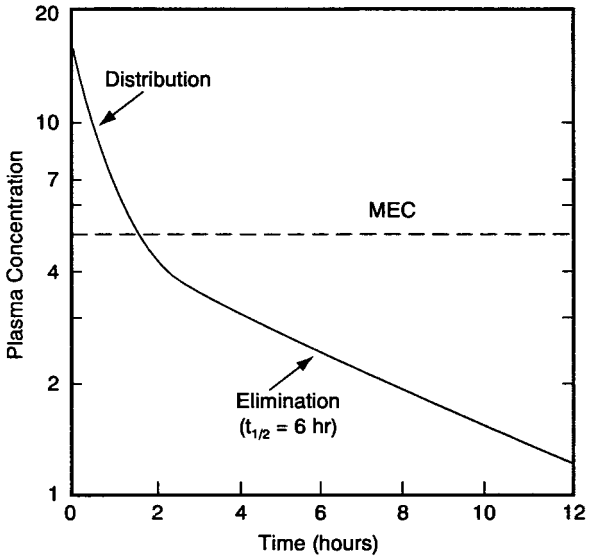

Figure 5
Plasma Concentrations of a Hypothetical Lipid-Soluble Drug after Intravenous Injection. Disappearance from plasma is biphasic. The initial rapid phase is mainly due to drug distribution from central to peripheral compartments (see Figure 4). The slower phase of elimination is mainly due to clearance. For this drug, the elimination half-life in the postdistributive phase is 6 hours. If a plasma concentration of 5 units represents the minimum effective concentration (MEC) below which the drug exerts no detectable pharmacological effect, this drug in the dosage administered has a duration of action of approximately 2 hours.

tissue, blood flow to each individual tissue, and the extent of drug that binds to plasma protein. The overall extent of drug distribution throughout the body can be quantified by the pharmacokinetic volume of distribution, which is a ratio—the total amount of drug present in the body divided by the concentration in a reference compartment, usually serum or plasma. Lipid-soluble psychotropic drugs, as well as drugs of abuse, typically have very large pharmacokinetic volumes of distribution, which may exceed body size by ten-fold or more. Although the drug cannot actually distribute to a space larger than the body, low plasma concentrations resulting from extensive uptake into peripheral tissues can yield a large apparent pharmacokinetic volume of distribution (Figure 4).

Drug distribution influences both the onset and the duration of drug action—as well as the observed value of elimination HALF-LIFE. After an intravenous (IV) injection, lipid solubility allows for the rapid crossing of the lipoidal blood-brain barrier, leading to a rapid onset of pharmacological action (drug effect). In behavioral terms, then, drug-taking produces immediate reinforcement. The duration of a drug's action, however, is determined mainly by the extent of its peripheral distribution. Plasma levels of lipid-soluble psychotropic drugs will decline rapidly and extensively after a single intravenous dose, because of peripheral distribution rather than elimination or clearance (Figure 5). A similar principle holds after oral administration of rapidly absorbed drugs (de Wit & Griffiths, 1991). Since duration of action after a single dose is determined more by distribution than by elimination or clearance, it is generally not accurate to equate elimination half-life and duration of action.

CLEARANCE AND ELIMINATION

The terms *clearance* and *elimination half-life* are commonly used to describe the bodily process of drug removal or disappearance. These two con-

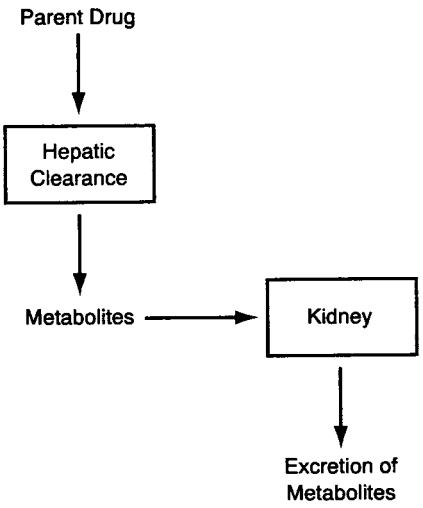

Figure 6
Psychotropic Drugs: Most, including drugs of abuse, are cleared via the liver by hepatic biotransformation to metabolic products. The metabolites may then be released into the circulation and excreted by the kidney.

cepts are related but are not identical. Clearance is the most important, since it is a unique independent variable that best describes the capacity of a given organism to remove a given drug from its system. Clearance has units of volume divided by time—for example, milliliters/minute (ml/min) or liters/hour (L/h)—and is the total amount of blood, serum, or plasma from which a substance is completely removed per unit of time. Clearance is *not* identical either to the rate of drug removal or to the elimination half-life. For most psychotropic drugs, clearance is accomplished by the liver via processes of bio-transformation that change the administered drug into one or more metabolic products (Figure 6); this is commonly called detoxification by the liver. The metabolites may appear in the urine, but the liver is still the organ that effects clearance. For drugs cleared exclusively by the liver, the numerical value of clearance cannot exceed hepatic blood flow.

Elimination half-life is described in units of time; it can be seen as the time necessary for the plasma concentration to fall by 50 percent after distribution equilibrium has been attained. The elimination phase of drug disappearance—at which time the concept of elimination half-life is applicable—may not be attained until completion of an initial phase of rapid drug disappearance resulting from peripheral distribution (see Figure 5). As discussed earlier, the duration of action of a single dose of a psychotropic drug is not necessarily related to its elimination half-life.

Pharmacokinetic theory yields the following relationship between a drug's elimination half-life, volume of distribution (Vd), and clearance:

$$\text{Elimination half-life} = \frac{0.693 \cdot \text{Vd}}{\text{clearance}}$$

The independent variables, appearing on the right side of the equation, are *Vd*, the physicochemically determined property reflecting the extent of distribution, and *clearance*, having units of volume divided by time, quantifying the capacity for drug removal. Elimination half-life is dependent on both of these. Note that a drug may have long elimination half-life, due either to a large Vd, a low clearance, or both.

PHARMACOKINETICS VERSUS PHARMACODYNAMICS

In contrast to pharmacokinetics, PHARMACODYNAMICS is the quantitative study of the time course of drug action. If drug distribution to the site of action occurs by passive diffusion from the systemic circulation, and if the intensity of drug action depends on the degree of RECEPTOR occupancy both in time and in quantity, then pharmacokinetics and pharmacodynamics are necessarily related. Kinetic-dynamic modeling, discussed in detail elsewhere (Greenblatt & Harmatz, 1993), addresses this relationship mathematically, by directly evaluating concentration versus effect. In the fields of psycho-pharmacology and substance abuse, kinetic-dynamic modeling is a major challenge, since (1) clinical drug effect (pharmacodynamic response) often is difficult to measure reliably and since (2) measured drug concentrations in systemic serum or plasma do not always parallel those at the central site of action. Nonetheless, recent advances in kinetic-dynamic modeling have significantly advanced our understanding of the relationship of the pharmacokinetics of psychotropic drugs to their pharmacodynamic effects.

TABLE 1
Principal Urinary Metabolites of Potentially Abusable Drugs

Parent Drug	*Urinary Metabolite*
Marijuana (Tetrahydrocannabinol, THC)	11-nor-delta-9-THC-9-carboxylic acid
Cocaine	Benzoylecgonine
Heroin	Morphine glucuronide

IMPLICATIONS FOR TESTING OF URINE FOR SUBSTANCES OF ABUSE

Mandatory unannounced testing of urine samples for illegal drugs of abuse is conducted to detect and deter the use of these drugs, as well as to prevent potentially dangerous impairment of performance. The application of the fundamental principles of pharmacokinetics and pharmacodynamics, however, clearly indicates that urine testing is the wrong way to approach these objectives (Greenblatt, 1989; Greenblatt & Shader, 1990).

HEROIN, cocaine, and MARIJUANA, the principal illegal drugs of abuse, are subject to hepatic clearance, so urinary excretion is in the form of drug metabolites rather than the originally taken parent compounds (Agurell et al., 1986; Jatlow, 1988) (see Figure 6). As such, analytical methods for chemical testing of urine samples must be devised to detect these metabolites (Friedman & Greenblatt, 1986) (see Table 1). Screening IMMUNOASSAYS are notoriously insensitive, and many actual drug users will escape detection by the screening test if the urine concentrations are below an arbitrary cutoff (Burnett et al., 1990). Negative tests can also be produced by dilution of urine via water loading (Lafolie et al., 1991) or by a variety of adulterants that interfere with analytical procedures (Schwarzhoff & Cody, 1993; Mikkelson & Ash, 1988). To complicate matters, immunoassays are nonspecific and have an unacceptably high false-positive rate. Most urine-testing programs deal with the false-positive problem by performing confirmatory tests on all positive results from the initial screening (Figure 7). However, even a positive test that is confirmed by gas chromatography/mass-spectroscopy does not conclusively identify that individual as a drug user. Positive urine tests may be produced by passive inhalation or dermal absorption, as (ironically) may occur in law-enforcement officials engaged in drug-enforcement activities (Baselt, Chang, & Yoshikawa, 1990; Elsohly, 1991). Recent evidence suggests that some nondrug-using individuals may excrete heroin metabolites resulting from foodstufts (poppy-seed cake) or from endogenous metabolism (Hayes, Krasselt, & Mueggler, 1987; Mikus et al., 1994). Thus evaluation of the problems of analytical chemistry inherent in urine testing indicates that a

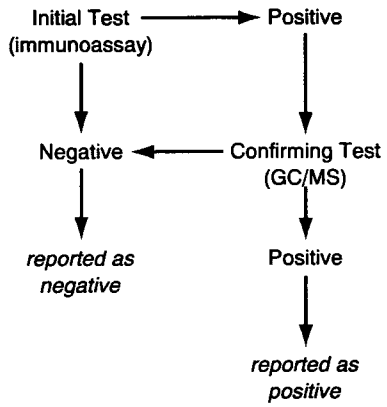

Figure 7
Urine-Testing Programs. Those for drugs of abuse typically use a two-tiered algorithm. An initial screening test is done with a relatively inexpensive, nonspecific, and insensitive immunoassay (such as enzyme-multiplied-, fluorescence-polarization-, or radioimmunoassay). If the initial test is negative, the result is reported as such, and no further testing is done. If the initial screen is positive, a second analysis is done on the same sample using a more accurate and specific method, such as gas-chromatography/mass-spectroscopy (GC/MS). If the confirmation test is negative, the result is reported as negative. If GC/MS confirms the initial screening test, the result is reported as positive.

negative test cannot rule out illegal drug exposure, nor can a positive test confirm it.

From a pharmacokinetic—pharmacodynamic standpoint, urine is an excretory product and not a body fluid. Urine concentrations of drug metabolites bear little relation to parent-drug concentrations in blood or at the site of action—the concentrations that actually determine pharmacodynamic effect (Osterloh, 1993). Even if chemically accurate, a "positive" urine test for a substance of abuse provides no useful information on the quantity of drug exposure, the duration or chronicity of exposure, or the pharmacodynamic effect of the drug at the time the urine sample was taken, or any time prior to or after that. A positive test does not confirm intoxication or impairment from that drug at any time, nor does a negative test rule them out. Thus, as a general rule, urine-testing programs are without adequate scientific foundation and cannot possibly attain the stated objectives (Greenblatt, 1989; Greenblatt and Shader, 1990; Sutherland, 1992). This does not mean that carefully controlled tests do not exist—for a discussion of this see DRUG TESTING AND ANALYSIS.

Detection and prevention of performance impairment in the workplace can, however, be achieved by the systematic testing of performance, using validated methods under properly controlled conditions. Such testing procedures would detect potentially dangerous impairment not only from illegal drugs of abuse but also from other causes, including use of legal substances (such as alcohol or antihistamines), sleep deprivation, other medical or psychiatric illness, or episodes of interpersonal stress. Chemical analysis of blood (not urine) could provide chemical confirmation for cases in which drug-induced performance impairment is suspected, provided a research database is available to link blood concentrations to probable impairment, as exists in the case of alcohol (ethanol). Such an approach would provide a fair and direct method of coping with this problem.

COMMENT

A comprehensive approach to understanding the biological bases of substance abuse must combine the neurochemical and molecular mechanisms that underlie the behavioral effects of these drugs, as well as understanding their properties of absorption, distribution, and clearance. Advances were made in the 1980s and will continue to be made as research techniques in both disciplines become increasingly refined.

ACKNOWLEDGMENTS

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(SEE ALSO: *Abuse Liability of Drugs: Testing in Humans; Benzodiazepines; Benzodiazepines: Complications; Pharmacokinetics of Alcohol; Psychomotor Effects of Alcohol and Drugs*)

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DAVID J. GREENBLATT

PHARMACOKINETICS OF ALCOHOL

The discipline known as pharmacokinetics deals with the way drugs are absorbed, distributed, and eliminated by the body and how these processes can be described in quantitative terms. The pharmacokinetics of alcohol (ethyl alcohol or ethanol) is an important issue in forensic toxicology and clinical medicine, when the amount of alcohol in the body is estimated from the concentration measured in a blood sample.

The Swedish scientist Erik M.P. Widmark (1889–1945) made pioneer contributions to knowledge about the pharmacokinetics of ethanol during the early decades of the twentieth century. Widmark observed that after the peak concentration in blood had been reached, the disappearance phase seemed to follow a near straight-line course, suggesting that the system for metabolizing alcohol was saturated (fully occupied), so that the amount of alcohol metabolized each hour did not depend on the amount in the blood. This situation is termed a *zero-order elimination process*. (Zero-order kinetics is contrasted with first-order kinetics, in which the metabolic system [e.g., the liver] is not saturated and in which the amount of drug metabolized per hour increases as the amount presented to the metabolic system increases.) Figure 1 (left

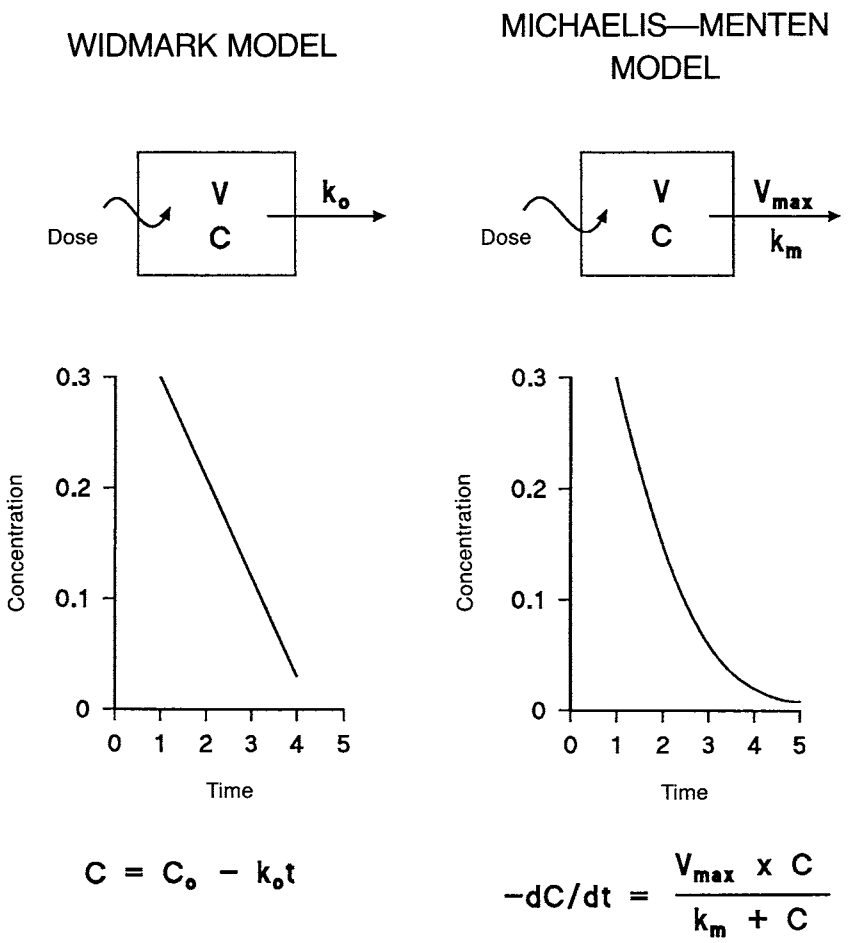

Figure 1
Elimination Kinetics of Ethanol. Schematic diagram illustrating the elimination kinetics of ethanol. The left frame shows Widmark's zero-order model. The right frame shows Michaelis-Menten (MM) capacity-limited kinetics. An intravenous bolus dose of ethanol enters a volume V to produce a concentration C; k_o is the zero-order elimination rate constant; V_{max} is the maximum velocity of the reaction; and k_m is the Michaelis constant—the concentration of ethanol at half maximum velocity. Concentration-time profile are shown for zero-order and MM kinetics, and the mathematical expressions for the elimination rates are given.

frame) depicts zero-order elimination kinetics of ethanol after rapid intravenous infusion. Widmark used the Greek letter β to represent the negative slope of the disappearance phase and not the notation k_o used in Figure 1. The terminology and choice of symbols used in articles and books dealing with clinical pharmacokinetics are often confusing. Moreover, the concentrations of ethanol in blood and other body fluids are reported using many different units, such as g% w/v, mg/dl, g/l, mmol/l; 21.7 mmol/l = 100 mg/dl = 1 g/l = 0.1 g% w/v.

Zero-order kinetics implies that the elimination rate of ethanol is independent of the BLOOD ALCOHOL CONCENTRATION (BAC) and therefore k_o should be the same regardless of the dose of ethanol administered; however, more recent studies have show that the slope of the BAC decay phase is steeper after larger doses of ethanol are ingested. Furthermore, when the BAC declines below about 10 mg/dl (0.01 g%, 2.17 mmol/l) the elimination curve of ethanol from blood flattens out and changes into a curvilinear decay profile.

Two different methods are described in the literature to portray the pharmacokinetics of ethanol. The method of choice seems to depend on the professional interests, the scientific background, and the training of those concerned. Specialists in forensic medicine and toxicology, as well as other disciplines, favor the mathematical approach developed by Widmark. In contrast, scientists with their basic training in pharmacy and pharmacology prefer Michaelis-Menten (MM) kinetics, that is, saturable or capacity-limited enzyme kinetics. The MM model is depicted in Figure 1 (right frame) after intravenous input of ethanol. A pseudolinear phase is evident for most of the elimination profile, provided that the BAC remains sufficiently high (> 10 mg/dl). At low substrate concentrations (C), a hockey-stick shape develops when data are plot-

ted on cartesian graph paper. Accordingly, when C is much greater than k_m, the elimination rate approaches its maximum velocity; $-dC/dt = V_{max}$ (Figure 1, right frame). When C is less than k_m the elimination rate is proportional to the substrate concentration; $-dC/dt = (V_{max}/k_m)\ C$ and the MM equation collapses into first-order kinetics. This collapsing of the model is a consequence of capacity-limited kinetics and does not reflect any sudden change in the order of the biochemical reaction.

ETHANOL AS A DRUG

Ethanol differs from most other drugs in the way it is absorbed into the blood, metabolized in the liver, and how it enters the brain and produces its pharmacological effect. Ethanol (CH_3CH_2OH) has a molecular weight of 46.05, mixes with water in all proportions and carries only a weak charge; this means that the molecules of ethanol easily pass through biological membranes, including the blood-brain barrier. After absorption into the portal blood, ethanol passes through the liver, where enzymes begin the conversion into acetaldehyde and acetate. The end products of ethanol metabolism are carbon dioxide and water. The concentrations of ethanol in biological specimens depend on the dose ingested, the time after drinking, and the water content of the materials analyzed. The concentration-time profiles of ethanol and the pharmacokinetic parameters will differ depending on whether plasma, serum, urine, or saliva are the specimens analyzed. Several detailed reviews of ethanol pharmacokinetics are available and included in the bibliography.

Information about the absorption kinetics of ethanol is much less extensive than that about elimination kinetics. Unlike most other drugs, the dose of ethanol is not swallowed instantaneously because the drinking is usually spread over a period of time. For research purposes, however, ingestion of a bolus dose usually infers drinking times of five to fifteen minutes. The dosage form of ethanol, whether ingested as beer (3–6% w/v), wine (9–12% w/v), spirits (32–40% w/v), or as a cocktail (15–25% w/v) might influence the pharmacokinetic parameters. Absorption of ethanol starts in the stomach where about 20 percent of the dose can become absorbed. The remainder is absorbed from the upper part of the small intestine. The speed of absorption of alcohol depends to a large extent on the rate of gastric emptying, which varies widely among different subjects. Assuming that the rate of absorption from the gut is a first-order process, one can represent the entire concentration-time profile of ethanol with a single equation:

$$C = C_o(1 - e^{-kt}) - k_o t$$

Where C = BAC at some time t after administration
C_o = Initial BAC extrapolated BAC (see Figure 2)
k = First-order absorption rate constant
k_o = Zero-order elimination rate constant
t = Time after drinking

The peak BAC and the time of reaching the peak after drinking are important aspects of the absorption kinetics. Table 1 gives examples of these parameters after healthy men drank neat whiskey (40% v/v or 80 proof) on an empty stomach. The absorption of ethanol occurs more slowly from the stomach than from the intestine owing to the enormous difference in the absorption surface available. Factors that influence gastric emptying, such as food in the stomach before drinking, will alter the rate of absorption and the peak BAC reached. The absorption of ethanol occurs progressively during a drinking binge or spree, and studies have shown that the BAC fifteen minutes after the last drink has reached about 80 percent of the final peak BAC. Because of the saturation-type kinetics, the peak BAC and the area under the curve (AUC) increase more than expected from proportional increases in the dose. The slower the rate of delivery of ethanol to the liver the smaller the AUC for a given dose and vice versa. The systemic availability (bioavailability) of drugs like ethanol with dose-dependent kinetics should not be calculated from the ratio of AUC after oral and intravenous administration.

THE WIDMARK EQUATION

Figure 2 gives examples of the concentration-time profiles of ethanol obtained from oral and intravenous administration of a moderate dose. The ratio of the dose administered (D) to the initial extrapolated concentration of ethanol in blood (C_0) is the apparent volume of distribution (V_d) having dimensions L/kg. This defines the relationship between the concentration of ethanol spread over the body weight (in kilograms, kg) and the concentration in the blood.

TABLE 1
Peak Blood Alcohol Concentration and Time to Reach the Peak after End of Drinking

Dose		*Peak BAC mg/dl*		*k_o mg/dl h*		*Time to peak (min)*[3]			
g/kg[1]	*N*	*mean*	*(range)*	*mean*	*(range)*[2]	*10*	*40*	*70*	*100*
0.34	6	56	(43–67)	12	(9–14)	5	1	—	—
0.51	16	74	(54–91)	13	(10–14)	11	3	1	1
0.68	83	92	(52–136)	13	(9–17)	33	26	21	3
0.85	44	120	(83–178)	15	(12–18)	13	24	7	—

Maximum concentration of ethanol in capillary (fingertip) blood and the time of reaching the peak after end of drinking. The zero-order rate of elimination of ethanol from blood (k_o) is also given. The subjects drank neat whiskey within 15–25 minutes after an overnight 10-hour fast.

[1]g ethanol/kg = 0.036 oz ethanol/kg.
[2]Zero-order elimination rate.
[3]Number of subjects reaching their peak BAC at 10, 40, 70 and 100 min., measured from end of drinking.

$$C_o = D/(kg \times V_d)$$
$$D = C_o \times kg \times V_d \quad [1]$$

Equation [1] is known as the *Widmark equation*; it is widely used to estimate alcohol in the body from measurements of alcohol in the blood. Widmark found that the average V_d for men was 0.68, with a range from 0.51–0.85, but in women the volume of distribution was less—with an average of 0.55 and a range of 0.44–0.66. These differences between the sexes stem from differences in body-tissue composition; proportionally, women carry more fat but less water than do men. Accordingly, women reach higher BACs than men if the same dose of ethanol is given according to body weight. A similar observation was made in studies of men with widely different ages, because body water decreases in the elderly. By dividing the dose of ethanol administered (g/kg) by the time needed to reach zero BAC ($time_0$) one obtains an estimate of the rate of clearance of ethanol from the body. This calculation neglects the nonlinear phase of ethanol elimination beginning at BAC below 10 mg/dl but does include the contribution from any first-pass metabolism occurring in the liver and gut.

If equation [1] is combined with the expression for zero-order elimination kinetics ($C = C_0 - k_0t$) rearrangement gives equations [2] and [3]:

$$D = kg \times V_d \times (C + k_ot) \quad [2]$$

or

$$C = D/(kg \times V_d) - (k_ot) \quad [3]$$

Equation [2] can be used to estimate the amount (dose D) of alcohol a person has consumed from knowledge of his or her BAC (C). Similarly, equation [3] allows estimating the BAC (C) that might exist after drinking a known amount of ethanol. For best results when using these equations, absorption and distribution of ethanol must be complete at the time of sampling blood. Owing to inter- and intra-individual variations in the pharmacokinetic parameters V_d and k_0 the results obtained are subject to considerable uncertainty. This uncertainty should be allowed for when these calculations are made for legal purposes, for example, in trials concerned with DRIVING UNDER THE INFLUENCE of alcohol. A variability of ± 20 percent seems appropriate for most situations.

RESEARCH ON ADH

The enzymes responsible for ethanol oxidation are mostly located in the liver, but recent research has focused on the existence of alcohol dehydrogenase (ADH)—the enzyme that transforms alcohol to acetaldehyde—in the gastrointestinal mucosa. Gastric ADH seems to be less effective in oxidizing ethanol in women (than in men) and in alcoholics (than in moderate drinkers). When a moderate dose of ethanol was ingested on an empty stomach, first-pass metabolism was negligible. This was explained by the ethanol bypassing gastric ADH, owing to rapid absorption occurring. However, the quantitative significance of gut metabolism in the overall disposal of ethanol remains controversial.

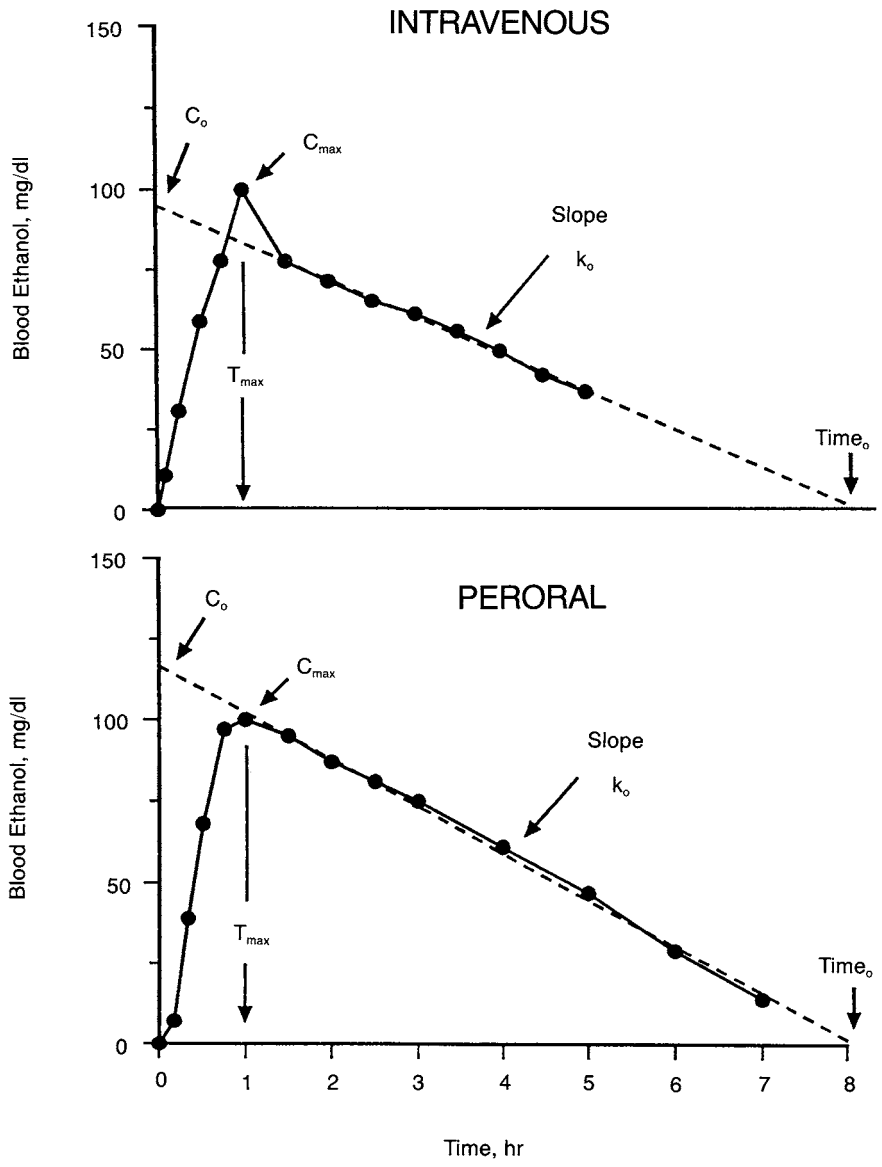

Figure 2
Examples of Concentration-Time Profiles of Alcohol Taken by Intravenous and Oral Routes of Administration. Examples of concentration-time profiles of ethanol obtained after intravenous infusion of 0.4 g ethanol/kg body weight in 15 minutes (upper part) and after ingestion of 0.8 g/kg (lower part). Several key pharmacokinetic parameters are shown.

ELIMINATION RATES AND ENZYMES

Differences in the rate of disappearance of ethanol from blood might depend on genetic and environmental factors influencing an individual's catalytic activity of alcohol-metabolizing enzymes. In humans, the enzyme ADH occurs in multiple molecular forms, designated class I, II, and III. Class I enzymes are located mainly in the liver cytosol and have a low k_m for ethanol. Various isozymes (variations within a class) exist and β_1-ADH (class I) is predominant in Caucasians whereas β_2-ADH (class II) is the most abundant isozyme in Asians. The rate of ethanol elimination in the various racial groups is not much different from the variations seen within a single racial group in well-designed studies that allow for racial differences in body composition—the proportion of fat to lean body mass.

Alcoholics have a greater capacity to eliminate ethanol than do moderate drinkers. Disappearance rates from blood of 30 mg/dl/h are not uncommon—compared with a mean rate of only 15 mg/dl/h (range 8–20 mg/dl/h) in moderate drinkers. The liver microsomes contain enzymes capable of oxidizing ethanol as well as other drugs, organic

solvents, and environmental chemicals. One particular form of the cytochrome P_{450} enzyme (denoted P450IIEI) metabolizes ethanol. This microsomal ethanol oxidizing system (MEOS) has a k_m of 40–60 mg/dl (8.7–13 mmol/l) compared with 2–5 mg/dl (0.4–1 mmol/l) for human ADH. More importantly, the P450IIEI isozyme becomes more active during prolonged exposure to ethanol—a process known as enzyme induction. Accordingly, because of continuous heavy drinking, alcoholics develop a high capacity for eliminating ethanol from the blood. Their enhanced capacity vanishes after a short period of abstinence, however, but liver disease (hepatitis, cirrhosis) in alcoholics does not seem to impair their ability to dispose of ethanol.

BEHAVIORAL EFFECTS OF ALCOHOL

Studies have shown that the behavioral effects of ethanol and its associated impairment of performance are more pronounced when the BAC is rising than when it is falling. This observation seems to depend, at least in part, on the distribution of ethanol between blood and tissue. The arterial blood concentration of ethanol is pumped to the brain and this exceeds the concentration measured in the venous blood, which is returning to the heart from skeletal muscles. This arterio-venous difference is most pronounced shortly after drinking; it decreases as ethanol diffuses equally into all body fluids. It seems that this is not the whole story, because some evidence points to the development of acute cellular tolerance to ethanol's effects—an aspect of tolerance that quickly develops.

Despite extensive studies of ethanol pharmacokinetics spanning many years, there are still a number of unsettled issues and areas of debate. Two such issues are (1) the practical advantages of Michaelis-Menten kinetics as opposed to Widmark's zero-order model and (2) the role of gastric ADH in presystemic disposal of ethanol. The importance of blood source (artery, capillary, or vein) and the sampling site (arm or leg) on ethanol pharmacokinetics deserves further study, as does whether multicompartmental models should be invoked.

(SEE ALSO: *Accidents and Injuries from Alcohol; Addiction: Concepts and Definitions; Alcohol; Chinese Americans, Alcohol and Drug Use among; Drug Interactions and Alcohol; Drug Metabolism; Drunk Driving; Psychomotor Effects of Alcohol and Drugs; Vulnerability As Cause of Substance Abuse*)

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PHARMACOLOGY In its broadest sense, pharmacology can be defined as the science dealing with interactions between living systems and molecules—in particular, chemicals (i.e., drugs)—usually introduced from outside the system. This definition also includes medical pharmacology, which is the science of drugs used to prevent, diagnose, and treat disease. Also included are the important roles played by chemicals in the environment that can cause disease, as well as the use of certain chemicals as molecular probes for the study of normal biochemistry and physiology. Toxicology is the branch of pharmacology that deals with the undesirable (i.e., toxic) effects of chemicals in biological systems.

(SEE ALSO: *Drug; Drug Metabolism; Drug Types; Pharmacodynamics; Pharmacokinetics; Poison*)

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PHARMACOTHERAPY *See* Treatment; Treatment Types

PHENCYCLIDINE (PCP) Although phencyclidine (PCP) and drugs of similar chemical structure (arylcyclohexylamines) are often called HALLUCINOGENS, they rarely produce HALLUCINATIONS, and the sensory distortions or apparent hallucinations that are produced are not the same type as LSD-induced hallucinations. Instead, phencyclidine belongs to a unique class of drugs called the dissociative anesthetics. Phencyclidine was developed in the 1950s as an anesthetic for veterinary medicine and later was tested in human surgical patients. There was great potential for PCP as an anesthetic because it produced minimal effects on the heart and breathing was not suppressed. Unfortunately, the adverse side effects of PCP (e.g., dysphoria [unhappy, ill] and psychotic symptoms) led to a termination of the human clinical trials. The drug is no longer manufactured for veterinary use because supplies were diverted (stolen) and the drug became widely abused in the 1970s. Ketamine, a drug chemically similar to PCP, is now used as a veterinary anesthetic and, in special cases, for anesthesia in humans. This drug is less powerful and shorter acting than PCP.

Phencyclidine abuse, mainly in pill form, peaked in the late 1970s and markedly declined throughout the 1980s and early 1990s. The most common route of administration in use in the 1990s was smoking. Phencyclidine is often added to MARIJUANA cigarettes, and it is commonly used while people are also drinking alcoholic beverages. Street names for PCP are "angel dust" or "crystal"; it is called "space base" when combined with COCAINE.

MECHANISM OF ACTION

Most investigators agree that the behavioral effects of PCP are mediated predominantly through RECEPTORS, which are proteins that are important for the normal functioning of cells within the body. Phencyclidine acts as an antagonist at the N-methyl-D-aspartate (NMDA) receptor-channel complex, which is one type of excitatory amino-acid receptor that is selectively activated by the agonists NMDA and GLUTAMATE. By definition, agonists produce stimulation while antagonists block the effects of agonists. When either glutamate or NMDA bind to the receptor, a channel within the cell membrane opens to allow sodium, calcium, and potassium ions to flow into and out of the cell. This movement of ions across the cell membrane causes a depolarization of the membrane which, if sufficiently large, causes the cell to fire. When the cell fires, an electrical charge passes along its membrane and NEUROTRANSMITTERS (chemicals that allow cells to communicate with each other) are released. Thus, glutamate and NMDA are important for normal cell-to-cell communication within the body.

PCP, as well as TCP, ketamine, dizocilpine (MK-801), and SKF 10,047 is representative of compounds that act as noncompetitive antagonists at the NMDA-receptor complex. The binding site for PCP resides within the channel and binding to this site physically prevents calcium and sodium ions from entering the cell while at the same time preventing potassium ions from leaving the cell. Blocking the movement of ions through the cell membrane in turn prevents the neuron from firing. In contrast to the noncompetitive antagonists, competitive antagonists such as CGS 19755, NPC 12626, CPP, and AP5 bind to the NMDA receptor itself without causing the ion channel to open. By simply occupying the receptor without activating it, competitive antagonists prevent NMDA from binding to and activating the receptor. Unlike noncompetitive antagonists, competitive NMDA-antagonist effects can be surmounted by higher doses of the agonist. However, the end result of both noncompetitive and competitive antagonists is a reduction of neuronal firing.

PHARMACOKINETICS AND METABOLISM

PCP use in humans occurs through several routes of administration, including intranasal (snorted), intravenous, oral, and inhalation (smoked). When PCP is smoked in parsley cigarettes, approximately 70 percent of the total amount of PCP is inhaled. Of this amount, 38 percent is inhaled as PCP and 30 percent is inhaled as phenylcyclohexene, a by-product of PCP created when it is heated. Peak blood concentration of PCP occur after only five to ten minutes, which is occasionally followed by a second peak one to three hours later. PCP is predominantly excreted in urine after intranasal, intravenous, and oral administration. The rate of PCP elimination through the kidneys depends on both urine pH and urine-flow rate. More specifically, PCP elimination occurs more

rapidly when urine is acidic and when urine is passed rapidly.

DISCRIMINATIVE STIMULUS EFFECTS

One useful method of evaluating the pharmacological characteristics of PCP, as well as a variety of other drugs, is the drug-discrimination procedure. Typically, animals that are slightly food restricted are trained to respond for food on one lever after drug administration and on another lever after saline. On days when the drug is administered before the session, responding on the drug-associated lever results in food delivery while responding on the saline-associated lever does not. Conversely, on days when saline is administered before the session, responding on the saline-associated lever results in food delivery while responding on the drug-associated lever does not. After a number of training days, animals learn to reliably respond on the drug lever after the drug injection and on the saline lever after saline injection. Once this discrimination has been established, a number of test drugs can be administered to determine whether or not they produce effects similar to the training drug. Test drugs that substitute for the training drug (i.e., cause responses on the drug-associated lever) are assumed to have discriminative stimulus effects that are similar to the training drug.

Using this procedure, several investigators have shown that PCP and other noncompetitive antagonists produce similar discriminative stimulus effects in a number of different species (see Willetts, Balster, & Leander, 1990 for a review). These results suggest that the mechanisms of action of PCP and other noncompetitive antagonists, such as ketamine and dizocilpine, are similar. Furthermore, the discriminative stimulus effects of competitive antagonists such as CGS 19755, NPC 12626 and CPP were also similar to each other, which is again consistent with the notion that the mechanisms of action of competitive antagonists are similar. Given that competitive and noncompetitive antagonists both reduce neuronal firing, it was of interest to compare the discriminative stimulus effects of these two types of antagonists. In most species, the discriminative stimulus effects of competitive and noncompetitive antagonists are very different from each other.

Another difference between the competitive and noncompetitive antagonists lies in their abilities to antagonize the discriminative stimulus effects of NMDA. While both types of antagonist are effective in blocking the convulsant and lethal effects of NMDA, competitive antagonists in general are much more effective than noncompetitive antagonists in blocking the discriminative stimulus effects of NMDA. The noncompetitive antagonists partially antagonize NMDA but only at doses that produced substantial behavioral suppression. While most effects of NMDA are antagonized by both competitive and noncompetitive antagonists, the behavioral-suppressing effects of noncompetitive antagonists often interfere with their ability to antagonize the discriminative stimulus effects of NMDA.

Finally, another important finding with competitive and noncompetitive antagonists involve their interaction with other receptor systems. Studies show that the discriminative stimulus effects of competitive antagonists such as CPP and NPA 12626 are similar to those produced by the BARBITURATE pentobarbital. Under certain conditions, the discriminative stimulus effects of PCP and pentobarbital were also similar. In addition to the interactions of NMDA antagonists with barbiturate receptors, some investigators have found similarities between PCP and ethanol (alcohol). These studies have proven to be important in describing both the similarities and differences between the noncompetitive and competitive NMDA-receptor antagonists.

TOLERANCE

Tolerance to a drug occurs when increasingly higher doses are needed to produce a specific effect or if drug effects diminish after repeated administration of the same dose of drug. It has not been possible to study tolerance to PCP in human subjects, but when interviewed, PCP users report that they increase the amount of PCP that they take over time (Carroll, 1990). Another indicator of tolerance development is that burn patients treated with ketamine for pain often require higher doses over time. It is easier to study tolerance to ketamine, PCP, and similar drugs in animals. Laboratory studies with rats have shown that tolerance developed to the effects of PCP on food-reinforced responding, to the effects of PCP and dizocilpine on steroid hormone (adrenocorticotropin and corticosterone) release, and to the cataleptic effects of ke-

tamine. Supersensitivity, the opposite of tolerance, occurs when repeated drug exposure produces a greater effect at a given dose. Some investigators have found that tolerance develops to some effects of PCP, such as head weaving, turning, and back pedaling, while supersensitivity occurs with other behaviors, such as sniffing, rearing, and ambulation. Although some scientists have hypothesized that PCP tolerance and supersensitivity are mediated through non-NMDA-receptor systems, others have suggested that PCP tolerance may be mediated through the NMDA receptor system. Repeated administration of dizocilpine, a PCP-like compound, produced a reduction in the number of NMDA receptors in the rat brain, and that was correlated with tolerance to some of the behavioral effects produced by dizocilpine. Further studies will clarify the role of different receptor systems in the development of tolerance to the effects of PCP and related compounds.

Studies indicate that there are interactions between PCP and other drugs with respect to tolerance and supersensitivity of drug effects. For example, dizocilpine blocked the development of tolerance to morphine's analgesic (painkilling) effects, but it did not alter the analgesic effects when MORPHINE was administered acutely. Also, dizocilpine attenuated the development of tolerance to ethanol (ALCOHOL), and it inhibited sensitization to amphetamine and cocaine (*DHHS Fourth Triennial Report to Congress on Drug Abuse and Drug Abuse Research,* 1992).

DEPENDENCE

Physiological dependence on a drug is usually defined by a set of withdrawal symptoms that occur when steady use of the drug is discontinued. The withdrawal symptoms are typically the same for a given drug, and they follow a specific time course which ranges from about six to forty-eight hours, depending on the drug. The withdrawal symptoms may be rapidly reversed after one administration of the drug.

Most of what is known about PCP dependence is from experimental studies with animals. There are only limited reports of PCP withdrawal effects in humans. In 1981, Tennant et al. studied sixty-eight regular PCP users; they found that one-third of them had sought treatment or medication to relieve the effects of PCP withdrawal. Withdrawal symptoms that they commonly reported were depression, drug craving, increased appetite, and increased need for sleep. Another way PCP dependence has been documented in humans is in studies of babies born to PCP-using mothers. Withdrawal signs that have been noted are diarrhea, poor feeding, irritability, jerky movements, high-pitched cry, and inability to follow a stimulus visually.

In laboratory studies with monkeys, similar signs of PCP withdrawal have been noted. Balster and Woolverton (1980) gave rhesus monkeys continuous access to PCP directly into the blood stream for fifty days, using an intravenous cannula system. The monkeys were trained to respond on a lever for an infusion of PCP. When PCP was replaced with a salt and water solution used to dissolve the drug (vehicle), withdrawal signs were noted, such as poor feeding, weight loss, irritability, bruxism (coughing), vocalizations, piloerection (hair standing up), tremors, less exploratory behavior in the cage, and poor motor coordination. The withdrawal syndrome began within four to eight hours, peaked between twelve and sixteen hours, and had disappeared by twenty-four to forty-eight hours. These results have been repeated in studies with rats. Some studies have reported PCP withdrawal effects after as little as two weeks of exposure. Thus, long-term use of the drug may not be necessary to produce physical dependence.

Recent studies with animals have shown that not only a short period of exposure to PCP but low doses of PCP result in withdrawal effects when drug administration is discontinued. Operant conditioning experiments are used as sensitive tests of drug-withdrawal effects in animals. In these experiments, animals are trained to respond on a lever or push a button or other device to obtain a food reward. At the same time they are allowed to self-administer drugs orally or intravenously. When drug access is removed, a decrease in operant responding for food is often seen, even when the drug dose is sufficiently low to produce no observable signs of withdrawal. These measures have also been used to demonstrate withdrawal effects from drugs such as cocaine, caffeine, and nicotine. When regular use of these drugs is discontinued there are no observable signs of withdrawal during abstinence. The most severe reductions in the operant behavioral baselines occur during the first forty-eight hours of drug withdrawal, a time during which

physical signs occur when higher maintenance doses are used; however, the behavioral disruptions often last for long periods of time. During withdrawal, when animals will not respond on a lever for food, they readily consume hand-fed food. Thus, the decrease in feeding may not be due to illness but to a decrease in the motivation to work for food.

In the first study that demonstrated disruption in operant behavior during PCP withdrawal, Slifer and coworkers (1984) treated monkeys with continuous intravenous infusions for ten days. They were required to make 100 responses on a lever for each food pellet. When access to PCP was terminated, responding for food decreased substantially for up to seven days and did not return to normal levels until the monkeys were again allowed access to PCP. Similar results were found by other investigators using monkeys trained to self-administer orally delivered PCP. There was little difference in the results, depending on whether the PCP was self-administered or experimenter administered. In the monkey studies, there was only a weak relationship between dose and the severity of the withdrawal effect, but in rats, PCP dose, blood levels, and magnitude of the withdrawal effect were closely related. Recent studies have shown that there is cross-dependence between drugs that are chemically similar to PCP—such as PCP and ketamine, dizocilpine, and the (+)isomer of SKF-10,047; however, cross-dependence was not demonstrated with either the racemate or (-)isomer of SKF-10,047 or with ethanol.

The PCP-withdrawal effect can be altered by changing schedules of reinforcement. In one study with monkeys, lever-press requirements or fixed ratios (FRs) for food were increased from 64 to 128 to 256 to 512 to 1024, and PCP-withdrawal effects were examined at each value. As the FR value increased, PCP withdrawal effects became more pronounced. At the two higher FRs, body weights declined and the severity of the withdrawal effect showed no further increases. To examine the effects of amount of food available, another experiment was conducted in which the FR was held constant at 1024 and the monkeys were either supplemented with 100 grams of hand-fed food or not. The amount of responding for earned food remained the same during supplemented and unsupplemented conditions, but when the effects of withdrawal were examined, a disruption in responding occurred only under the supplemented condition. When the monkeys had to earn their entire daily food ration, the withdrawal effect disappeared. These studies suggest that the severity of the PCP withdrawal effect is determined by the behavioral economics of food availability. The magnitude of PCP withdrawal increased as the price (FR of food) increased; but as the price became so high that body weight was lost, the PCP-withdrawal effect entirely disappeared. These data also suggest that PCP withdrawal is not necessarily an illness but a decreased level of motivation.

The use of drugs to treat the PCP-withdrawal syndrome has produced mixed results. When monkeys had access to orally delivered (+)SKF-10,047, the PCP-withdrawal-induced disruptions in food-maintained responding were reversed. This was not the case with (-)SKF-10,047 or the racemate (±)SKF-10,047. Injections of dizocilpine before PCP withdrawal, or two days into PCP withdrawal, greatly reduced or reversed, respectively, the disruptions in food-reinforced responding. Dizocilpine also dose-dependently reduced PCP self-administration. In contrast, while BUPRENORPHINE, a partial AGONIST at the mu-opiate receptor, also dose-dependently reduced PCP self-administration, it had no effect on PCP-withdrawal-induced disruptions in food-maintained responding. When PCP was self-administered concurrently with ethyl alcohol (ethanol) and then PCP access was removed, PCP-withdrawal effects were as severe as when ethanol had not been available. Thus, ethanol did not alleviate the PCP withdrawal effect, although, as noted earlier, PCP and ethanol share discriminative stimulus effects (Grant et al., 1991). In other studies, PCP was self-administered concurrently with ethanol or caffeine. When PCP and the other drug were removed simultaneously, the withdrawal disruption was more severe than when PCP alone was withdrawn. (Further details of these withdrawal studies may be found in reviews by Carroll [1990] and by Carroll and Comer in the *DHHS Fourth Triennial Report to Congress on Drug Abuse and Drug Abuse Research*, 1992.)

REINFORCING EFFECTS

The reinforcing effects of a drug are determined by demonstrating that self-administration of the drug plus the solution it is dissolved in (vehicle)

occurs in excess of self-administration of the vehicle alone. When drug-reinforced behavior is readily achieved in the animal laboratory, it is usually a good predictor that the drug has considerable abuse liability in the human population. The reinforcing effects of PCP have been studied using two animal models of self-administration, oral and intravenous. The intravenous route of self-administration requires the animal to make a specified number of responses on a lever or other manipulandum within a predefined time—then a fixed dose of the drug is delivered by an infusion pump via a catheter that is surgically implanted in a large vein that leads to the heart. Studies from various laboratories have demonstrated that intravenously delivered PCP functions as a reinforcer for rats, dogs, monkeys, and baboons.

Drugs that are chemically similar to PCP are also self-administered intravenously. These include drugs that have similar receptor-binding sites in the brain, such as ketamine, (+)SKF-10,047, dexoxadrol, and cyclazocine; and phencyclidine-like drugs that function as noncompetitive antagonists at the NMDA receptor, such as dizocilpine. Phencyclidine and dizocilpine self-administration is more reliably obtained when the animal has a history of self-administration of a drug with similar pharmacological or discriminative-stimulus effects. It has also been found that drugs that share discriminative-stimulus effect with PCP, such as (+)SKF-10,047, ketamine, PCE, TCP, and ethanol, are readily substituted for PCP in self-administration studies.

Oral PCP self-administration is established by presenting gradually increasing concentrations of PCP after the animal is given its daily food ration. After sufficient quantities of PCP are consumed, food is given after the drug self-administration session, and PCP consumption usually persists. This procedure provides a long-term stable baseline to examine variables that affect PCP-reinforced behavior. For example, alternative nondrug reinforcers, such as saccharin, reduce PCP-reinforced responding up to 90 percent of baseline if the FR for PCP is high or if the PCP concentration is very low. Free access to food decreases PCP self-administration, while even small reductions in the daily food allotment markedly increase PCP self-administration. Concurrent availability of ethanol also reduces PCP-reinforced responding.

A limited amount of information is available concerning drug pretreatment and PCP self-administration. Buprenorphine and dizocilpine pretreatment both resulted in dose-dependent decreases in PCP self-administration; however, potential treatment drugs such as fluoxetine and carbamazepine had no effect. Treatment with other drugs such as AMPHETAMINE or PENTOBARBITAL had a biphasic effect on PCP self-administration. Low doses of the pretreatment drugs increased PCP self-administration, and high doses decreased PCP self-administration.

TOXICITY

There is little evidence that long-term PCP use in adult humans (Luisada, 1981) and monkeys (see *DHHS Fourth Triennial Report to Congress on Drug Abuse and Drug Abuse Research*, 1992) results in any detectable organ or cellular damage. In monkeys that had been self-administrating PCP for eight years, tests of all organ systems, clinical chemistries, physical exams, and X rays revealed no differences between PCP-experienced and control animals that were the same age but had little drug experience. In humans, the form of toxicity most commonly associated with PCP use is a change in behavior. There have been a few accounts of bizarre and/or violent behavior associated with PCP use. Such reports have diminished since the preferred route of self-administration has shifted from oral (pill) to inhalation, which offers the users an ability to more carefully control the dose.

In monkeys, PCP produces a calming, tranquilizing effect. The immediate effects in humans are not seen in the hospital or clinic. Instead, the PCP user arrives in the emergency room several hours after PCP use, possibly while suffering acute withdrawal effects. Approximately twelve to fifteen hours after PCP was last taken, monkeys become agitated, violent, and aggressive. It is possible that many of the early reports of human violence and the PCP-related homicides were related to the withdrawal effects. It is necessary to determine the time course of unusual behavior and important to know the time of drug intake, although this is difficult to establish because the patient often loses memory of the drug-taking event.

Another unusual aspect of PCP toxicity is that users often complain of unpleasant effects long af-

ter chronic use has stopped. These reports could be caused by the fact that PCP is highly fat soluble and becomes stored for long periods of time in the body fat. During periods of weight loss, there is subsequent mobilization of fat-stored PCP into blood and brain tissues. Recent laboratory research with rats supports this hypothesis by demonstrating the ability of food deprivation to increase PCP levels in blood and brain (Coveney & Sparber, 1990).

Increasing data has become available on the effect of drugs of abuse on the offspring of dependent mothers, and it appears that the offspring of PCP users may be more vulnerable to the adverse effects of PCP than their adult counterparts. Golden and coworkers (1987) studied ninety-four PCP-exposed newborns and ninety-four nonexposed as controls; they found neurological abnormalities such as abnormal muscle tone and depressed reflexes in the exposed group. Another study followed twelve exposed infants for eighteen months and found a high percentage of medical problems (Howard et al., 1986). At six months the infants were irritable and hyperresponsive, and later they showed varying degrees of abnormalities in fine-motor, adaptive, language and social skills. A recent study of the offspring of forty-seven PCP abusers and thirty-eight nonusers found that neurological dysfunction was common in the infants of PCP-abusing mothers (Howard, Beckwith, & Rodning, 1990). There was greater apathy, irritability, jitters, and abnormal muscle tone and reflexes. Follow-up interviews at six and fifteen months, using the Gesell Developmental Exam, revealed poor language development and a lower developmental quotient in general; however, the long-term outcome for PCP-exposed newborns is unknown.

TREATMENT

There are currently no PCP ANTAGONISTS that are useful for treatment of PCP OVERDOSE. Symptomatic treatment may be given for suppressed breathing rates, fever, high blood pressure, and increased salivation. Convulsions are treated with DIAZEPAM. Elimination of the drug may be enhanced by making the urine more acidic and/or pumping stomach contents. Attempts to minimize environmental stimuli have helped to control violent and self-destructive behavior. Psychiatric care may be needed for an extensive psychotic phase that may follow overdose (Jaffe, 1989).

(SEE ALSO: *Abuse Liability of Drugs: Testing in Animals; Addiction: Concepts and Definitions; Adjunctive Drug Taking; Aggression and Drugs; Fetus: Effect of Drugs on the; Phencyclidine (PCP): Adverse Effects; Research, Animal Model: Drug Discrimination Studies; Tolerance and Physical Dependence*)

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PHENCYCLIDINE (PCP): ADVERSE EFFECTS Widely known as PCP, PHENCYCLIDINE

is an important drug of abuse in the United States, even though its use has declined since the 1980s. PCP is difficult to classify pharmacologically and is considered separately from the hallucinogens. The drug has not been studied systematically in animals, although research done in 1973 and 1980 indicated that it produces dependence in monkeys. As of 1999, its effects on the human central nervous system are poorly understood. It produces a unique type of hallucinatory effect and is used both by smoking and ingestion. Persons under the influence of PCP experience mood changes, perceptual distortions, and feelings of dissociation from their surroundings. Since their judgment is impaired, they may take unnecessary risks. They may become unpredictable and violent. In certain individuals, PCP use, especially if repeated often, can result in the production of a mental disturbance referred to as PCP psychosis. It is not, however, known with certainty whether PCP itself, or a combination of factors involved in the lifestyle of PCP abusers, is the cause of brain damage or of long-term behavior impairment that also sometimes occurs in PCP abusers.

HISTORY

Phencyclidine was developed in the 1950s for use as an anesthetic, but its use was discontinued because patients developed delusions, severe anxiety, or frank psychosis after their operation. It was also used by veterinarians as an anesthetic for some years; at present, however, all PCP sold on the street is manufactured illegally. The initials "PCP" are derived from a nickname, "The Peace Pill." The history of PCP as a drug of abuse began in the United States in the mid-1960s, when it was primarily taken by ingestion; but the real epidemic of PCP abuse occurred in the 1970s, when smoking and insufflation ("snorting") became the more common forms of use (Burns & Lerner, 1976). Because it is not difficult for an experienced chemist to synthesize the drug, PCP and its abuse spread rapidly, peaking about 1978. After 1980, its prevalence declined—however, PCP abuse continues to occur precisely because the drug is relatively easy to make. National Institute on Drug Abuse surveys show that more Americans have experimented with PCP than with heroin, and the prevalence of recent use of PCP is about the same as with heroin, so it remains a significant public-health problem (National Institute on Drug Abuse, 1991). As of 1999, most PCP abusers either inject the drug or smoke it by sprinkling it on smoking material (mint leaves, parsley, marijuana, or tobacco).

PSYCHOLOGICAL EFFECTS OF PCP

The psychological effects of PCP abuse can be discussed under three headings: (1) the effects accompanying acute intoxication, (2) the personality disturbances that can sometimes develop in PCP abusers, especially when associated with chronic use, and (3) the possible neurobehavioral toxicity that might result from chronic use.

SIGNS AND SYMPTOMS OF PCP INTOXICATION

Low Dose. Dreamy carefree state, mood elevation, heightened or altered perception, impaired judgment, partial amnesia.

Intermediate Dose. Inebriation, motor incoordination, dissociation and depersonalization, confusion and disorientation, perceptual distortions and preoccupation with abnormal body sensations, diminished pain sensitivity, partial amnesia, and sometimes exaggerated mood swings and panic.

High Dose. Catatonia, "blank stare," drooling, nystagmus (eye-rolling), delirium and hallucinations, psychotic behavior, severe motor incoordination, total amnesia.

ACUTE PCP INTOXICATION

As with all drugs, the effects of PCP depend on the dose that is taken. The section above lists the typical effects of PCP at various doses. PCP abusers usually adjust their dosage to experience only the low-dose effects. High-dose effects are similar to a mild type of dissociative anesthesia.

Experienced drug abusers can readily distinguish the experience of PCP intoxication from that produced by other drugs such as MARIJUANA, MESCALINE, and LYSERGIC ACID DIETHYLAMIDE (LSD). Users typically report a feeling of dissociation from the environment and abnormal body sensations and body image. The perceptual distortions often cause things to appear far away or abnormal in size. Compared to LSD, the effects of PCP are not very PSYCHEDELIC.

The most dangerous effects of PCP intoxication arise from the impaired judgment and altered perceptions that occur. People can engage in risk-taking behavior and harm themselves or others. DRIVING, swimming, or other activities requiring coordination and good judgment become extremely dangerous. Someone on PCP may also engage in casual but high-risk sexual behaviors. PCP users experience profound mood swings—where what begins as a pleasant experience can turn into panic and terror—and their behavior is unpredictable. Sometimes these "bad trips" can lead to violent and uncharacteristic behaviors with disastrous results. In cases of high-dose intoxication, users can experience a toxic psychotic episode with DELIRIUM, profound HALLUCINATIONS, and paranoia. In cases of severe overdose, seizures, stroke, or kidney failure may lead to death (Burns & Lerner, 1976).

MEDICAL TREATMENT

As of 2000, there is no medication that can serve as an antagonist to the effects of PCP or that can speed up its excretion. PCP is easily soluble in fats, thus can remain in the central nervous system for long periods. A patient who has overdosed on PCP must be placed on life support. Patients with anxiety or seizures can be given diazepam (Valium). Patients with psychotic episodes are usually treated with haloperidol (Haldol). Chlorpromazine (Thorazine) should *not* be given to patients who have taken PCP, as it may produce hypotension. Patients with severe hypertension due to PCP should be given diazoxide (Proglycem). Gastric lavage has been used successfully to treat patients who have ingested PCP directly.

PCP intoxication is considered a psychiatric emergency. It is recommended that these patients be placed in a secure room under observation. The health professional should not attempt to "talk the patient down." Physical restraints or a sedative such as lorazepam (Ativan) may be needed if the patient becomes violent.

LONG-TERM USE

In persons who abuse PCP in large amounts over a long period, or in those who have psychological problems that make them especially vulnerable, a chronic psychosis may develop. This PCP psychosis is evident even when abusers are not high on PCP, and it may be quite difficult to treat. The symptoms of PCP psychosis differ considerably from person to person, but patients may show many features of SCHIZOPHRENIA, including the appearance of a thought disorder, paranoid ideation, hallucinations, mood changes, and aberrant behavior. These patients often require psychiatric hospitalization and treatment with ANTIPSYCHOTIC medications.

In research studies where PCP has been given repeatedly to animals, it has been possible to show the development of PHYSICAL DEPENDENCE (e.g., Balster & Woolverton, 1980). The doses required for dependence are quite high, so it may be that dependence in human PCP abusers is difficult to develop. There have been some clinical reports of withdrawal effects in heavy PCP abusers, but these do not appear to be present in most individuals needing treatment for PCP abuse. There are no specialized treatment methods for PCP abusers, and since many PCP abusers also abuse other drugs and/or alcohol, they are usually helped by the same counseling and psychotherapy programs that are used for other forms of drug abuse.

NEUROPSYCHOLOGICAL AFTEREFFECTS OF PCP ABUSE

It is not known for certain whether or not PCP causes brain damage or long-term neurological or behavioral impairment in chronic abusers. Although some PCP abusers develop neurobehavioral impairment, controlled experiments of the type that would need to be carried out to show that PCP alone was the cause of the problems have not been done. PCP abusers typically abuse many other drugs in addition to PCP, which may contribute to their problems, and they may have lifestyles and health habits that lead to neuropsychological dysfunction. For example, while under the influence of PCP, they may be involved in an accident resulting in brain injury, so the risk factors that accompany PCP abuse may be responsible for the clinical problems sometimes seen in abusers. It should be pointed out that PCP was used in humans for medical research for a number of years, and ketamine—a close analog of PCP—is, even in the early 1990s, given to thousands of patients. No legacy of neuropsychological impairment is seen in these individuals.

Does this mean that chronic PCP abuse does not cause neuropsychological impairment? Certainly,

PCP—like all drugs—must be considered as a possible source of neural damage. In animal testing, it was found that even a single injection of a fairly high dose of PCP produced reversible pathomorphological changes in neurons of the cingulate and retrosplenial cortex in the brains of rats (Olney, Labruyere, & Price, 1989). Although it is not known if PCP produces these effects in humans, it is possible that it does and that this could lead to adverse health effects. Another possibly important basis for concern comes from studies which show that PCP, and related drugs, impair learning and memory in various animal models. PCP's ability to do this may be greater than for other classes of drugs of abuse, possibly due to PCP's ability to interfere with specific brain mechanisms for learning that involve N-methyl-D-aspartate (NMDA) RECEPTORS.

PCP AND VIOLENCE

Many people associate the abuse of PCP with violence and aggression, so this concern deserves special mention. Those under the influence of PCP often behave erratically and exercise poor judgment. These effects of PCP could certainly lead to violent behavior, and there are certainly numerous examples of extremely violent acts being performed by persons under the influence of PCP. This raises the question of whether PCP is uniquely associated with the production of violence and aggression: Is someone intoxicated with PCP more likely to be violent than someone who is intoxicated with COCAINE or alcohol?

Unfortunately, the answer to this question is not known. A great deal of criminal conduct in the United States is certainly carried out by people under the influence of alcohol or drugs. In addition, the public often associates drug use they do not understand with criminal and violent behavior. Every new drug epidemic is greeted with public concern that this drug causes violence. There is also the common practice of criminal attorneys using the defense of diminished capacity, because of drug use, to lessen the responsibility that their clients might bear for criminal conduct. All these factors undoubtedly contribute to the public attention focused on the relationship of PCP to violence.

Few good research studies have attempted to determine the specific role that PCP abuse may have in crime and violence. In one study (Wish, 1986) of nearly five thousand arrestees in New York City in 1984 who agreed to leave a urine specimen for drug analysis, it was found that 56 percent tested positive for at least one drug of abuse. For those who had used PCP recently, most had committed robbery, not bizarre violent offenses. In fact, assault was more common among arrestees who had not used PCP than among those who had. These results support the conclusion that PCP may be no more likely to cause violence than some other drugs of abuse—but, clearly, more research on this question is needed.

The NATIONAL INSTITUTE ON DRUG ABUSE estimates that as many as six million Americans have tried PCP at least once. The very large majority of these occasions of PCP use were not associated with violent acts; however, if some users prone to violence take PCP and are faced with a threatening situation, they may act unpredictably and violently. Although there is no scientific evidence that PCP actually increases muscular strength, PCP users unmindful of their own potential safety or injuries can be a formidable risk, so law enforcement personnel are on guard against these dangerous situations. Alternatively, it should not be assumed that most people who abuse PCP will become violent—nor should every inexplicable act of violence be casually or speculatively attributed to PCP abuse.

(SEE ALSO: *Addiction: Concepts and Definitions; Amphetamine Epidemics; Complications: Mental Disorders; Crime and Drugs; Tolerance and Physical Dependence*)

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PHENOBARBITAL This is the prototypic BARBITURATE central nervous system (CNS) DEPRESSANT. It is prescribed and sold as Luminal and was introduced into clinical medicine in 1912. It was used for a long period as a SEDATIVE-HYPNOTIC drug but has now largely been replaced by the much safer BENZODIAZEPINES.

Phenobarbital's long duration of action makes it useful for treating many forms of general and partial seizure disorders, such as epilepsy. Chronic use can result in TOLERANCE AND PHYSICAL DEPENDENCE, so it is classified as a Schedule III drug in the CONTROLLED SUBSTANCES ACT. Chronic treatment with phenobarbital can increase the activity of certain liver enzymes that metabolize other drugs. Thus a potential side effect is that other drugs (e.g., steroids, oral anticoagulants, digitoxin, beta-blockers, oral contraceptives, phenytoin, and others) are metabolized more quickly—and their

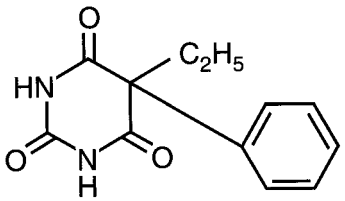

Figure 1
Phenobarbital

effectiveness is reduced. Combinations of phenobarbital and other CNS depressants, such as ALCOHOL (ethanol), can lead to severe motor impairment and reduced breathing.

(SEE ALSO: *Drug Metabolism; Drug Interactions and Alcohol*)

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SCOTT E. LUKAS

PHOENIX HOUSE *See* Treatment Programs/Centers/Organizations: An Historical Perspective

PHYSICAL DEPENDENCE A state, produced by repeated or prolonged drug exposure, in which the presence of drug in the body is required to maintain normal physiological function. This state is recognizable only by the occurrence of a withdrawal reaction when the drug is removed, that is reversed when the drug is again administered. Such dependence is believed to result from adaptive changes in the nervous system, opposite in direction to the drug effects, which offset these effects when drug is present, and produce a "drug-opposite" effect in its absence. Physical dependence is not synonymous with addiction, and can occur in nonaddicted persons.

(SEE ALSO: *Addiction: Concepts and Definitions: Disease Concept of Alcoholism and Drug Abuse; Tolerance and Physical Dependence*)

HAROLD KALANT

PILL POPPING *See* Slang and Jargon

PLANTS, DRUGS FROM Humans have used their local plants for medicinal effects since prehistoric times. They gathered and ate plants and noticed the effects that some offered—whether therapeutic, mind-altering, or toxic. From trial and error they fashioned associations between cause and effect, keeping certain mushrooms, roots, barks, leaves, or berries for certain situations—the treatment of accidents, ill health, childbirth, coughs, fevers, rashes, and so on. Over the centuries, they established herbal medicine, as it is now called; they had also found certain plants that produced immediate and mind-altering effects, many of which were relegated to religious ritual. By the nineteenth century, Europeans had developed the science of chemistry to the point where the activator in many plants could be isolated and concentrated.

If experimentation with plant materials has led to cures, such as quinine for malaria or digitalis for heart disease, it has also led to the discovery of unpleasant effects or the discovery of poisons. From the literally thousands of substances that have been self-administered over the centuries, only a few continued to be used for nonmedicinal purposes. Even fewer have given rise to serious problems of chronic use and dependence. The legal and readily available drugs that are found naturally in plants (e.g., NICOTINE, CAFFEINE) or are derived from plants (e.g., ALCOHOL) will be described here first, because the use and abuse of these drugs is more widespread than all the other abused drugs combined. The health problems associated with the chronic use of alcohol and TOBACCO are, therefore, a very serious problem in our society, not only because of the large number of people who suffer and die each year from the direct toxic effects of these drugs but also because of the costs—the absenteeism from work and the unnecessary health-care cost. The illegal drugs will be discussed next; although the illicit use of MARIJUANA, COCAINE, OPIOIDS, and PSYCHEDELICS remains a major social, legal, financial, and health problem in the United States today, the proportion of the population physically dependent on these drugs is actually relatively low—only a small fraction of a percent. Finally, it is important to note that people often do not restrict their drug use to a single type. Alcohol users typically smoke cigarettes and may sometimes use other drugs as well. HEROIN users may also smoke and consume alcohol, marijuana, coffee or COLAS, and, in some instances, various STIMULANTS. Multiple drug use is, therefore, a relatively common occurrence.

ALCOHOL

Alcohol is perhaps the most widespread drug in use. It forms naturally by the fermentation process of plant materials and has been produced on purpose since at least neolithic times, when grains were first farmed, harvested, stored, and processed into gruels, porridges, puddings, and so forth. Often these spoiled, forming a fermented base. Alcohol is made as well from other starchy or sugary plant materials, such as fruits, canes, roots, and such. Fermentation (also called anaerobic respiration, or glycolysis) is the chemical process by which living cells, such as yeast, use sugar in the absence of air to produce part or even all of their energy requirements. In fermentation, sugar molecules are converted to alcohol and lactic acid. BEER, wine, and cheese production, as well as certain modern commercial processes require the fermentation by specific kinds of yeast, bacteria, and molds.

Ethyl alcohol, also called ethanol, is the type of alcohol that is usually produced for human consumption. In its pure form, alcohol is a clear liquid with little odor. People drink it primarily in three kinds of beverages: (1) beers are made from grains through brewing and fermentation and normally contain from 3 to 8 percent alcohol; (2) wines are fermented from fruits, such as grapes, and naturally contain from 8 to 12 percent alcohol (up to 21% when fortified by adding more ethanol); (3) beverages or spirits DISTILLED from a fermented base, such as whiskey, gin, or vodka, contain about 40 to 50 percent alcohol, on average (often expressed in proof, so that 40% equals 80 proof; 50% is 100 proof).

NICOTINE AND TOBACCO

TOBACCO is a tall, herbaceous plant, the leaves of which are harvested, cured, and rolled into cigars, shredded for use in cigarettes and pipes, and processed for chewing or snuff. Tobacco has become a commercial crop in almost all tropical countries as

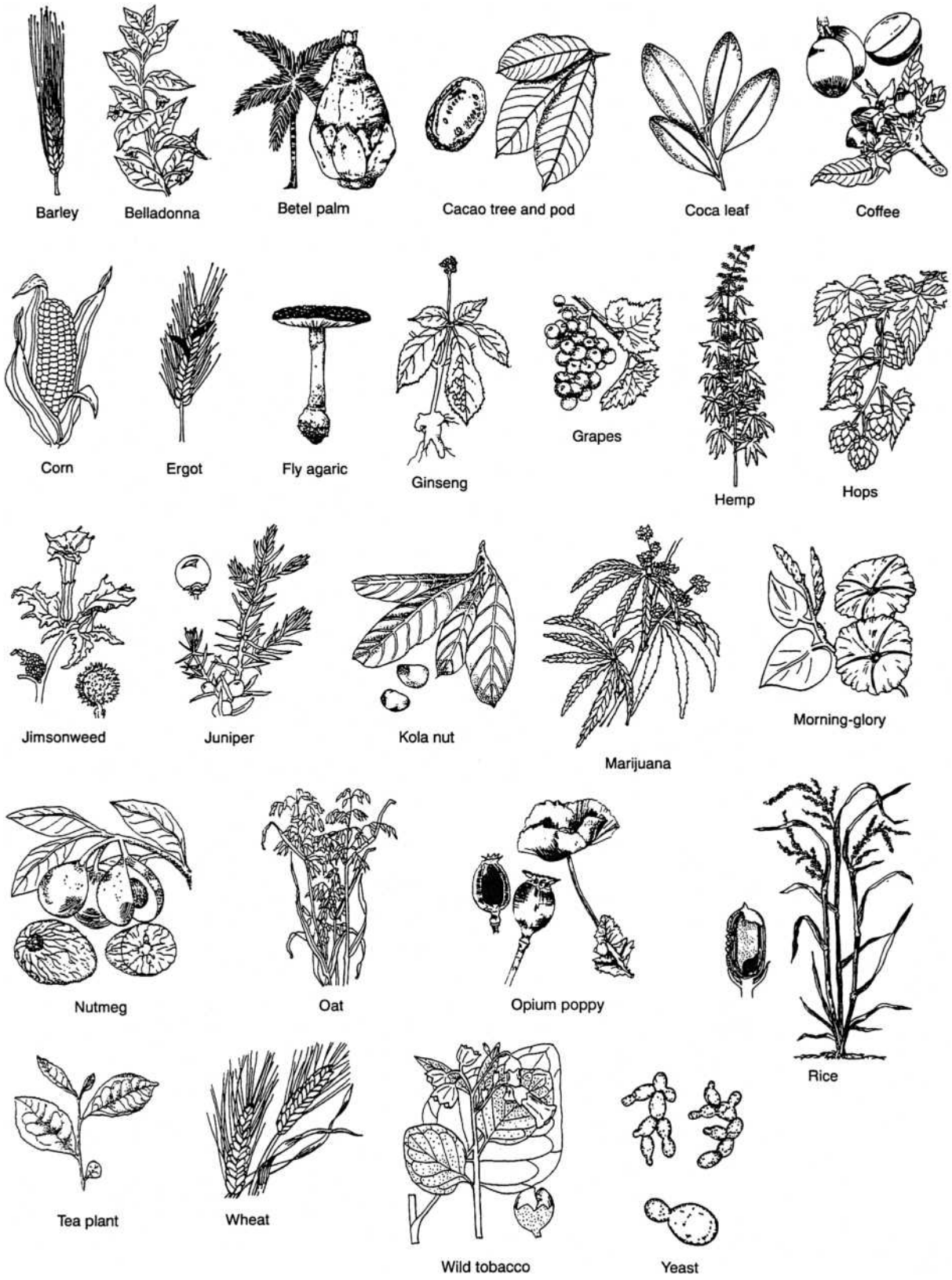

Figure 1
Some of the Plants Used in Making Drugs and Alcoholic Beverages.

well as in many temperate ones. The main source of commercial tobacco is *Nicotiana tabacum*, although *Nicotiana rustica* is also grown and is used in Asian tobaccos. Tobacco has been developed to yield a wide range of morphologically different types, from the small-leaved aromatic tobaccos to the large, broad-leaved cigar tobaccos. Tobacco is native to South America, where it was used in a drink for ritual purposes long before inhaling the smoke of the dried plant material was first documented by the Maya more than 2,000 years ago. Tobacco was then traded and grown in Central America; it moved into Mexico and the Caribbean and eventually into North America by about 800 A.D. The Arawaks of the Caribbean smoked tobacco, and during Columbus's voyage of 1492, he found the Arawaks smoking loosely rolled cigars. The Spanish took tobacco seeds to Europe, where Jean Nicot, France's ambassador to Portugal, sent tobacco to Paris in 1560 and gave the plant its genus (*Nicotiana*). In England, Sir Walter Raleigh began the popularization of pipe smoking in 1586, and the cultivation and consumption of tobacco spread with each voyage of discovery from Europe. Two kinds of tobacco were traded between Europe and America: "Spanish," from the West Indies and South America, and "Virginia," from the British plantations in their colony of Virginia. Despite its popularity in England, King James I forbade its production there since he vehemently disapproved of tobacco. Europeans at first smoked their tobacco in pipes, and later in cigars. It was often provided free to drinkers of coffee in coffee houses and cafés, as was the new product sugar. (Both remain strongly associated with coffee drinking.) Cigarettes spread in popularity only after the Crimean War (1854–1856), and their spread was especially aided by the first cigarette-making machine, developed in the United States in 1881.

NICOTINE is the most powerful ingredient of the tobacco plant, found primarily in the leaves. Nicotine is an extremely poisonous, colorless, oily alkaloid that turns brown upon exposure to the air. Nicotine can affect the central nervous system, resulting in respiratory failure and general paralysis. Nicotine can also be absorbed through the skin. Only two to three drops—less than 50 milligrams—of the pure alkaloid placed on the tongue can be rapidly fatal to an adult. A typical cigarette contains 15 to 20 milligrams of nicotine; however, the actual amount that reaches the bloodstream (and, therefore, the brain) through normal smoking is only about 1 milligram. Nicotine is responsible for most of the short-term as well as the long-term effects of smoking and plays a major role in the reinforcing properties.

CAFFEINE

CAFFEINE is an odorless, slightly bitter, alkaloid chemical found in coffee beans, tea leaves, and kola nuts, and several other plants used by humans such as cacao (CHOCOLATE) and maté (a South American holly used as a popular drink). In small amounts, caffeine acts as a mild stimulant and is harmless to most people. In large amounts, however, caffeine can result in insomnia, restlessness, and cardiac irregularities.

Tea. Tea is the beverage made when the processed leaves of the TEA plant are infused with boiling water. Native to Southeast Asia, the tea plant, *Camellia sinensis*, is a small, shrub-like evergreen tree that belongs to the family Theaceae. The seeds of the tea plant contain a volatile oil, and its leaves contain the chemicals caffeine and tannin. Although second to coffee in commercial value, tea ranks first as the most often consumed beverage. More than 50 percent of the world's population drink some form of tea every day. Many also use tea medicinally, as a stimulant. The tea plant originated in the region encompassing Tibet, western China, and northern India. According to ancient Chinese legend, the emperor Shen-Nung learned how to brew the beverage in 2737 B.C., when a few leaves from the plant accidentally fell into water he was boiling. Tea leaves began to be processed in China (dried, smoked, fermented, pressed, etc.) and were sold in cakes of steamed leaves, as powder, or in leaf form. Tea was introduced by Chinese Buddhist monks into Japan (9th to 13th centuries), where the preparation and consumption of tea developed into the ritual tea ceremony called *cha no yu*. Tea culture then spread into Java, the Dutch East Indies, and other tropical and subtropical areas. British merchants formed the East India Company (1600–1858) and introduced teas from China and India into England, the American colonies, and throughout the British Empire.

Coffee. The COFFEE bean is the world's most valuable legal agricultural commodity. In 1982, for example, the coffee-importing bill for the United States alone was 2.537 billion dollars. Of the many

varieties of the genus *Coffea* (family Rubiaceae) known to exist, only two species have significant commercial importance—*C. arabica* and *C. robusta* together constitute 99 percent of production. Coffee is native to the Ethiopian highlands and has been cultivated and brewed in Arab countries for centuries. The drink was introduced into Europe in the mid-seventeenth century and European colonial plantations were established in Indonesia, the West Indies, and Brazil, soon making coffee cultivation an important element in imperialist economies. Today, Latin America and Africa produce most of the world's coffee. The United States is the largest importer, having broken with the British tea tradition during the Revolutionary War to maintain the new American drink of coffee instead (purchased from non-British sources).

MARIJUANA

MARIJUANA is the common name given to any drug preparation derived from the hemp plant, CANNABIS SATIVA. Two varieties of this plant are *Cannabis sativa* variety *indica* and variety *americana*. The several forms of this drug are known by various names throughout the world, such as *kif* in Morocco, *dagga* in South America, and GANJA in India. HASHISH refers to a dried resinous substance that exudes from the flowering tops of the plant (also known as *charas* in Asia). In Western culture, cannabis preparations have acquired a variety of slang names, including grass, pot, tea, reefer, weed, and Mary Jane or MJ. Cannabis has been smoked, eaten in baked goods, and drunk in beverages. In Western cultures, marijuana is prepared most often from the dried leaves and flowering shoots of the plant as a tobaccolike mixture that is smoked in a pipe or rolled into a cigarette. As one of the oldest known drugs, cannabis was acknowledged as early as 2700 B.C. in a Chinese manuscript. Throughout the centuries, it has been used both medicinally and as an intoxicant. The major psychoactive component of this drug, however, was not known until the mid-1960s. This ingredient is TETRAHYDROCANNABINOL, commonly known as THC. PSYCHOACTIVE compounds (cannabinoids) are found in all parts of the male and female plant, with the greatest concentrations found in the flowering tops. The content of these compounds varies greatly from plant to plant, depending on genetic and environmental factors.

COCAINE

COCAINE is an ALKALOID drug found in the leaves of the coca plant, the common name of a shrub, *Erythroxylon coca*, of the coca family, Erythroxylaceae. Coca is densely leaved and grows to heights of 8 feet (2.5 m). It is cultivated in its native South America but also in Africa, Southeast Asia, and Australia for the narcotic alkaloids of its leaves, particularly cocaine. Whole or powdered dried leaves, usually mixed with lime (calcium carbonate), have been chewed by the people of what is now Colombia, BOLIVIA, and Peru for centuries, to dull the sense of hunger and to lessen fatigue. The coca shrub should not be confused with the cacao tree, the source of cocoa and chocolate.

Cocaine was first used in Western medicine as a local anesthetic. In 1884 it was used by Carl Koller, an ophthalmologic surgeon. Historically, the chief medical use for cocaine has been as a local anesthetic, especially for the nose, throat, and cornea, because of its effectiveness in depressing nerve endings. This has been largely replaced by less toxic, synthetic local anesthetics. Used systemically, cocaine stimulates the central nervous system, producing feelings of excitation, elation, well-being, enhanced physical strength and mental capacity, and a lessened sense of fatigue. It also results, however, in increases in heart rate, blood pressure, and temperature, and its use can result in death. Cocaine use became popular because of its stimulating properties. In Western countries, it is frequently ingested by sniffing its fine white powder, often called snow. It is sometimes injected intravenously, although repeated injections can result in skin abscesses, hepatitis, and the spread of AIDS. Cocaine can also be inhaled (smoked) once it has been converted to its free-base form; some preparations of freebase cocaine are known as rock, or crack. CRACK-cocaine gained popularity in the late 1980s and early 1990s, because it is relatively inexpensive as a single dose, (e.g., $10 to $20 per "hit"); usually smoked in a special pipe, it produces an extreme euphoria as it is rapidly absorbed from the lungs and carried by the blood directly to the brain.

OPIUM

OPIUM is a drug obtained from the juice of the immature seed pods of the oriental poppy, *Papaver somniferum*. There are over 20 natural alkaloids of

opium, including CODEINE and MORPHINE. Morphine is the largest component and it contributes most significantly to opium's physiological effects. HEROIN (diacetylmorphine) was derived from morphine and is the most important drug synthesized from opium's natural alkaloids. As a folk medicine, opium has been used to relieve pain, reduce such drives as hunger and thirst, induce sleep, and ease anxiety and depression. Opium and some of its derivatives are highly addictive, and their use has led to abuse and serious drug problems. Drugs from opium or derived from opium are still used widely in medicine, despite the development of synthetic opioid drugs such as MEPERIDINE (Demerol). The therapeutic effects of the opioids include PAIN relief, suppression of the cough reflex, slowing of respiration, and slowing of the action of the gastrointestinal tract. Opium's constipating effect led to its initial use, in the form of paregoric, in treating diarrheas and dysenteries. The main producers and exporters of opium are located in India and Turkey. About 750 tons (680 metric tons) of opium are annually needed to meet medical uses worldwide.

Opioids have been used since ancient times, both for medicinal purposes and for pleasure. Opium was taken orally, as a pill or added to beverages, for centuries in the Middle East, India, and Asia. Addiction did not become a wide problem until the practice of opium smoking was introduced by the British from India into China in the late seventeenth century (in an effort to gain a trade opening to the "closed" empire of China). China attempted to deal with the problem by restricting the cultivation and importation of opium in the nineteenth century. This led to the Opium Wars (1839–1842), since the opium trade became highly profitable to the British East India Company. Britain won over China, and opium was sold to the Chinese through treaty ports until the twentieth century.

In Europe and North America in the eighteenth century, opioids became widely used as most effective and reliable analgesics (painkillers). Heroin was developed in Germany in the 1890s and used from 1898 as a cough suppresser and analgesic with the hope that it would not lead to addiction, as did morphine (from which it was derived). From the first year or two after introduction, some clinicians agreed that it did not show addictive properties. A few even suggested that it might be useful in treating people addicted to morphine. Within a few years it became clear that, like morphine, its use could lead to addiction comparable in gravity to that of morphine.

On the street, opium is seen as a dark brown chunk of gum (from the pod of the opium poppy) or in dried powdered form. It is smoked, eaten, and drunk or injected as a solution for medicinal and recreational purposes. Indian and Chinese immigrants brought the practices with them, but the number of users is not great. During the early phases of addiction, opium produces a feeling of euphoria or well-being. With time, one may become dependent through physical and emotional factors. Tolerance develops and larger and larger doses of the drug are required to produce the same effect. If denied access to the drug, an addict will experience severe withdrawal symptoms; sudden withdrawal in a heavily dependent person has occasionally been fatal.

MESCALINE

PEYOTE, or mescal, is the common name of the small spineless cactus *Lophophora williamsii*, found in the southwestern United States and north-central Mexico. Peyote is used in Native American religious rituals, primarily for its HALLUCINOGENIC effects. At the end of the nineteenth century, Arthur Heffter demonstrated that MESCALINE (3,4,5-trimethoxyphenethylamine) was responsible for peyote's pharmacological effects. Mescaline is related to the AMPHETAMINES. When ingested, it can produce HALLUCINATIONS, frequently of a visual nature, characterized by vivid colors, designs, and a distorted space perception. It stimulates the autonomic nervous system and can cause nausea, vomiting, sweating, tachycardia (rapid heartbeat), pupillary dilation, and anxiety. The use of peyote in Native American ritual, referred to as Peyotism, was documented by Europeans in the sixteenth century. The modern practice of the peyote-based religion began in the late nineteenth century, was widely practiced by Native Americans in the southwestern United States, and was incorporated as the Native American Church in 1918. This church claimed more than 200,000 members in the 1960s. From the church member's point of view, peyote symbolizes spiritual power; the peyote "button"—the dried top of the cactus—is eaten as a sacrament to induce a hallucinogenic trance (of a few hours duration) for communion with God.

PSILOCYBIN

PSILOCYBIN is the active substance contained in the fruiting bodies of the *Psilocybe mexicana* mushroom (called the MAGIC MUSHROOM); it is a potent hallucinogen that can cause psychological disturbances. Taken orally or injected, the drug produces effects similar to those of the chemically unrelated LSD (LYSERGIC ACID DIETHYLAMIDE), and cross-tolerance has been experienced between psilocybin, LSD, and mescaline. The use of psilocybin is illegal in the United States, except for the direct consumption of mushrooms by a few religious groups as part of their ritual.

OTHER SUBSTANCES

Throughout the world, many other natural plant substances are used for mind- and mood-altering effects. These include the use of the KAVA root (*Piper methysticum*) for an intoxicating drink in the South Pacific; indole-containing snuff (distilled from indigo, genus *Indigofera*) among the Amazonian Indians of Brazil; KHAT leaves of a bush indigenous to East Africa containing an amphetamine-like drug (cathinone); BETEL NUT derived from the betel palm (*Areca catechu*) and widely used throughout the Pacific rim; and FLY AGARIC (a toxic mushroom, *Amanita muscaria*) among the Uralic-speaking tribes of Siberia.

(SEE ALSO: *Alcohol: History of Drinking; Asia, Drug Use in; Ginseng; Ibogaine; Jimsonweed; Morning Glory Seeds; Nutmeg; Opioids and Opioid Control: History; Tobacco: Industry*)

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POISON A substance that, when introduced into the body in relatively small quantities, causes destruction or malfunction of some tissues and organs. Depending on the quantity in the body (the dose), a poison can kill. The word poison usually implies that a substance has no healthful use and is to be considered dangerous even in small quantities. Most common household substances are poisonous, including bleach, ammonia, drain cleaners, paint supplies, and so on.

SUBSTANCES CAUSING DEATHS FROM ACCIDENTAL POISONING

- DRUGS
  - Analgesics and antipyretics
  - Sedatives and hypnotics
  - Tranquilizers
  - Antidepressants
  - Other psychotropic agents
  - Other drugs acting on nervous system
  - Antibiotics and other antimicrobial agents
  - Cardiovascular drugs
  - Hormones
  - Hematological agents
  - Other drugs
- OTHER SUBSTANCES
  - Alcohols
  - Cleaning and polishing agents and paint
  - Petroleum products
  - Pesticides
  - Corrosives and caustics
- GASES
  - Utility gas
  - Carbon monoxide
  - Nitrogen oxides
  - Freon
  - Other gases

In the practice of medicine, many useful DRUGS, such as antibiotics for treating infections or antihypertensive drugs for treating high blood pressure, can be poisonous or toxic in higher doses. Almost

all drugs that are abused can be poisonous or toxic; some, even at relatively low doses.

A few drugs that are commonly used in medicine in small amounts to produce important therapeutic effects are also used in other contexts as poisons. For example, the drug warfarin is used medically as an anticoagulant (to increase the time it takes blood to clot), an important effect for people who have had strokes or heart-valve replacement—but warfarin is also used as rat poison, because when rats eat it in large amounts they die soon after from massive hemorrhages. The same "mustard gas" (nitrogen mustards) that, as poison gas, caused much death and suffering in World War I, actually has medical use in the treatment of certain leukemias. Similarly, a series of extremely potent chemicals were developed during World War II as "nerve gases" for warfare, which act by flooding the body with excess acetylcholine (a body substance necessary for synaptic transmission), causing muscle paralysis and death. Consequently, close chemical relatives of some of the most potent nerve gases ever developed are being used to treat medical disorders, such as myasthenia gravis, in which there is not enough acetylcholine in nerve endings.

Treatment of someone who has been poisoned may require removal of the poison from the body (e.g., with the use of a stomach pump for ingested poisons), administration of an antidote if one exists, or simply support in repairing the damage done to the body. Many cities have a telephone "hot line" or poison-control center number where information about poisons, antidotes, and actions to take in case of poisoning can be obtained; often, they will alert emergency medical service (EMS) units to arrive in mere minutes. In case of a poisoning, including a drug overdose, it is essential to call for expert medical help as quickly as possible to minimize damage to the victim.

(SEE ALSO: *Complications: Medical and Behavioral Toxicity Overview; Drug Types; Inhalants; Methanol*)

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POISON CONTROL CENTERS *See* Appendix I, Volume 4

POLICY ALTERNATIVES This section includes two articles that introduce the reader to some of the issues surrounding public debate on the decriminalization of, or the legalization of, drugs. *Prohibition of Drugs: Pro and Con* is a short summary of the diverse opinions, expressed continually over the last 80 years, about the optimal way to deal with the reality that psychoactive drugs exist; that many people like the effects of those drugs; that some who use them do so to excess; that some are necessary for medical purposes; and that the substances themselves can be toxic not just for the user but for others who are affected by the user's behavior. The second article, *Safer Use of Drugs*, takes the view that society can reduce the toxic personal and social effects of drugs by informing potential users about how the risks of drug use can be minimized.

The argument that harm from drug use can be reduced by teaching people how to use drugs safely is viewed by many experts as counterproductive and likely to foster drug use. The *Partnership for a Drug-Free America* has developed its media campaign on the premise that the decision to try a drug is powerfully driven by two specific attitudes: perception of risk and social disapproval. This premise is supported by data emerging from the national *High School Senior Survey* (*Monitoring the Future* study) that the likelihood of drug use, especially initial experimentation, goes down as appreciation of the risks associated with drug use goes up. The more a young person feels that drugs are socially acceptable and/or not dangerous, the more she or he is likely to try them. It is difficult to imagine an educational process that can teach "ways of using drugs safely" without simultaneously communicating a message of tolerable risk and a degree of social acceptability.

Analysis of Drug Legalization Whether or not a drug should be prohibited or legalized is perhaps the most fundamental question in drug policy. It is a moderately complex question and most who write about the issue do so from an

advocacy perspective, so the debate is even more confusing than it needs to be.

It is important to start with a clear definition of what is meant by legalization vs. prohibition. There is a spectrum of policy positions. Some drugs can be used for medical but not recreational purposes (e.g., cocaine), whereas others cannot even be used for medical purposes (e.g., heroin). Some drugs cannot be used recreationally but are legal with a prescription (Valium) or when taken under medical supervision (methadone). Some drugs are legal only for adults (alcohol); others are legal for all ages (e.g., the caffeine in soda).

When a sharp line needs to be drawn between legalization and prohibition, it is useful to say that a drug is legal if it is legal for that substance to be produced and distributed for unsupervised consumption by a significant portion of the population (e.g., all adults). By this definition making marijuana available for medical use is not legalization if prescriptions are restricted to those experiencing specific, medically-diagnosed conditions (glaucoma), but it would be if any adult could write his or her own prescription. Likewise by this definition the Netherlands has legalized retail production, distribution, and use of marijuana, although wholesale (large-volume) marijuana production and distribution is still prohibited. Most other drugs in most countries are either clearly legal or clearly prohibited by this definition.

Having defined prohibition vs. legalization, the next important observation is that different people use different criteria for deciding what policy *should* be. Some people are implicitly if not explicitly consequentialists. They think the right policy is the policy that leads to the fewest problems. Others believe that there is a moral imperative to make substances legal (e.g. libertarians who believe people should be free to consume anything, even if it hurts them) or prohibited (e.g., people who believe the substance is evil for religious reasons) regardless of the consequences.

The challenge for the moral prohibitionists is defending to others why they favor prohibiting some drugs but not others. There are defensible positions predicated on consistent principles (''all intoxication is immoral'' or ''being physically dependent on a drug is idolatry''), but it is hard to articulate such a defense for US policy. Cigarettes are highly addictive, and alcohol is clearly an intoxicant, but they are both legal. In 1930 alcohol was prohibited, but marijuana was not. Ten years later, marijuana was prohibited but alcohol was not. One does not have to be very cynical to believe that the moral distinctions enshrined in public policy are just the legal formalization of arbitrary popular prejudices.

The challenge for the libertarian view is less simplistic but no less compelling (at least for those who recognize *homo economicus* as an ideal type, not a descriptively accurate model of human behavior). The basic idea is that at least some addictive, mind-altering substances may merit an exception to the general rule that a liberal society should not interfere in the private consumption decisions of its citizens. Mark Kleiman, a drug policy scholar and professor at UCLA, eloquently makes the case in his 1992 book *Against Excess*. The distinguishing characteristics are a combination of factors such as: drugs are intoxicating so consumption decisions are often made ''under the influence,'' for some drugs cessation is physically painful, drugs offer immediate pleasures and the possibility but not guarantee of delayed pain, drug initiation occurs primarily among minors, social influences play a prominent role in initiation decisions, etc. That skepticism of government regulations is healthy for a liberal democracy does not imply that prohibiting a drug is necessarily a bad idea. Liberal democracies tolerate other paternalistic infringements on freedom of behavior (such as a minimum wage, motorcycle helmet laws, and prohibitions against swimming where there are dangerous rip tides).

Furthermore, few want minors to have ready access to drugs, but legalizing use by adults inevitably makes a drug readily available to minors because every adult is a potential supplier, whether consciously (adults buying alcohol for minors) or unconsciously (minors stealing cigarettes from adults). Legalizers sometimes deny this, asserting that cocaine is more readily available to minors than alcohol is, but those assertions are contradicted by minors' self-reports (e.g., in the Monitoring the Future surveys).

The moral arguments for or against prohibition are in one sense unassailable. Every person is entitled to his or her values. But at the same time they are not very persuasive to people who do not hold those values.

For consequentialists, opinions about legalization tend to depend on how people trade off

or value the problems associated with drug use and those associated with prohibition and black markets and on predictions about how legalization would affect those outcomes. Prohibiting a drug will generally reduce but not eliminate its use. The use that persists despite prohibition supports a black market, which generates problems of its own. Indeed, the social cost per gram or per ounce consumed will typically be greater than would be the case if the drug were legally available. So prohibition typically reduces use but increases harm per unit of use.

Those who favor legalization tend to believe that a drug's legal status has little impact on its use. They also tend to be very mindful of the problems associated with black markets (stereotyped as drug dealers shooting people in battles over ''turf''), drug enforcement (e.g., racially biased enforcement tactics), and prohibition's increasing the damage per episode of use (e.g., restricting needle availability increasing spread of HIV by needle sharing). Those who favor prohibition tend to believe that prohibition substantially suppresses use (tobacco and alcohol are used far more than cocaine or heroin) and that many problems stem directly from drug use (e.g., the damage addiction can do to familial relations) not primarily from the drug's illegal status. To them, legalization is jumping out of the frying pan and into the fire. It might eliminate the black market and associated crime, but if legalization led to a ten-fold increase in the number of addicts the country could still be worse off.

Unfortunately, the public debate about the consequences of legalization is clouded with specious arguments. For example, prohibitionists argue that drugs should be illegal because they are associated with so much crime (something on the order of one-fourth of crime in the US). Legalizers counter that most of the drug-related crime is attributable to the prohibition. Only about one-sixth of drug related crime is ''psychopharmacological'' in nature (i.e., driven directly by intoxication or withdrawal). Conflicts between market participants turn violent in part because they cannot resort to the court system to resolve disputes, and one reason addicts commit robberies is to get money to buy drugs that would cost far less if they were legal. Ironically, alcohol is one of the most violence-promoting substances per se, and it is legal.

To give an example from the other side, legalizers cite statistics showing that illegal drugs such as cocaine and heroin kill only thousands of people per year, whereas alcohol and cigarettes kill hundreds of thousands. What they neglect to point out is that far more people use cigarettes and alcohol, so the death statistics per user are not so different. Furthermore, the death statistics for illicit drugs are restricted to acute effects (e.g., overdose deaths), whereas the cigarette and alcohol figures include indirect effects (e.g., deaths caused by intoxicated drivers) and delayed or chronic effects (e.g., from lung cancer). Focusing on overdose deaths would make cigarettes seem safe, whereas the expansive definition suggests that they kill more people than all other drugs combined, including alcohol.

Both sides lend a patina of scientific rigor to their arguments by citing trends in data, but the divergent trends of different indicators makes it easy to tell statistical lies. An advocate of prohibition might point out that the number of drug users fell dramatically during the 1980s when enforcement expanded rapidly. A legalizer could counter that emergency room mentions of drug use rose as fast as prevalence fell. What is lost in such bickering is the observation that the legal status of the major drugs has been stable in the US for many decades. Looking at contemporary trends might tell us about the wisdom of a more or less stringent prohibition, but we have no direct experience with legal cocaine, heroin, marijuana, or methamphetamines in recent US history. Many seek to draw lessons drawn from other times (e.g., when cocaine was legal in the US in the late 19th century) or places (e.g., Europe), but casual comparisons can be misleading and careful study of those analogies does not give definitive guidance (MacCoun and Reuter, forthcoming).

Even anecdotal evidence can be spun in different ways. Consider the periodic accounts of a mother selling her baby for crack. Some argue this proves drugs should be legalized. If they were cheap enough, addicts would not have to resort to such draconian measures. Others counter that the fundamental problem is that the drug is so powerful that it becomes more important to a mother than her own child, and we should do everything we can to protect people from the temptation to use things that so distort such societal pillars as the value of family.

The next important observation is that different drugs are different, and it may well make sense to prohibit some but not others because they have different properties (e.g., some drugs can trigger violent outbursts [PCP]; others tend to sedate [heroin]). It is by no means the case, however, that one can unambiguously rank order substances from the most to the least dangerous because a substance can be very threatening in one respect but not in others. Cigarettes are highly addictive, but they are not intoxicating. Heroin can be deadly (overdose deaths are not uncommon) but in and of itself creates almost no chronic health damage. Heroin addicts are usually in poor health because they are poor, spend money on heroin not food or shelter, and inject with dirty needles, but the heroin per does not degrade organs the way alcohol destroys the liver or smoking causes emphysema. The following table illustrates the concept.

The table divides the substances by legal status. The legalization question asks whether any substances on one side of the line should be moved to the other. It does not address changes in laws, programs, or policies that do not move a substance across the line. It might or might not be a good idea to repeal mandatory minimum sentences, cut the number of drug arrests in half, expand treatment and prevention programs, approve marijuana for medical use, eliminate profiling as an enforcement tactic, reduce the military's role in drug control, and repeal drug-related civil forfeiture statues. Doing so would blunt many of the criticisms of "prohibition," but it would not constitute legalization. Conversely, one could raise the "smoking age," require people to pass a "drinker's test" to get an alcohol consumption license, or ban smoking from all public spaces, but none of those would extend prohibition to a new substance.

There is no constituency for prohibiting caffeine, and prohibition of alcohol is perceived to have failed so badly in the last century that there is little stomach for trying it again. There is some discussion of banning tobacco use, but such proposals are probably political non-starters.

The more seriously debated proposals would legalize one or more of the currently prohibited substances. For discussion purposes, it is convenient to differentiate three groups of substances: (1) cocaine, heroin, and methamphetamines, (2) marijuana, and (3) all other illicit substances.

Cocaine, heroin, and methamphetamine are not all similar pharmacologically, but they have key commonalities. They (particularly cocaine and heroin) are expensive, are subject to stringent enforcement, can dominate the lives of an abuser, and have large, established black markets. These are the substances whose use can most confidently be predicted to rise substantially and to be problematic if they were legalized. These substances are very simple to produce, but sell for many times their weight in gold because they are prohibited and subject to severe sanctions. They are also the source of most of the corruption, violence, and disorder associated with drug markets, so legalization would bring

	Caffeine	*Tobacco*	*Alcohol*	*Marijuana*	*Heroin*	*Cocaine*
Acute health risk	None	None	High	Minimal	High	High
Chronic health risk	None	Huge	High	Some	Minimal	Some
Use affects health of others	No	Yes	Fetuses	Possibly	No	Fetuses
Problems caused by withdrawal	Minimal	Unpleasant	Physical risk	Minimal	Physical risk	Extremely unpleasant
Intoxication leads to accidents	No	No	Yes	Some	Moderate	Unclear
Intoxication leads to violence	No	No	Yes	No	No	Some
Likelihood of addiction given use [as observed in the US in last 30 years]	Minimal	High	Moderate	Moderate	High	High
Addiction disruptive to daily functioning	No	No	Yes	Somewhat	Yes	Yes

many benefits. Most observers, though, believe this would be an example of "out of the frying pan and into the fire." At a minimum, legalizing these substances is a high stakes gamble that is only partially reversible. There are other, safer alternatives to exhaust first (e.g., mending rather than ending prohibition) and more information that should be gathered about how legalization would affect use before seriously contemplating such a radical change.

Marijuana presents a quite different situation. Prohibition makes marijuana more expensive than it otherwise would be, but a daily habit is still only modestly more expensive than a two pack a day cigarette habit. Likewise, daily marijuana use is not a recipe for enhancing performance, but it does not preclude most daily functions (personal hygiene, holding down a job, etc.). So a ten-fold increase in use is a less likely outcome of legalizing marijuana than for cocaine, and even if it did happen, that outcome would be less catastrophic. On the other hand, the benefits of legalizing marijuana are far smaller than the benefits of legalizing cocaine, heroin, and methamphetamines because marijuana markets are less violent and marijuana users generally do not resort to crime to support their habit. There is no consensus about whether legalizing marijuana is wise. Some say yes. Most say no. What is clear though is that the risks, uncertainties, and potential benefits are all much smaller when considering legalizing marijuana than when considering legalizing cocaine, heroin, and methamphetamines.

The last category is diverse, so general statements are difficult. It includes drugs that can be used as a weapon in sexual assault (e.g., GHB) and drugs used not for their mind or mood altering properties but to enhance athletic performance (e.g., anabolic steroids). Two general observations are possible, however. First, prohibitions are relatively more effective and relatively less costly when preventing the spread of substances that are not commonly used than they are at reducing the use of an established drug. Second, by definition, there is more to lose in terms of increased availability and use when altering the status of drugs that are now rare. By those principles, it would be easier to make a case for legalizing XTC or LSD, for example, than for PCP, but they are not frequently the focus of legalization proposals, which typically address just marijuana or all drugs collectively.

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Prohibition of Drugs: Pro and Con The history of U.S. social and legal policy in regard to psychoactive and intoxicating drug use has been characterized by periodic shifts, strong ideological presuppositions, and deep disappointment. Any analysis of current policy and the debate about drug legalization must recognize the historical roots of current policy that affect the various positions in the debate.

A brief historical note may help place the current discussion of drug policy in the United States in perspective. To borrow a phrase from Ecclesiastes, there is nothing new under the sun. Those engaged in the current, often heated, discussions about national drug policy often act as if their concerns, insights, and positions about intoxication, drug use, and society are unique to our age. A cursory review of history indicates that the debate on the meaning and effects of alcohol and other drug use on morals, public safety, productivity, and health is at least as old as written language. Some of the earliest recorded civilizations struggled with the issue and often adopted laws and policies that attempted to strictly regulate or prohibit the use of alcohol and other drugs.

Often these laws were based on a culture's perspective on the will of the divine or combined with basic civil codes. For example, the Torah appears to be very concerned with excessive alcohol use. It was seen as leading to gross immorality. The Christian New Testament holds similar views particularly on the excess use of alcohol. The theme seems to be one of avoiding all things that harm the body or one's relationship with God and moderation even in all things that are good. The Koran takes a very strong prohibition stand against alcohol and all intoxicating substances. Since much of modern Western civilization derives from these religious traditions, they continue to influence public thinking and policy. From a less theocentric perspective, many ancient civil codes also struggled with the

regulation or prohibition of intoxicating chemicals. For example, the Romans seemed especially concerned that slaves and women not use alcohol and forbade its use by them. The concern appeared to be that alcohol would make slaves less productive and more difficult to control and that it would also lead to female sexual impurity. Chinese emperors prohibited the use of opium among their subjects. In addition, during the sixteenth and seventeenth centuries when tobacco use began to spread around the world, many societies, including the Ottoman Empire, Great Britain, Russia, and Japan, initially tried prohibiting the substance.

These ancient and more recent laws and codes show that the regulation or prohibition of socially perceived harmful substances is not new to our age, nor is the range of views on the negative consequences of regulation or prohibition and what would constitute a more effective, less harmful policy.

Among the many legacies that underpin the present discussion of drug policy in contemporary society are four at times overlapping and sometimes contradicting philosophical and cultural traditions. The first is the basic American heritage of individual liberty and limited government interference with any variety of human choice, even if that choice is harmful to the individual making the decision and morally repugnant to the majority of society. This position was eloquently argued by British philosopher and economist John Stuart Mill (1806–1873) in his essay *On Liberty* (1859). It perhaps finds contemporary expression in such a social phenomenon as the pro-choice movement and in the proponents of the legalization or decriminalization of drugs.

A second major social tradition is rooted in the moral utilitarian view of government that is also a part of the nation's heritage. The utilitarian perspective, also argued by Mill in his book *Utilitarianism* (1863), emphasized that government had a legitimate right to prohibit the behaviors that actually caused real harm to others. From this viewpoint, government had the right and responsibility to protect the common welfare by legally prohibiting individuals from engaging in behavior that was demonstrably harmful, not to themselves (which would have been an interference with liberty), but to other citizens.

The moral utilitarian perspective was an important underlying element in many of the late nineteenth- and early twentieth-century social-reform movements that culminated in the many state laws prohibiting narcotics and other drug use and the national HARRISON NARCOTICS ACT OF 1914 and the Volstead Alcohol PROHIBITION Act of 1920. The utilitarian perspective was that narcotics and alcohol use caused real harm to others and society in general in the form of family poverty, crime, violence, and health-care costs.

A third social tradition that has influenced U.S. drug policy is commercialism. There is ample evidence that through the nineteenth century, U.S. society had a strong commercial attitude toward alcohol use and the use of a variety of powerful drugs. As has been documented by historians, merchandise catalogs, as well as a variety of traveling entrepreneurs, legally distributed OPIUM, BARBITURATES, and COCAINE as wonderful cure-alls for the ills of the human condition. These merchants were an organized, respected part of the commercial establishment. Perhaps based on British narcotics commercialism, there has always been a commercial attitude toward alcohol and drug distribution in the United States. From the commercial perspective, alcohol and drugs are a wonderful commodity. They are often rapidly metabolized, highly addictive, and easily distributed, However, by the end of the nineteenth century, this rather freewheeling distribution of drugs caused a widespread public reaction that became incorporated into a variety of health- and social-reform movements.

A fourth significant element in the development of national alcohol and drug policy is a public-health perspective. As was noted, at the turn of the twentieth century, the United States was in the midst of major social and health reforms. After the passage of the 1906 Pure Food and Drug Act, a host of public-health-based government bureaus and regulations emerged, focusing on improving the quality of meats and other foods and requiring the accurate labeling of drugs. In addition, the American Medical Association initiated major reforms in the medical profession, eliminating over-the-counter narcotic drug advertisements in their journal and supporting the licensing of physicians as the only legitimate prescriber of many drugs. The public-health reform movements attempted to decommercialize drug distribution and make drug use a medical, not commercial, decision. The passage of the Harrison and Volstead acts probably

represented a significant triumph of the moral utilitarian and public-health perspectives.

Following the Harrison Act and further legislation, the U.S. government instituted various bureaus and departments to carry out law enforcement and antidrug educational programs. Any review of the education programs of the Bureau of Narcotics would tend to conclude that they primarily constituted a heavy dose of propaganda with little basis in scientific fact. The federal proclivity for restricting the availability of drugs and arresting users and dealers continued strongly through the 1960s. During the decades following the Harrison Act and until the 1960s, the media and government were fairly united in their opposition to drug use, and there were few questions about the efficacy of drug laws or the social policy on which those laws were based.

In the 1960s, U.S. society experienced the coming of age of the first of the baby boomers—those born between 1946 and 1958. By their sheer numbers, a proportion of this generation challenged the traditional socialization mechanisms of society and significantly questioned traditional assumptions, rationales, explanations, and authority. In a drive for generational self-discovery, drug use, particularly as a means to alter consciousness, became a part of the youth movement of the late 1960s and the 70s. Most of the baby boomers who used drugs explored the use of MARIJUANA and HALLUCINOGENS, but over the same years HEROIN use was increasing in inner cities across the country; crime, too, was increasing. Despite the declaration of a "war on drugs" by the Nixon administration in 1970 through 1971, national surveys conducted during the 1970s and early 80s showed annual increases in almost all types of drug use among high school seniors, household residents, and criminal justice populations. The one exception was heroin, the major target of the Nixon drug war. Heroin use levels declined and then remained stable, but COCAINE use rose dramatically during the 1970s and early 1980s, as did marijuana use among young people. By 1985, more than 20 percent of U.S. adults had taken drugs illegally, and for persons aged 18 to 34 more than 50 percent had done so.

Perhaps because of the fundamental changes in national drug-using behavior that occurred during this period, the modern movement to legalize drugs began. The basis of the argument was that (1) many of the drugs that were then illegal were not as harmful as government and media propaganda portrayed them to be; (2) drugs such as marijuana were relatively less harmful than alcohol and tobacco; and (3) the use of marijuana was a generational choice. In fact, the 1978 NATIONAL HIGH SCHOOL SENIOR SURVEY showed that in the prior thirty days, a higher proportion of seniors had smoked marijuana than had smoked tobacco. By 1979, the media and American households were holding serious discussion about the legalization of marijuana, moving toward the BRITISH SYSTEM of heroin maintenance, and considering the legalization of cocaine as a nonaddictive stimulant. Social political movements such as NORML were organized to achieve passage of laws decriminalizing marijuana use. With the tacit support of the Carter administration, there were eleven states, including Alaska, that decriminalized the possession of small amounts of marijuana for personal use. Even the director of the National Institute on Drug Abuse in the late 1970s, Robert Dupont, appeared to accept the likelihood that marijuana would be decriminalized. However, in 1977, in reaction to growing marijuana use by young people and a perception that government itself was being tolerant of drug use, groups of parents organized a grassroots campaign to buttress the resistance to drug law liberalization. By 1978, the PARENTS MOVEMENT had become a force to be considered, and their views had ready access to the White House policy office. The apparently about-to-be-successful national movement to legalize many drugs in the 1970s came to an abrupt end with the 1980 election of President Ronald W. Reagan.

Corresponding with the election of President Reagan, there was a general conservative shift in national consciousness. First Lady Nancy Reagan, who made drug use among young people one of her prime topics of concern, was a welcome speaker at annual national meetings of the parents groups. The public debate on legalization during the early 1980s was also affected by increasing evidence of the physical and psychological consequences of drug use, declining illegal drug use among high school students, decreasing use among household members, and, maybe, the initiation of maturation among the baby boomers. During the 1980s, U.S. policy was characterized by the increasing intolerance of drug addiction or even recreational drug

use. On an official level, this came to be called ZERO TOLERANCE.

According to the official federal policy of the 1980s, the assumption was that to a large extent drug use was an individual choice that could be affected by raising the cost of drug use to the users. It was believed that if enforcement reduced the availability of drugs, thus raising their prices, and increased the consequences of use by increasing the severity and certainty of punishment, individuals would choose to say no to illegal drug use. During the 1980s, funding shifted from a balance between demand reduction (treatment) and supply reduction (enforcement), to one primarily focusing on enforcement. The federal government became disengaged from a primary responsibility for treatment, while at the same time it increased its involvement in enforcement. The change in support was not dramatic at first. The total federal budget for all demand-side and supply-control activities was about $1.5 billion in 1981, with about two-thirds allocated to law enforcement and supply control. This amount escalated sharply, starting in 1986, when President Reagan redeclared a "war on drugs." By 1989, the total had reached $6.7 billion, with two-thirds allocated to controlling drug supply. The resources escalated still further during the Bush administration, reaching $12.2 billion in fiscal year 1993.

By the end of the 1980s, the national drug-abuse policy of zero tolerance with a heavy focus on enforcement without any comparable increase in support for treatment began receiving critical reviews from policymakers, public administrators, clinicians, and academic researchers. These critical reviews were generally based on civil libertarian and public health harm-reduction perspectives. The key points made by national policy critics were:

1. About two-thirds of all felony arrestees in major metropolitan areas were currently using cocaine.
2. A large proportion of all criminal charges were drug charges. This had resulted in a significant expansion of prisons and the proportion of the population incarcerated. All this had occurred at a very high economic cost.
3. The high profits from the drug trade were funding international terrorism and resulting in a rapidly increasing rate of violence in American urban areas.
4. Because of the vast amount of cash generated in the drug trade, there was corruption at every level of each branch of government.
5. In an attempt to reduce illegal drug use, draconian laws focusing on search and property seizures had been passed that undermined hard-won civil rights.
6. Treatment availability for the poor had been reduced, with many cities reporting month-long waiting lists for publicly funded treatment slots.

All these real consequences have resulted in a major reinvigoration of the interest in legalizing or decriminalizing drug use. Those who argue for legalization come from a wide variety of professions and ideological positions, but they all essentially believe that U.S. society has reached the point where it can no longer afford to enforce existing law. There simply are not enough police, courts, prosecutors, or jail cells, nor is there the sense of justice that will allow U.S. society to enforce laws that have been broken by more than 20 percent of U.S. citizens.

In summary, the zero-tolerance just-say-no policy of the 1980s had come to be viewed by critics as resulitng in a virtual saturation of the criminal justice and prison system with drug law offenders, the undermining of crucial civil rights, and the decreasing availability of drug treatment for the poor accompanied by increasing violence in high drug-trafficking areas and large-scale public corruption. Many critics came to view drug laws as contrary to the very basis of a libertarian civil government. These critics saw the war on drugs declared in the 1980s and continued to the present as inimical to civil liberty. In addition to the civil libertarian perspective, there are many critics of current drug-prohibition policy that focus on a public-health harm-reduction perspective. From this perspective, current policy is not reducing the public-health harm caused by drug use. Strict law enforcement and reduction in treatment availability have resulted in denying treatment to those being personally harmed by drug abuse. The public-health-reduction model emphasizes that drug abuse and addiction are the product of a complex set of psychological, sociological, and economic variables that are very little affected by the threat of prison. This perspective argues that the best way

to reduce the personal and public-health harm of drug use would be to increase drug education and prevention, increase drug-treatment availability, and reduce the harm caused by drug abuse by providing clean needles and, perhaps, decriminalizing use—thus significantly reducing the cost of drugs and the associated CRIME.

Although there are very few detailed legalization proposals, those who advocate decriminalization generally argue that national policy should move toward an approach in which the distribution of drugs such as marijuana, cocaine, and heroin would not be governed by criminal law but by governmental regulations that controlled the manufacture, distribution, and use of these substances so that they would go only to those already addicted or be dispensed under very regulated conditions. Advocates of this policy believe that the movement of drug policy from criminal law to regulatory restrictions would result in the relatively easy availability of drugs and inexpensive access to them for those who are addicted, thus resulting in a significant reduction in corruption and violence as well as an increasing willingness on the part of addicts to enter treatment. This, it is asserted, would relieve the severe overcrowding of the criminal justice system. At the same time, it is argued, because of strict regulation, this policy change would more effectively protect young people as well as public health and safety than the current policy (see Nadelmann, 1988; Wisotsky, 1991).

Critics of the legalization perspective do not question many of the basic judgments of the consequences of the 1980s national policy, but they do severely question the assumptions on which legalization is based. Those who are opposed to drug legalization often draw on the moral utilitarian and public-health perspectives. They make the following arguments:

1. During the 1980s and continuing into the early 90s, drug use, by all measures, significantly decreased among high school and college students as well as in the general population.
2. It is naive to assume that increasing availability, lowering cost, and reducing legal consequences will have no effect on the incidence and prevalence of marijuana, cocaine, and heroin use. From this perspective, it is argued that once these drugs are legalized, even though regulated, they will enter the arena of advocacy through free speech and thus the realm of market creation and expansion through advertising. Alcohol use, which is severely regulated and illegal for those under 21 years of age, is initiated in junior high school. In addition, about a third of high school seniors report being drunk each month. In most states, tobacco cannot be sold to minors, but smoking among junior high school students is common. These facts imply that regulation to make a drug available to one age group actually makes it available to all age groups.
3. The resulting increase in use in society and broadening of the societal base of use will result in detrimental health, behavioral, and economic consequences that will far outweigh any proposed benefit of legalization.
4. There is no broad societal base for legalizing drugs. Surveys among high school seniors clearly show that a large majority oppose the legalization of drugs—even the legalization of marijuana. Traditionally liberal countries such as Switzerland and SWEDEN have tried relaxing drug laws and were forced to modify their positions by their citizens, who daily had to experience the consequences of wide drug availability. Additionally, in a referendum in November 1991, Alaskans voted to rescind a marijuana legalization law passed in the 1970s and recriminalized marijuana possession. In a democracy, governmental policy cannot ignore the voice of the public. Finally, in the first presidential debate of the 1992 election, one of the few things that all three candidates agreed on was that drugs absolutely should not be legalized and that the criminal justice system plays a useful role in forcing users into treatment. Dr. Joycelyn Elders, the first Surgeon General in the Clinton Administration, was criticized for merely suggesting that the issue of legalization should be debated.
5. In these times of concern with HIV infection and AIDS, it may be hard to conceive of popular or governmental support for any policy that may increase intravenous-drug use.
6. Although the costs of drug law enforcement and incarceration of offenders may seem high, it is a misconception to assume that those incarcerated are all petty first-time violators of the drug laws. DiIulio (1993) asserts that "... in 1991 more than 93 percent of all state prisoners were

violent offenders, repeat offenders (one or more prior felony convictions) or violent repeat offenders.'' He suggests that the vast majority of ''drug'' criminals were not arrested of simple possession, but of sale or manufacture. In short, most people would probably want to have these offenders behind bars even if the antidrug laws did not exist.

Many of those opposed to legalizing drugs, such as former Secretary of Health, Education and Welfare Joseph A. Califano, Jr., and Mathea Falco, a former Carter administration official, argue that the existing policy should be drastically modified to increase the availability of treatment and educational and economic opportunities in societal groups with high drug-use rates. Specifically, what is called for is an increase in treatment availability in the criminal justice system, either through diversion or probation to treatment or through the provision of therapeutic services in jails and prisons, as well as a major increase in the availability of publicly funded treatment slots in the United States. It is argued that every dollar invested in treatment results in several dollars saved in terms of other social costs, including crime.

Some who oppose drug legalization believe that the current discussion has subtly eroded the public's will to fight illegal drug use. From this perspective, the only way to retain the *reduction* in general societal drug use that occurred during the 1980s is to retain a vigorous enforcement of drug laws. The advocates of strict law enforcement have taken note of the most recent high school surveys and other studies that indicate an increase in drug use among students, and they believe that this increase reflects the weakening of the war on drugs in the current administration and a kind of backdoor legitimization, a demoralizing discussion of the failure of drug policy. Previous drug policy leaders such as William J. Bennett argue that national drug policy during the 1980s was effective in reducing drug use in the general youth and adult population by making use morally, socially, and legally unacceptable and that the current discussion is making drug use more acceptable, resulting in recent increases in use (Bennett & Walters, 1995a, 1995b; Rosenthal, 1995). Bennett and Walters do not believe that support of treatment programs is a useful investment, and they would leave it to state governments to decide to exactly what degree treatment should be supported.

Although it may be very difficult to reconcile the extremes of the drug legalization debate, there is some common ground that could emerge into a broadly acceptable public policy. Many involved in the current drug policy debate share a common belief that there is a need for increasing drug education, prevention, and treatment availability, as well as expanding economic opportunities.

Some of those on both sides have strongly endorsed the need to restore the balance between interdiction and treatment in favor of treatment. Ignoring federal responsibility for treatment has been disastrous. Both sides would probably agree that a crucial priority for the federal administration would be to provide treatment availability for all those who seek it and to incorporate drug-abuse treatment into national health-care policy. In addition, many on both sides of the debate would probably also agree as to the convincing need of addressing basic questions of educational and economic opportunity, as well as that of institutionalized racism, which may function as societal underpinnings of drug-use epidemics.

(SEE ALSO: *Anslinger, Harry J. and U.S. Drug Policy; Crime and Drugs; Opioids and Opioid Control: History; Prevention Movement; Prohibition; Temperance Movement; U.S. Government Agencies*)

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Safer Use of Drugs People commonly use drugs in safe ways, that is, nonabusively. Safe use means that drug use does not significantly impair health or interfere with social or economic functioning. For example, most users of alcohol consume that drug in moderation, not to the point of extreme intoxication, during specified hours, and for specified purposes, such as relaxation after daily work or promotion of social interchange.

Any drug can be used or abused, although some drugs and some ways of introducing them into the body may favor safe use. In general, less potent forms of drugs taken by mouth are more likely to be associated with safe use, whereas more potent forms taken parenterally (that is, introduced other than by way of the intestinal track) are less likely to be associated with safe use.

It is difficult to discuss the safe use of illegal drugs, because foes of those substances regard them as "drugs of abuse" that cannot possibly be consumed in nonabusive ways. This attitude is unhelpful. Whether a drug is used or abused has little to do with whether a drug is legal or illegal; it depends, rather, on the relationship an individual forms with it. One can as easily find examples of abusive use of legal drugs (TOBACCO, ALCOHOL, and OVER-THE-COUNTER medications) as of safe use of illegal ones. Take for example, the majority of coffee drinkers in our society who are addicted to the CAFFEINE in coffee (meaning they will have a withdrawal reaction on sudden cessation of intake). Many of these people also experience adverse effects on health as a result of their coffee addiction (cardiac arrhythmias, stomach and intestinal problems, irritation of the urogenital tract, tremors, insomnia, mood swings, and more). Many users of MARIJUANA, however, consume that drug moderately and occasionally, without suffering ill effects on health or behavior.

By observing safe use of drugs throughout the world—from Native Americans who use HALLUCINOGENIC PLANTS ritually to the many people who have figured out how to enjoy alcohol, tobacco, and caffeine nonaddictively and nonabusively—one can draw up a list of suggestions for users to increase the likelihood of safe use.

1. Know that the substance you are using is a drug or contains a drug.
2. Know how it affects your mind and body and what the risks are of moderate to excessive use.
3. Use lower potency (dilute) forms of drugs rather than higher potency (concentrated, refined) forms.
4. It is always safer to take drugs by mouth rather than by other routes of administration.
5. If the substances are illegal, it is important to know your sources in order to avoid adulterated, toxic, or misrepresented products.
6. Limit frequency of use by defining appropriate occasions and purposes for use. Regular, especially daily, use of any psychoactive drug com-

monly leads to loss of desired effects (tolerance) and to dependence.
7. Do not use any drug without good reason or just to go along with the crowd.
8. Seek advice about drugs from books and from people who know from experience what their real benefits and risks may be.
9. Reactions to drugs are strongly shaped by dose, mind set (expectations) and setting. Pay attention to these variables to reduce the risk of bad reactions.

Clearly, it is in society's interest to discourage the unsafe use of drugs. It is also in society's interest to foster the safe use of drugs by those who are inclined to use them. Of course, abstinence is a sure way to avoid problems, but there is no reason to think that most people will choose it in regard to drugs any more than they choose it in regard to sex. Therefore, providing good education about ways of using drugs safely should be a priority along with encouraging abstinence.

In addition, government drug policy should not work against safe use. Strongly prohibitionist policies may drive out of circulation dilute, natural forms of drugs, while encouraging the growth of black markets in concentrated, refined, and adulterated forms. This has certainly been the case with coca leaf and COCAINE. Coca leaf, with a low abuse potential and significant medical usefulness, has disappeared from our world, as powder and CRACK-cocaine have become more available—a change that has favored unsafe use rather than safe use. It would therefore be in society's interest to make dilute, low-potency forms of natural drugs more available.

(SEE ALSO: *Drugs from Plants; Education and Prevention; Partnership for a Drug-Free America; Prevention Movement*)

ANDREW T. WEIL

POLYDRUG ABUSE This term refers to the common observation that individuals who are considered drug abusers often abuse more than one type of drug. Almost all drug abusers smoke NICOTINE cigarettes and a large proportion consume alcoholic beverages, but many of them do not consider the co-occurrence of these two forms of drug use as an instance of polydrug abuse.

There are several types of polydrug abusers. They include those who abuse two or more substances but with a definite preference for one; only when they are not able to get supplies of their preferred drug do they abuse other types of drugs. These other types of drugs may either be from the same pharmacological class (e.g., HEROIN abusers may abuse other NARCOTICS as CODEINE or Demerol) or from different pharmacological classes (e.g., STIMULANT abusers—such as COCAINE abusers—may also use heroin, a narcotic). Some polydrug abusers do not necessarily have a favorite drug but instead may select different drugs for consumption at different times (e.g., stimulants in the morning, SEDATIVES at night) or under different conditions.

Polydrug abuse can also refer to the consumption of a drug to counteract an unpleasant effect produced by another drug or by withdrawal from another drug. For example, individuals who take enough stimulants to become highly agitated and aroused may take a tranquilizer to counteract the unpleasant side effects. Finally, polydrug abuse can refer to the consumption of different drugs simultaneously (e.g., speedballs). The assumption is that the different drugs in combination constitute more than the sum of their individual parts, producing a unique, highly reinforcing effect.

(SEE ALSO: *Barbiturates: Complications; Drug Abuse Warning Network; Drug Interactions and Alcohol; Prescription Drug Abuse; Sedatives; Adverse Consequences of Chronic Use*)

CHRIS-ELLYN JOHANSON

POPPY/OPIUM POPPY *See* Opium; Papaver Somniferum

POT *See* Marijuana; Slang and Jargon

POVERTY AND DRUG USE One of the most popular stereotypes about drug use is that it is more prevalent among the poor. In fact, a lack of money—in itself—does not seem to be associated with drug use. Empirical research has found, however, that in the United States, a number of attitudes, behaviors, and conditions linked to drug use

also are linked to poverty, thus creating a situation that encompasses more than a lack of money. The study of poverty and drugs in the United States is complicated by the complexity of poverty as a conceptual category and by methodological problems in the measurement of drug use.

Merriam-Webster's Collegiate Dictionary, Tenth Edition defines poverty as "the state of one who lacks a usual or socially acceptable amount of money or material possessions." The sociological definition focuses on the relational aspect of poverty: Poor people are those who are at the bottom of a hierarchy of social stratification. Such a system is marked by unequal distribution of resources and income and also by differences in prestige, lifestyle, and values. In one review of the literature about poverty, authors listed the "critical features" of poverty—attitudes, behaviors, or conditions that are believed to distinguish poor from people who are not poor. Poor people often are categorized by unemployment or intermittent employment, low-status and low-skill jobs, unstable family and interpersonal relationships, low involvement in the community, alienation from the larger society, low aspirations, and individual feelings of helplessness. Poverty was also correlated with divorce and unhappy marriage, illegitimacy, low rates of voting, dropping out of school, high arrest rates and incidence of mental disorders, poor physical health, and high mortality rates. The literature concluded that poor people differed quantitatively, but not qualitatively, from people who were not poor; that is, the differences in their attitudes, their behaviors, and their conditions were differences of degree, not kind. Interestingly, however, extreme poverty is not necessarily linked to a lack of education. Some research has shown that drug users with little education were less likely to be homeless than those with considerably more education, perhaps because those with less education look for and easily find unskilled labor jobs, and they earn enough to keep them in stable housing.

When studying the relationship of poverty to drug use, some of the literature is devoid of attempts to use the multidimensional conception of poverty. Instead, researchers have tended to choose one critical feature and look at its relationship to the use of specific drugs. Such studies have examined the association of U.S. drug use with income; educational attainment; educational success; employment; mental health; HOMELESSNESS; and neighborhood. The results of these studies are largely inconclusive, thereby pointing not to a simple correlation between poverty (or poverty-linked attitudes, behaviors, and conditions) and drug use, but to more subtle pathways of direct and indirect effects.

Some of the sociological literature on poverty since the 1980s has focused on the concept of an American underclass—a population caught in an intergenerational cycle of poverty, isolated from mainstream society, living in an urban ghetto, and at risk for a number of social ills, including drug use. It should be noted that only a small proportion of poor people lead lives fitting this description. Many poor people are poor for only a short time. Poor people are also a highly heterogeneous group. They live in all regions of the United States, in both rural and urban areas, and they are represented in all age and ethnic groups.

Collecting valid information about poverty and drug use has proved to be methodologically problematic. For example, for various reasons, some individuals misrepresent the severity of their drug use or their level of poverty. In addition, some surveys of drug use are based on household samples. Those who are poor are less likely to live in stable households and more likely to live in extended or amorphous households—both situations that would result in their being excluded from such a survey. Some reporting of drug use also comes from testing of arrestees, and this may introduce a bias in the estimation of the amount of drug use by people who are poor. Some statistical information on the drug-using population also comes from treatment programs or outreach services and not all individuals with drug abuse problems seek such programs or services. A number of individuals avoid treatment programs because problems with mental health interfere with their ability to desire or seek treatment. Finally, many studies focus on certain drugs (e.g., crack-cocaine, HEROIN) and not others (e.g., MARIJUANA, COCAINE), and this may tend to misrepresent the extent of drug use among poor people as compared to its extent among people of the middle and upper classes.

Regardless of these many obstacles, researchers have reached some conclusions about drug use among the poor, especially the extreme poor—the homeless. The homeless do appear to be at higher risk for drug abuse, and some findings suggest that drugs may have displaced alcohol as an important

precursor of homelessness for many people. Researchers have also found that the homeless population is no longer primarily older, white males, but that women now make up a large portion of the homeless and that among them are many drug users.

One area in the study of drug use among the poor that has recently received much attention is the prevalence of mental health problems among the drug-using, homeless population. Researchers have found that this is a heterogeneous population in which not all individuals have the same health problems or severities of drug abuse, but studies have found a high incidence of mental health problems among homeless substance abusers. Some of the disorders seen in connection with this population include mood disorders, conduct and antisocial personality disorders, and anxiety disorders. Researchers have speculated that individuals with mental health problems use drugs as a means of self-medicating. Studies have also suggested that dealing with the mental health of drug-abusing homeless individuals may take first priority in the treatment of these individuals because mental health problems can prevent people from finding stable housing situations, getting a job, connecting with family, and staying with a drug-treatment program.

The risk of HIV infection among impoverished drug users is also an issue of increasing concern. Youths with mental illness are at particular risk for HIV infection as they have been found more likely to engage in such risky behavior as prostitution and unprotected sex, drug dealing, and drug use by injection.

Perhaps the greatest impact of poverty on the life of a drug user is how it can make prevention and treatment efforts inaccessible to that person. With private inpatient and outpatient treatment costing thousands of dollars and the long waiting lists for admission to publicly funded programs, impoverished drug users are less likely to obtain access to treatment. The heterogeneity of the poor and the lack of an empirical association between income level and drug use imply that making the poor the object of a targeted prevention and treatment effort might not be successful. Instead, the extant research on poverty and drug use suggests that policy efforts be directed at ensuring that lack of money does not become a barrier to participation in prevention and treatment programs.

Researchers have also suggested that special efforts must be made to target homeless youths due to their high risk of drug abuse. The range of services needed included outreach and sheltering services, substance abuse treatment, counseling, and HIV prevention programs. Unfortunately, many youths who engage in risky behavior do not seek traditional services or programs, and consequently those most in need may be underserved.

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(SEE ALSO: *Alcohol: History of Drinking; Ethnicity and Drugs; Families and Drug Use; Homelessness and Drugs; Vulnerability As Cause of Substance Abuse*)

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PREGNANCY AND DRUG DEPENDENCE: OPIOIDS AND COCAINE During the 1980s, increasing numbers of pregnant drug-dependent women went to medical facilities—some to receive ongoing prenatal care, but others only to deliver their babies without the benefit of any prenatal care. Such women fear the threat of confrontation with legal authorities. The general lack of women-oriented drug-treatment programs contributes to this major health problem—addiction in pregnancy. It has also contributed to increased medical and social maladies and mortality in such mothers and their infants.

The 1990 NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE estimated that almost 50 percent, approximately 29 million of the 60 million women of child-bearing age, used an illicit drug at least once in their lifetimes. In 1988, one study reported for the United States an annual occurrence rate (prevalence) of 11 percent, resulting in an estimated 375,000 drug-exposed births; these data cannot be applied to the entire country, since they were collected from a limited number of mainly urban hospitals—and the frequency, amount, type, and duration of drugs used were unavailable. The basis is also unclear for the reported estimates of 50,000 to 100,000 cocaine-exposed babies born each year. The occurrence of drug abuse among pregnant women varies widely in local studies—from 7.5 percent in Rhode Island, to 14.8 percent in Pinellas County, Florida, to 17 to 31 percent in a Boston hospital. These local rates cannot be used to estimate the prevalence of drug abuse among pregnant women in the United States; they can only provide data for averages.

As a result of the uncertainty among data sources, in 1992, the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) began a national hospital-based study known as the *National Pregnancy and Health Survey*. This survey collected data on the prevalence of licit and illicit drug use by pregnant women, limited data on infant birth weight, and the duration of hospital stay. The results were released in late 1994 and the summary tables are included here. Additional surveys in progress include the *National Maternal and Health Survey* conducted by the National Center for Health Statistics, which will collect data on drug-abusing women who had a live birth, stillbirth, or an infant who died before one year of age, and the *National Longitudinal Survey*, which collects data on the frequency of marijuana and cocaine use during pregnancy by women who have given birth to a child since 1986.

OPIOIDS

Due to preexisting conditions and ongoing active drug use, the opioid-dependent woman frequently suffers from chronic ANXIETY and DEPRESSION. Social problems, such as POVERTY, HOMELESSNESS, involvement in an abusive or battering relationship, and ALCOHOLISM, may overwhelm her ability to cope with life activities. She usually lacks confidence and hope for the future and has extreme difficulty with interpersonal relationships, especially with men. One study found that 83 percent of addicted women were raised in households marked by parental drug or alcohol abuse, 67 percent of those women had been sexually assaulted, 60 percent had been physically assaulted, and almost 100 percent of the women wished that they were someone else as they were growing up. In addition to these problems, the treatment and resolution of their addiction is a complex biopsychosocial matter which requires understanding and patience. Addiction is a chronic, progressive, relapsing disease, and one cannot expect a smooth and rapid recovery. It should not be surprising, therefore, that the lifestyle of the pregnant addict has a profound influ-

TABLE 1
Estimated Use of Selected Substances during Pregnancy: Total U.S.

	Percentage		*Population (in Thousands)*	
Substance	*Estimate*	*95% C.I.**	*Estimate*	*95% C.I.*
Illicit drug use and nonmedical use of psychotherapeutics				
Any illicit drug use[1]	5.5	4.2–7.2	220.9	168.1–289.2
Marijuana	2.9	1.9–4.5	118.7	77.1–181.6
Cocaine	1.1	0.8–1.7	45.1	30.5–66.6
Crack	0.9	0.6–1.4	34.8	22.3–54.3
Other cocaine	0.3	0.1–0.7	12.7	6.0–26.6
Methamphetamine	0.1	0.0–0.4	4.5	1.2–17.3
Heroin	0.1	0.0–0.4	3.6	0.8–17.1
Methadone	0.1	0.0–0.4	3.4	0.8–14.2
Inhalants	0.3	0.1–0.7	12.1	5.1–28.6
Hallucinogens	0.2	0.1–0.6	8.7	3.0–25.1
Nonmedical use of any psychotherapeutics[2]	1.5	1.0–2.3	61.2	40.1–93.2
Amphetamines	0.0	0.0–0.3	1.2	0.1–13.4
Sedatives	0.3	0.1–0.8	10.3	3.4–30.8
Tranquilizers	0.0	0.0–0.3	1.9	0.3–12.9
Analgesics	1.2	0.8–1.9	48.7	30.2–78.4
Alcohol	18.8	16.2–21.7	756.9	653.4–872.7
Cigarettes	20.4	18.5–22.4	819.7	744.0–901.0
Medical use of any psychotherapeutics[3]	10.2	7.7–13.6	412.3	308.2–546.3
Amphetamines	0.3	0.2–0.7	13.4	6.4–28.0
Sedatives	3.6	2.3–5.6	144.1	91.3–225.7
Tranquilizers	1.4	0.8–2.4	55.4	31.2–97.9
Analgesics	7.6	5.6–10.2	305.2	226.2–408.9

*Confidence Interval
[1]Use of marijuana, cocaine (all forms), methamphetamine, heroin, methadone, inhalants, hallucinogens, or nonmedical use of psychotherapeutics during pregnancy.
[2]Nonmedical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
[3]Medical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
SOURCE: National Institute on Drug Abuse, 1994.

ence on her psychological, social, and physiological well-being.

She may have several other children who are currently not living with her, but instead with a relative or in placement. Drug-dependent women are frequently intelligent, although the average level of high school achievement is usually at the tenth-grade level. Housing situations are frequently chaotic, and plans for the impending birth of the child may not have been considered.

It is well known that medical complications impact many drug-involved pregnancies; the most frequently encountered complications include anemia, various infections such as pneumonia, hepatitis, urinary tract infections, and sexually transmitted diseases. The women are at risk for human immunodeficiency virus (HIV) disease culminating in acquired immmunodeficiency syndrome (AIDS).

The HIV disease has been increasingly linked to drug usage. The practice of sharing contaminated needles to inject HEROIN or COCAINE, the practice of prostitution to buy drugs, or the direct sex-for-drugs transaction associated with "crack" smoking have all contributed to this serious international health crisis. Currently, the spread of HIV is less linked to homosexual spread and more to hetero-

TABLE 2
Estimated Use of Selected Substances during Pregnancy, by Race/Ethnicity: Percentage, Total U.S.

	Race/Ethnicity					
	White, non-Hispanic		*Black, non-Hispanic*		*Hispanic*	
Substance	*Estimate*	*95% C.I.**	*Estimate*	*95% C.I.*	*Estimate*	*95% C.I.*
Illicit drug use or nonmedical use of psychotherapeutics						
Any illicit drug use[1]	4.4	2.8–6.7	11.3	8.2–15.4	4.5	2.9–7.0
Marijuana	3.0	1.6–5.4	4.6	3.0–7.1	1.5	0.5–3.9
Cocaine	0.4	0.1–1.1	4.5	2.9–7.0	0.7	0.2–2.7
Crack	0.3	0.1–1.1	4.1	2.6–6.5	0.1	0.0–2.1
Other cocaine	0.1	0.0–0.6	0.7	0.2–2.1	0.6	0.1–2.8
Methamphetamine	0.1	0.0–0.7	0.1	0.0–1.9	(no)	(no)–(no)
Heroin	0.0	0.0–0.7	0.2	0.0–1.7	(no)	(no)–(no)
Methadone	0.1	0.0–0.5	0.2	0.0–1.7	(no)	(no)–(no)
Inhalants	0.2	0.1–0.8	0.4	0.1–1.8	0.4	0.1–2.7
Hallucinogens	0.1	0.0–0.6	0.3	0.1–1.7	0.7	0.2–2.2
Nonmedical use of any psychotherapeutics[2]	1.1	0.6–2.0	3.1	1.7–5.4	1.8	0.8–3.8
Amphetamines	0.0	0.0–0.6	(no)	(no)–(no)	(no)	(no)–(no)
Sedatives	***	***–***	1.0	0.3–3.5	0.4	0.1–1.8
Tranquilizers	***	***–***	(no)	(no)–(no)	0.2	0.0–1.7
Analgesics	1.0	0.5–1.9	2.2	1.2–4.1	1.3	0.5–3.3
Alcohol	22.7	19.3–26.4	15.8	11.5–21.3	8.7	5.4–13.7
Cigarettes	24.4	21.7–27.2	19.8	14.9–25.8	5.8	3.8–8.6
Medical use of any psychotherapeutics[3]	11.2	8.0–15.5	10.4	7.2–15.0	6.2	3.9–9.7
Amphetamines	0.3	0.1–0.9	0.4	0.1–1.8	0.3	0.1–1.7
Sedatives	4.0	2.2–7.0	4.4	2.0–9.5	1.4	0.5–4.2
Tranquilizers	1.1	0.7–2.0	2.9	0.8–9.6	0.9	0.3–3.3
Analgesics	8.3	5.8–11.7	7.7	5.6–10.7	4.4	2.7–6.9

*Confidence Interval
[1]Use of marijuana, cocaine (all forms), methamphetamine, heroin, methadone, inhalants, hallucinogens, or nonmedical use of psychotherapeutics during pregnancy.
[2]Nonmedical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
[3]Medical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
, ***– Low precision, no estimate reported.
(no) No observations, C.I. not computed.
0.0 Estimate <0.05, rounded to 0.0 with valid C.I.
SOURCE: National Institute on Drug Abuse, 1994.

sexual transmission and intravenous drug abuse. Although the exact risk of an infected mother passing the disease to her offspring is not precisely known, it is estimated that approximately 25 to 30 percent of infants exposed in this fashion will actually contract AIDS. Counseling in an effort to prevent HIV infection, therefore, forms an essential part of services that must be offered to pregnant substance-abusing women or women involved in relationships with addicted men.

TABLE 3
Estimated Use of Selected Substances during Pregnancy, by Race/Ethnicity: Population (Thousands), Total U.S.

	Race/Ethnicity					
	White, non-Hispanic		*Black, non-Hispanic*		*Hispanic*	
Substance	*Estimate*	*95% C.I.**	*Estimate*	*95% C.I.*	*Estimate*	*95% C.I.*
Illicit drug use or nonmedical use of psychotherapeutics						
Any illicit drug use[1]	113.1	72.8–174.0	75.0	54.2–102.4	28.1	18.0–43.3
Marijuana	77.5	42.5–139.5	30.8	19.9–47.2	9.1	3.4–24.2
Cocaine	9.2	3.0–27.9	30.0	19.3–46.2	4.4	1.2–16.4
Crack	7.0	1.7–28.4	27.2	17.1–42.9	0.6	0.0–12.6
Other cocaine	3.0	0.6–14.8	4.4	1.4–14.0	3.8	0.8–17.1
Methamphetamine	3.7	0.8–17.6	0.8	0.1–11.0	(no)	(no)–(no)
Heroin	0.8	0.0–15.8	1.3	0.1–11.6	(no)	(no)–(no)
Methadone	2.2	0.3–14.2	1.2	0.1–11.1	(no)	(no)–(no)
Inhalants	6.4	2.1–19.6	2.9	0.7–12.1	2.7	0.4–16.7
Hallucinogens	2.2	0.3–15.9	2.2	0.4–11.4	4.3	1.3–13.7
Nonmedical use of any psychotherapeutics[2]	27.8	14.7–52.1	20.4	11.4–36.1	11.0	5.1–23.4
Amphetamines	1.2	0.1–15.0	(no)	(no)–(no)	(no)	(no)–(no)
Sedatives	***	***–***	6.6	1.8–23.3	2.2	0.4–10.8
Tranquilizers	***	***–***	(no)	(no)–(no)	1.0	0.1–10.6
Analgesics	24.9	12.2–50.6	14.8	7.8–27.6	7.8	3.0–20.1
Alcohol	588.6	501.0–686.5	105.0	76.6–141.4	53.6	33.3–84.7
Cigarettes	632.9	564.4–706.8	131.6	98.9–171.7	35.6	23.5–53.3
Medical use of any psychotherapeutics[3]	291.9	208.8–402.5	69.4	47.6–99.6	38.5	24.4–60.1
Amphetamines	8.9	3.5–22.4	2.5	0.5–11.7	2.0	0.4–10.6
Sedatives	102.9	57.6–181.3	29.4	13.3–63.2	8.7	2.8–26.2
Tranquilizers	29.6	17.0–51.5	19.0	5.4–63.6	5.8	1.7–20.1
Analgesics	215.1	150.4–304.1	51.5	36.9–71.1	27.0	16.9–42.5

*Confidence Interval
[1]Use of marijuana, cocaine (all forms), methamphetamine, heroin, methadone, inhalants, hallucinogens, or nonmedical use of psychotherapeutics during pregnancy.
[2]Nonmedical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
[3]Medical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
, ***– Low precision, no estimate reported.
(no) No observations, C.I. not computed.
0.0 Estimate <0.05, rounded to 0.0 with valid C.I.
SOURCE: National Institute on Drug Abuse, 1994.

Nutritional deficiencies associated with drug addiction are due largely to the lack of proper food intake, which may result in iron and folic-acid deficiency anemias. Toxic responses to narcotics may contribute to malnutrition by interfering with the body's ability to absorb or utilize nutrients. Abnormalities result because of the high incidence of altered function of the intestine, liver, and pancreas; malnutrition is often related to the presence of liver disease (since nausea causes addicts to eat infrequently or to vomit). Low sugar levels in the bloodstream or certain vitamin (B_6, thiamine) and

TABLE 4
Estimated Use of Selected Substances during Pregnancy, by Age: Percentage, Total U.S.

	Age in Years					
	Under 25		*25–29*		*30 and older*	
Substance	*Estimate*	*95% C.I.**	*Estimate*	*95% C.I.*	*Estimate*	*95% C.I.*
Illicit drug use or nonmedical use of psychotherapeutics						
Any illicit drug use[1]	5.7	3.5–9.3	5.1	3.4–7.6	5.5	3.9–7.6
Marijuana	3.5	1.7–7.1	2.4	1.3–4.2	2.8	1.8–4.3
Cocaine	0.4	0.1–1.3	1.6	0.9–3.0	1.6	0.9–2.8
Crack	0.2	0.0–0.8	1.3	0.7–2.6	1.3	0.7–2.5
Other cocaine	0.2	0.0–1.3	0.5	0.1–1.5	0.3	0.1–1.1
Methamphetamine	0.2	0.0–1.2	0.1	0.0–1.2	(no)	(no)–(no)
Heroin	(no)	(no)–(no)	0.1	0.0–1.4	0.2	0.0–1.0
Methadone	0.1	0.0–0.8	0.1	0.0–1.4	0.1	0.0–1.0
Inhalants	0.3	0.1–1.2	0.3	0.1–1.3	0.3	0.0–1.7
Hallucinogens	0.0	0.0–1.1	0.2	0.0–1.2	0.4	0.1–1.3
Nonmedical use of any psychotherapeutics[2]	1.7	1.0–2.8	1.2	0.5–2.6	1.5	0.7–3.4
Amphetamines	0.1	0.0–0.8	(no)	(no)–(no)	(no)	(no)–(no)
Sedatives	0.5	0.2–1.7	0.2	0.0–1.2	(no)	(no)–(no)
Tranquilizers	0.1	0.0–0.9	0.1	0.0–1.3	(no)	(no)–(no)
Analgesics	1.1	0.6–2.1	0.9	0.3–2.4	1.5	0.7–3.4
Alcohol	12.4	9.6–15.8	21.8	17.7–26.5	24.0	20.2–28.2
Cigarettes	21.9	18.5–25.8	19.4	16.5–22.8	19.3	15.3–24.0
Medical use of any psychotherapeutics[3]	10.1	6.9–14.4	10.1	7.6–13.3	10.6	6.9–15.9
Amphetamines	0.1	0.0–0.8	0.8	0.3–2.1	0.2	0.0–1.0
Sedatives	5.0	2.9–8.5	2.9	1.6–5.0	2.5	1.3–4.5
Tranquilizers	1.0	0.3–3.4	2.6	1.4–4.9	0.9	0.3–3.0
Analgesics	6.5	4.4–9.6	7.9	5.6–11.1	8.6	5.6–12.9

*Confidence Interval
[1]Use of marijuana, cocaine (all forms), methamphetamine, heroin, methadone, inhalants, hallucinogens, or nonmedical use of psychotherapeutics during pregnancy.
[2]Nonmedical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
[3]Medical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
, ***– Low precision, no estimate reported.
(no) No observations, C.I. not computed.
0.0 Estimate <0.05, rounded to 0.0 with valid C.I.
SOURCE: National Institute on Drug Abuse, 1994.

mineral (magnesium) deficiencies may cause seizures in both alcoholics and drug addicts. Hepatitis, a viral infection of the liver, often accompanies the abuse of injectable drugs; it causes addicts to eat infrequently—due to fatigue, swollen liver, nausea, and vomiting—which in turn diminishes the intake of nutrients, vitamins, minerals, and trace elements. Consequently, intensive diet therapy is needed in correcting drug and alcohol addiction—to balance fluids, electrolytes, trace elements, minerals, and vitamins—especially in acutely ill patients.

In addition to many potential medical problems, the lifestyle of some pregnant addicts becomes bur-

TABLE 5
Estimated Use of Selected Substances during Pregnancy, by Age: Population (Thousands), Total U.S.

	Age in Years					
	Under 25		*25–29*		*30 and older*	
Substance	*Estimate*	*95% C.I.**	*Estimate*	*95% C.I.*	*Estimate*	*95% C.I.*
Illicit drug use or nonmedical use of psychotherapeutics						
Any illicit drug use[1]	91.5	55.5–148.6	55.5	37.2–82.1	73.9	52.8–102.9
Marijuana	55.3	26.7–112.4	25.5	14.2–45.6	37.8	24.5–58.0
Cocaine	6.4	1.9–21.4	17.6	9.3–32.9	21.1	11.8–37.6
Crack	2.6	0.5–13.1	14.1	7.0–28.2	18.1	9.6–33.7
Other cocaine	3.8	0.7–20.6	5.0	1.5–16.1	3.8	1.0–15.0
Methamphetamine	3.3	0.6–18.7	1.1	0.1–13.3	(no)	(no)–(no)
Heroin	(no)	(no)–(no)	0.8	0.0–15.0	2.8	0.6–13.8
Methadone	1.4	0.1–12.8	0.8	0.0–15.0	1.2	0.1–13.7
Inhalants	5.2	1.4–19.4	3.2	0.7–13.8	3.6	0.6–22.7
Hallucinogens	0.6	0.0–18.1	2.2	0.4–12.9	5.9	1.9–18.2
Nonmedical use of any psychotherapeutics[2]	27.4	16.7–44.9	12.9	5.8–28.3	20.9	9.5–45.6
Amphetamines	1.2	0.1–13.0	(no)	(no)–(no)	(no)	(no)–(no)
Sedatives	8.4	2.6–26.6	1.9	0.3–12.7	(no)	(no)–(no)
Tranquilizers	0.9	0.0–14.4	1.0	0.1–13.5	(no)	(no)–(no)
Analgesics	18.0	9.5–33.8	9.9	3.7–26.2	20.9	9.5–45.6
Alcohol	197.4	153.1–252.2	235.6	191.5–286.7	324.0	273.3–380.7
Cigarettes	349.3	295.2–410.3	209.9	177.8–246.2	260.5	207.2–323.6
Medical use of any psychotherapeutics[3]	160.2	110.6–228.8	109.4	82.3–144.1	142.6	92.6–215.0
Amphetamines	2.4	0.4–12.9	8.4	3.0–22.8	2.7	0.5–13.7
Sedatives	80.1	46.5–135.9	30.8	17.5–53.7	33.2	17.2–61.3
Tranquilizers	15.2	4.2–54.5	28.2	14.7–53.3	12.0	3.5–40.5
Analgesics	103.9	69.8–152.8	85.5	60.1–120.4	115.8	75.6–174.5

*Confidence Interval
[1]Use of marijuana, cocaine (all forms), methamphetamine, heroin, methadone, inhalants, hallucinogens, or nonmedical use of psychotherapeutics during pregnancy.
[2]Nonmedical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
[3]Medical use of any prescription amphetamines, sedatives, tranquilizers, or analgesics during pregnancy.
, ***– Low precision, no estimate reported.
(no) No observations, C.I. not computed.
0.0 Estimate <0.05, rounded to 0.0 with valid C.I.
SOURCE: National Institute on Drug Abuse, 1994.

densome. To meet the high cost of maintaining a drug habit, she may often indulge in robbery, forgery, the sale of drugs, and/or prostitution. Because most of her day may be consumed by the activities of either obtaining drugs or using drugs, she spends most of her time unable to function in society's usual activities. She may have intermittent periods of normal alertness and well-being, but for most of the day, she will be either "high" or "sick." The high (euphoric) state will keep her sedated or tranquilized, absorbed in herself, and incapable of fulfilling familial responsibility. The sick (withdrawal) state is generally characterized by craving for more drugs, malaise, nausea, tearing, perspira-

tion, tremors, vomiting, diarrhea, and cramps. Since hormonal changes in pregnancy manifest some of these symptoms in nondrug users, the sick state may be more frequent or intensified for addicts.

IMPACT OF MATERNAL OPIOID USE ON FETAL WELFARE

Opioid dependence in the pregnant woman is not only overwhelming to her own physical condition but also dangerous to that of the fetus (and eventually to the newborn infant). Because of her lifestyle, and because she may fear calling attention to her drug habit, the pregnant addict often does not seek prenatal care. Obstetrical complications associated with heroin addiction include miscarriages, premature separation of the placenta, infection of the membranes surrounding the FETUS, stillbirth, retardation of the growth of the fetus, and premature labor.

Because no quality control exists for street drugs, doses and substances used to stretch the dose may cause repeated episodes of underdose, withdrawal, and/or overdose. Maternal narcotic withdrawal has been associated with the occurrence of stillbirth. Severe withdrawal is associated with increased muscular activity, thereby increasing the rates of metabolism and oxygen consumption; during maternal withdrawal, fetal activity also increases, as does the oxygen need of the fetus. The oxygen reserve in the placenta may not be able to supply the extra oxygen needed by the fetus. During labor, contractions further inhibit the blood flow through the uterus. If labor coincides with withdrawal symptoms in the mother, the fetus will also withdraw. Since uterine blood flow will vary at this time, and less oxygen will be delivered to the fetus, fetal death may occur.

COCAINE

Cocaine is known to cause many medical complications in adult users, including heart attacks, irregular heart beats, rupture of major blood vessels, strokes, fevers, seizures, infections, as well as a range of psychiatric disorders. The medical impact of cocaine on human pregnancy must consider all associated variables such as poverty, homelessness, inadequate prenatal and postpartum care, deficient nutrition, varying types of cocaine usage, multiple drug use, sexually transmitted diseases, and the possible presence of toxic chemicals that are mixed with or used to process cocaine.

Suppression of maternal appetite with inadequate nutritional intake is well recognized in cocaine "binging." Many cocaine users admitted for treatment may have at least one vitamin deficiency (B_1, B_6, C). Correction of these vitamin deficiencies is important during pregnancy so that essential chemicals (neurotransmitters) that transmit messages in the brain can be replenished.

Cocaine's chemical properties (low molecular weight and high solubility) allow it to cross the placenta easily and enter the fetus. The passage from maternal circulation to the fetus is enhanced by the injection or smoking of cocaine. In addition, because of acid/base balance issues and low levels of certain enzymes, which usually metabolize the drug, accumulation of cocaine in the fetus occurs. Furthermore, the "binge" pattern commonly associated with cocaine use may lead to even higher levels of cocaine in the fetus. Transfer of cocaine appears to be greatest in the first and third trimesters of pregnancy. Cocaine has a very potent ability to constrict blood vessels. A deleterious effect of this blood vessel constriction is fetal deprivation of essential nutrients and decreases in the amount of fetal oxygen. In addition to an acute oxygen deprivation, long time use of cocaine may produce a chronic decrease in nutrients and oxygen, leading to diminished growth of the fetus.

The use of cocaine by the mother may also affect the course of labor. CRACK (smokable cocaine in its base form) also appears to increase directly contractions of the uterus and may thus precipitate the onset of premature labor. Higher rates of early pregnancy loss and third-trimester separations of the placenta appear to be major complications of maternal cocaine use. Increased blood pressure and increased body temperature caused by cocaine may be responsible for early fetal loss and later separation of the placenta. The latter is hazardous to the fetus and the mother because of bleeding, shock, and the chance of death for both, if an emergency cesarean section is not performed.

The major fetal effect of cocaine is retardation of growth, resulting in smaller than normal babies at the time of birth. Although animal studies suggest that cocaine may cause malformations of the fetus, data from studies in humans are contradictory. Some reports have shown an increased chance of

abnormalities of the heart, limbs, and urinary tract, but others show no differences; studies in humans have not included large populations, and good scientific methods have not been utilized to control for many other factors that may contribute to abnormalities. Studies like these are very difficult to design for human populations.

It is currently thought that the incidence of malformations in infants as a result of cocaine taken by pregnant women is very low and that those that do occur are the result of disruption in the fetal blood vessels due to the constriction that occurs. This vessel constriction diminishes blood supply, which causes organs to malform at varying stages of fetal development. Abnormalities have been observed in the intestines, the kidneys, and the extremities.

RECOMMENDATIONS TO AMELIORATE THE EFFECTS OF DRUGS ON WOMEN AND THEIR CHILDREN

Despite the increased use of other drugs of abuse, such as cocaine, opioid abuse continues to be a major problem in the United States. Numerous investigators have reported the extremely high incidence of obstetrical and medical complications among street addicts, as well as the increase in medical conditions and death among their newborn infants.

Insufficient data exist for measuring the long-term effects of maternal drug usage. Controversy exists on how best to prevent anti treat the adverse effects of addiction. It now seems clear, however, that providing comprehensive multidisciplinary drug-treatment services and prenatal care for addicts will significantly reduce the medical and psychological conditions and the death rate in both mothers and infants. Recommendations for treatment for drug-dependent women are multifaceted. The pregnant woman who abuses drugs must be designated as high risk; she warrants specialized care in a perinatal center where she can be provided with comprehensive addictive and obstetrical care and psychosocial counseling. Care must be provided in a supportive, proactive, and nonjudgmental fashion. The women must know that sharing of confidential information with healthcare providers will *not* render them liable to criminal prosecution under state law statutes that define drug addiction in pregnancy as a form of fetal abuse.

Treatment of addiction in pregnancy may involve voluntary drug-free THERAPEUTIC COMMUNITIES, outpatient or day treatment, and, in narcotic-dependent women, METHADONE MAINTENANCE. The pregnant drug-dependent woman should be evaluated in a hospital setting where a complete history and physical examination may be performed and targeted laboratory tests carried out to evaluate her overall health status. Opioid dependent women should receive appropriate methadone maintenance, with support from an extensive medical and psychosocial network. Psychosocial counseling should be provided by experienced social workers who are aware of the medical needs, as well as the social and psychological needs, of these women. The pregnant woman addicted to BARBITURATES or major tranquilizers along with opioids should be medically withdrawn during her second trimester in a setting that furnishes appropriate monitoring of fetal well-being.

Maternal-infant attachment should have special emphasis. Parenting skills of these women need to be strengthened in an effort to nullify the anticipated (assumed) increase in child neglect and abuse that occurs in this population. Social and medical support should not end with the hospitalization. An outreach program, incorporating public health nurses and community workers, should be established. The ability of the mother to care for the infant after discharge from the hospital should be assessed by frequent observations in the home and clinic settings. Mechanisms by which to follow and supervise the infant's course after discharge from the hospital must be developed.

The major impact of comprehensive care, coupled with methadone maintenance for opioid-dependent women, has been the reduction of perinatal illness and mortality and the reduction of rates of low birthweight in offspring. Increases in birthweights, in themselves, have dramatically reduced illness and mortality for drug-exposed infants and children (mortality rates for low-birthweight newborns are forty times that of the full-term infants of normal weight).

Moreover, it is known that low-birthweight infants contribute greatly to the population of infants who test as mentally retarded (IQ of 70 or below), as well as those who will have great difficulty in school because they are "poor learners." These

handicapped individuals will be unable to compete fully in our increasingly complex society. In addition, the incidence of cerebral palsy and lethal malformations are increased in low-birthweight infants. Emotional disturbances, social maladjustments, and visual and hearing deficits are also increased. With the increasing number of addicted women, custodial facilities for their mentally and neurologically deficient infants may be necessary if programs do not deal with prevention and treatment during pregnancy.

(SEE ALSO: *Addicted Babies; Complications; Fetal Alcohol Syndrome; Fetus: Effects of Drugs on the; Injecting Drug Users and HIV; Opioid Complications and Withdrawal; Substance Abuse and AIDS*)

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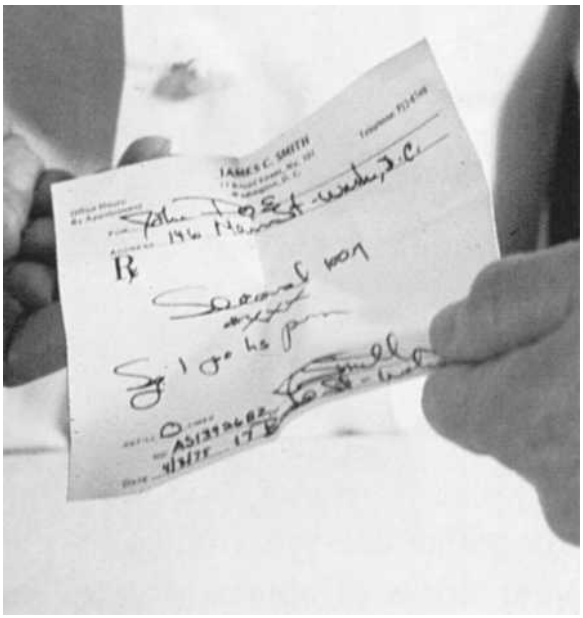
PRESCRIPTION DRUG ABUSE Unfavorable responses to medical treatments—addiction to prescribed drugs or to those used in treatments—is termed *iatrogenic*. A wide array of medicines can be associated with addiction or abuse in some people. Such drugs include the OPIOIDS, antihistamines, anticholinergies, and steroids, among others—but the most common are those prescribed for psychological problems.



Some prescription drugs, such as barbiturates and amphetamines, have a high potential for abuse and dependence when taken in non-prescribed doses or combinations. (Drug Enforcement Administration)

Some drugs acting on the mind have a low potential for abuse and dependence, for example, the ANTIPSYCHOTICS, antidepressants, and lithium salts. Others, such as the BARBITURATES and AMPHETAMINES, have a high potential.

BARBITURATES

Although barbiturates are more or less obsolete as tranquilizers and sleeping tablets, addiction to them is still encountered. TOLERANCE AND PHYSICAL DEPENDENCE can rapidly occur during therapy—and abrupt withdrawal can result in a severe and life-threatening withdrawal state. Studies in abusers show them to greatly prefer barbiturates to BENZODIAZEPINES, which have replaced them pharmacologically and are discussed below. MEPROBAMATE, a carbonate used as a tranquilizer, is similar in many ways to the barbiturates, including its abuse potential.

Clinically, patterns of nonmedical use of nonopioids vary greatly; large quantities can be injected into a vein or muscle, often producing abscess formation. Other users take large amounts by mouth, on a binge or spree basis, the most popular

being pentobarbital, amylbarbital, quinalbarbital, and Tuinal—the amylbarbital/quinalbarbital combination. Some users become permanently intoxicated and totally engrossed in maintaining their supply, licit or illicit. POLYDRUG use in combination with amphetamines or opioids is common.

Withdrawal can be hazardous, with the risk of SEIZURES or psychotic features, when discontinuing chronic usage of 500 milligrams a day or more. Withdrawal DELIRIUM (similar to DELIRIUM TREMENS, DTs) is common and often difficult to treat; a chronic state with HALLUCINATIONS may ensue.

BENZODIAZEPINES

The benzodiazepines supplanted the barbiturates because they seemed to be at least as effective, with few side effects and less likelihood of producing addiction. Benzodiazepines are preferred to placebo by drug abusers but vary in this regard; for example, diazepam (Valium) and lorazepam (Ativan) seem more likely to be taken than is oxazepam (Serax or Serehid). Benzodiazepines have been abused in various countries at various times. They have been injected as the main drug of abuse or as part of a polydrug-abuse pattern. Abusers of alcohol may also abuse benzodiazepines, finding that with drug interaction a potentiation occurs, that is, the combination is particularly powerful. Most benzodiazepine abuse is with drugs obtained legally from a number of complaisant prescribers, but the very heavy user may have to resort to illicit sources of supply. About 50 percent of abusers of benzodiazepines were introduced to the drug within the medical context.

Within polydrug abuse, the benzodiazepine is used to eke out the supply of opioid or to ease the crash from the high euphoria of COCAINE use. Patterns of usage and beliefs about the possible effects of benzodiazepine use vary widely among hard-drug abusers, but, generally speaking, benzodiazepines are viewed as potential drugs of dependence in their own right and not as relatively innocuous adjuncts.

It is fairly uncommon for patients started on benzodiazepines for therapeutic purposes to increase their dosage steadily. Nevertheless, since benzodiazepine use is widespread, high-dose users are seen fairly often. It is unclear why some patients escalate their dosage, whereas most remain at therapeutic levels indefinitely.

AMPHETAMINES

Amphetamines are stimulants, which raise mood, increase the sense of well-being, energy, and alertness, and decrease appetite. Some few users, paradoxically, become the opposite—drowsy, anxious, and irritable.

Normal-dose usage was typically prescribed; an obese, middle-aged, mildly depressed housewife might have taken two or three doses every day as a pick-me-up, a mild stimulant and appetite suppressant. (Some weak physical dependence ensued from such use, mainly seen as sleep changes on withdrawal.) With the discouragement of such indications, usage by physicians and patients has fallen off. Another obsolete use was as a vigilance-enhancer in those who felt the need to keep awake for excessively long periods, such as medical interns or long-distance truck drivers. Few people progressed from iatrogenic oral misuse to intravenous abuse.

Intravenous amphetamine produces euphoria, similar to but more sustained than that following the use of cocaine. Alter a few hours, the effects wear off, leaving the abuser feeling exhausted, drowsy, and depressed. Clandestine laboratories manufacturing amphetamine are still at work. Their preferred substance is METHAMPHETAMINE, which can be synthesized easily. Since intravenous use of methamphetamine is usual, and tolerance quickly occurs, larger and more frequent doses become required to achieve the desired effect. Toxic effects supervene, with repetitive face and hand movements and stereotyped behavior—for example, the user assembling and dismantling mechanical objects. A full-blown paranoid type of psychosis may develop, with loss of reality and delusions of persecution. Individual susceptibility to these toxic effects varies greatly. Polydrug abuse of amphetamines is common; co-administration of amphetamine with heroin (“speedball”) or a barbiturate is believed to optimize the pleasurable effects while minimizing the toxic ones.

APPETITE SUPPRESSANTS

Appetite suppressants cover a range of compounds, from the decongestant phenylpropanolamine (often available without prescription), to powerful amphetamine analogues (chemical variants). Most are stimulant, although

one, fenfluramine, is quite sedative. As with the amphetamines, patterns of use and abuse vary a great deal, from chronic daily ingestion of a therapeutic dose to binge or spree use of large quantities. As a general rule, the more amphetamine- like the appetite suppressant, the more likely it is to be abused.

Trying to stop the use of appetite suppressants may be difficult for abusers, because of withdrawal symptoms such as tiredness, dysphoria (discomfort), or frank depression. These problems and growing doubts about sustained effectiveness (for their original dietary purposes) have led many doctors to cease prescribing them.

In the early to mid-1990's, two prescription diet drugs, fenfluramine (often taken with phentermine and popularly known as fen-phen) and dexfenfluramine (Redux), grew in popularity. These drugs stimulated production of the brain chemical serotonin, creating a feeling of satiety. Stories in the news media hailed fen-phen and Redux as a miracle cure for obesity. By 1996, millions of prescriptions had been written for the diet pills.

In 1997, reports of heart valve disease in women taking fen-phen or Redux began to surface. *The New England Journal of Medicine* published a study by doctors at the Mayo Clinic that reported twenty-four women taking fen-phen developed symptomatic heart valve disorders. At the same time, the Food and Drug Administration issued a Public Health Advisory reporting the Mayo Clinic findings and reporting that it had received reports of thirty-six cases of unusual heart valve abnormalities in women ages 30 to 72 taking fenfluramine or dexfenfluramine.

By September 1997, the drugs dexfenfluramine and fenfluramine were withdrawn from the U.S. market by their manufacturer, American Home Products. In December 1999, the company agreed to compensate thousands of people who took either drug in a $3.75 billion dollar settlement of a nationwide class action suit.

Since 1997, subsequent studies have confirmed a causal link between fenfluramines and valve disorders. There is also persuasive evidence of a significant duration effect. In a 1998 study of more than 17,000 obese patients in the United Kingdom, 92 percent of the patients with symptomatic valve disorders had used fenfluramines for more than 3 months. For those who took fen-phen or Redux for less than 3 months, the risk of heart valve disorders appears to be minimal.

(SEE ALSO: *Iatrogenic Addiction; Obesity*)

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PREVENTION By the 1980s, many urban neighborhoods in the United States became seriously debilitated by the departure of middle-class residents to the suburbs, the influx of illegal immigrants, growing unemployment rates, weak family structures, and a host of other under-class problems. In the mid-1980s, the proliferation of cheap CRACK cocaine, used mainly by inner-city adolescents and young adults, transformed a bad situa-

tion into a desperate one. For some residents, this new upsurge in drug use was the last straw, they got angry and began looking for ways to reclaim their neighborhoods and their children.

The Citizens' Drug Prevention Movement The drug-prevention movement led by private citizens and nonprofit organizations began in the mid–1970s with parents who were concerned about the health and safety of their children. During this decade, drug use among American adolescents escalated from relatively low levels to the highest levels in the history of the world. Some young drug users were addicted and needed treatment. Others were in trouble with drugs but had not yet become addicted. Some were dying of drug overdoses and many were being killed in alcohol– and drug–related automobile crashes.

Many social and environmental factors appeared to contribute to the escalation in the use of alcohol and other drugs among the young. Parents organized to address these factors in order to prevent drug use among young people. In many cases, youth groups also formed to help parents prevent substance abuse among their peers.

Media groups soon organized in response to parents' concerns about the glamorization of drug use on television, in films and in song lyrics, and, more recently, on the Internet that influenced young people. A few years later, the advertising community initiated a campaign to design and air commercials with strong anti-drug messages targeted to children and adolescents.

A drug-related tragedy on the aircraft carrier Nimitz, in which many young servicemen were killed during routine practice maneuvers, brought the military into the prevention movement. It instituted universal drug testing to ensure that such an event would never happen again. The business community adopted drug–testing policies similar to those initiated by the military to prevent drug use in the workplace, particularly in jobs that involved public safety.

Educators and researchers concerned about drug use in primary and secondary schools and on college campuses developed school-based approaches to drug prevention. The law–enforcement community added its voice to the prevention effort through community–policing programs. It also joined forces with the education community through efforts such as DARE (Drug Abuse Resistance Education), in which police officers teach DARE's drug–education curriculum to students in elementary, middle and high schools, and to their parents.

Local, state, and national political leaders created policies and allocated resources to stem the flow of drugs into the country and to help people prevent substance abuse in their families and communities.

Specific ethnic and cultural groups created prevention groups as well. They focused on strengthening their communities through a renewed appreciation of their respective heritages and building on the resiliency that had enabled them to survive the long-term, debilitating effects of racism and poverty.

Seeing the opportunity to contribute its considerable strength and human resources, the faith community also initiated drug-prevention programs. When researchers established the links between substance abuse and the transmission of HIV/AIDS, the AIDS-prevention community joined hands with the substance-abuse prevention community.

Community partnerships and coalitions formed to bring all parts of the local community together to develop and implement substance-abuse-prevention strategies collaboratively.

The citizens' drug-prevention movement continues to expand at this writing. Each component that joins it seeks to create communities in which individuals and families can live healthy lives free of drug abuse and addiction and the problems they generate.

THE PARENT MOVEMENT

Parents initiated the prevention movement in response to the escalation of drug use among teenagers throughout the 1970s. Surveys conducted by the government indicate that in 1962, just seventeen years before drug use peaked at the highest levels in history, less than 2 percent of the entire U.S. population, and less than 1 percent of adolescents, had tried any illicit drug. By 1979, when the use of most drugs peaked, twenty-four million Americans used drugs regularly. Seventy percent of young adults (ages 18–25), 65 percent of high school seniors, and 34 percent of youth (ages 12–17) had tried an illicit drug. Even higher rates of

use occurred with alcohol and tobacco. Ninety-five percent of young adults, 93 percent of seniors, and 70 percent of youth had tried alcohol, while 83 percent of young adults, 74 percent of seniors, and 54 percent of youth had tried cigarettes. One in nine seniors smoked marijuana daily.

Several social and environmental factors appeared to be contributing to this escalation in drug use among young people.

Between 1972 and 1978, eleven states decriminalized marijuana. The political rhetoric that accompanied the decriminalization effort tended to deny or minimize the harmful effects of marijuana and other drugs. State governmental action that equated penalties for marijuana possession with that of a traffic violation tended to reinforce this denial. Most people thought that state legislatures would not make marijuana more available through decriminalization if it were truly harmful.

Prevention research in the early 1970s persuaded some that drug education did not reduce drug use, and government funding for drug education materials ceased. This created a vacuum that was filled by decriminalization advocates and those who stood to profit from increased illicit drug sales. A great deal of the educational materials available throughout the 1970s taught people how to "use drugs responsibly," rather than teaching them what scientists were learning about the harmful effects of drugs. These materials tended to promote drug use rather than prevent it.

As states decriminalized marijuana, an industry emerged to assist people in their drug-taking. This industry manufactured drug paraphernalia—toys and gadgets designed to enhance drug use—and sold it in "head shops," places where so–called "pot heads," "acid heads," "coke heads," and other drug users could go to buy implements to help them take drugs. Head shops also sold promotional materials and "starter kits" targeted to young aspiring drug users. By 1977, some 30,000 head shops were conducting business across the nation.

Each of these factors helped drive drug use up among children and teenagers. Parents organized to help young people who were using drugs stop using them through education, prevention, counseling, or treatment. They also sought to prevent nonusers from starting in the first place, and to reinforce those who decided not to use drugs by emphasizing the desirability of living drug-free lives. Groups that led this effort included the Parents Resource Institute on Drug Education (PRIDE), National Families in Action, the National Federation of Parents for Drug-Free Youth, the American Council on Drug Education and Committees of Correspondence, as well as state groups, such as Texans War on Drugs, Florida Informed Parents, Tennessee Families in Action, and Alaskans for Drug-Free Youth, to name a few, and thousands of local groups in cities, towns, and counties across the country.

Parent groups targeted the social and environmental factors they felt were contributing to the escalation in drug use among young people and developed strategies to address those factors. They did this by first establishing clear definitions. They defined drug abuse to include all illegal drugs and all legal drugs and substances used illegally. These latter included alcohol and tobacco for those under the legal purchasing age, as well as medicines, glue, gasoline, and other substances people abused. Then, parent groups set clear goals: To prevent use before it started, to persuade users to stop and to help those who couldn't stop, find treatment. For alcohol, the goals were slightly different: To prevent use before the legal drinking age, to persuade those who chose to drink when they reached the legal drinking age to follow low-risk drinking guidelines, and to help those who were addicted to alcohol find treatment.

To achieve these goals, parent groups developed several strategies. They mounted an intensive effort to obtain laws to ban the sale of drug paraphernalia. Over a four-year period they succeeded in getting such laws passed in several communities and states. By 2000, nearly every state had such laws. Challenges to the paraphernalia laws were brought by the National Organization for the Reform of Marijuana Laws (NORML), which argued that they were unconstitutional. Many of NORML's board members were also members of the drug-paraphernalia industry. However, in the early 1980s after several conflicting rulings issued by federal district and appeals courts across the nation, the United States Supreme Court upheld these laws as constitutional. This ended the joint effort between NORML and the paraphernalia industry to defeat the paraphernalia laws.

NORML also led the marijuana decriminalization movement. Between 1972 and 1978, the organization persuaded eleven states to

decriminalize marijuana. The parent prevention movement stopped this effort: Parents prevented additional states from decriminalizing after 1978, and defeated a federal effort to decriminalize marijuana nationwide. In some decrim states, such as Alaska, parents worked to re–criminalize the drug, after surveys showed that marijuana use among young people was considerably higher in decrim states than in non–decrim states.

Parent groups placed primary focus on ensuring that drug-education materials convey a no-use message, rather than recommending the "responsible use" of drugs that were both illegal and harmful. They did this by going to the medical and scientific literature and insisting that drug-education materials reflect what was reported in that literature about drug effects.

These strategies seemed to have contributed to the peak and then steady decline in drug use among adolescents, young adults, and the entire population since parents first initiated the prevention movement. The Monitoring the Future Survey, conducted by the government annually since 1975, shows a direct correlation between rates of use and young people's belief that drugs are harmful. The more students who believe a specific drug will hurt them, the fewer students use that drug. Sadly, for reasons not yet fully understood, the steady rise of high school students who perceived drugs to be harmful leveled off and began to decline in the early 1990s. As a result, the steady decline in drug use over fourteen years reversed shortly after. Starting in 1993 student drug use once again began rising and doubled throughout the decade.

A re–emergence of calls for drug decriminalization and legalization worries prevention advocates. This effort was being led once again by NORML and by other organizations that emerged from NORML, including the Drug Policy Foundation and The Lindesmith Center. Many leaders of these organizations were once active in NORML. The glamorization of drug use in song lyrics and films was also reappearing, as were claims by legalization proponents that people can use highly addictive drugs "safely." Whether these shifts in environmental conditions were contributing to the turnaround in drug use was not yet clear. Nor was it clear whether the rise in drug use among high school students was a temporary aberration or a permanent trend. Nonetheless, the prevention community has redoubled its efforts to ensure that drug abuse resumes and sustains it downward trend.

THE ANTI–DRUNK–DRIVING MOVEMENT

At the same time the parent drug-prevention movement was targeting illicit drug use and the problems it generated among young people, another group of parents and families took aim at the problem of drunk driving and the devastation it was creating on the highways, particularly among young people. At the time, deaths from alcohol-related crashes were so prevalent that drunk-driving crashes had become the leading cause of death among adolescents. Families of many young people whom drunk-driving crashes had killed organized groups such as Mothers Against Drunk Driving (MADD), Remove Intoxicated Drivers (RID), and Students Against Drunk Driving (SADD) (now Students Against Destructive Decisions) to stop the carnage on the highways. As with the parent-led, drug-free movement, parents who led the anti-drunk-driving movement first raised the nation's awareness about the problem and then developed strategies to address it.

Among the many contributions this movement has made, perhaps the most significant deals with the age at which young people may legally purchase and consume alcohol. For many years the legal drinking age in every state was twenty-one. During the Vietnamese War, however, when young men aged 18 and over were drafted into the military, most states lowered their legal drinking ages to eighteen in the belief that if young men were old enough to fight for their country, they ought to be old enough to drink. The unanticipated consequence of this action, however, was to further drive down the age at which young teenagers and even pre-adolescents were able to purchase alcohol, albeit it illegally. This led to the appalling rise in the number of young people who were killed in drunk–driving crashes.

As anti–drunk–driving groups tried to persuade state legislatures to return the legal drinking age to twenty-one, their efforts were consistently defeated, year after year, by the alcohol industry, which had considerably more dollars to spend lobbying against such an action. In many states, drug–free parents groups joined forces with MADD, RID and other parent–led, anti-drunk-driving groups,

but to no avail. What broke the log jam was MADD's strategy of advocating for a federal bill that would deny federal highway funds to states that refused to increase the drinking age to 21. Although the alcohol industry also succeeded in defeating the federal effort for several years in a row, the anti-drunk-driving forces finally overwhelmed the industry, and Congress passed the federal bill.

Faced with the loss of federal highway funds, nearly all states have raised the drinking age to twenty-one. The U.S. Department of Transportation estimates changes to the drinking age laws have saved some 13,000 teenage lives to date. Furthermore, when MADD, RID, and similar groups first organized, some 52,000 Americans were killed on the highway each year. About half of those deaths, 26,000, were due to alcohol–related crashes. By the year 2000, both highway deaths and those caused by drunk–driving had been reduced considerably.

These groups continue to work to reduce drunk–driving and the problems it generates by advocating for better enforcement of existing laws, passage of new laws, and effective methods to mandate repeat DUI offenders into treatment.

THE MEDIA

In response to parental concerns about the glamorization of drug and alcohol use in films and on television, several groups organized to address these concerns. The family of actor Paul Newman founded the Scott Newman Center in memory of Mr. Newman's only son, Scott, who died of an overdose of alcohol and drugs. The Center bestowed awards to producers and writers who created television programs and films that contained strong no-use messages and that enhanced the public's understanding of substance-abuse issues and ways to deal with them successfully. The Entertainment Industries Council has advanced this strategy with its PRISM Awards, conducted in partnership with the National Institute on Drug Abuse. The Council also developed programs to work with film makers to educate them about the issue of substance abuse and to encourage them to de-glamorize drug use in movies. The National Academy of Television Arts and Sciences implemented strategies to enhance the industry's awareness of the impact it could have in reducing substance abuse through the power and reach of the mass media.

In the mid-1980s, advertising and public–relations agencies formed the Partnership for a Drug-Free America. These agencies volunteer their talent and time to create and produce anti-drug commercials targeted to young people. The Partnership originally solicited free air time and space in the electronic and print media in which to place these commercials and ads, securing several billion dollars worth of media placement for the anti-drug messages it created. In the late 1990s, after media interest in drug abuse waned and coverage of the issue plummeted, the Partnership and the federal government joined forces. Congress appropriated $195 million over five years to purchase time and space in the media to conduct a public–education campaign to prevent drug use among young people. The campaign appeared to be working, driving drug use down 21 percent among adolescents between 1997 and 1999.

ETHNIC AND CULTURAL GROUPS

The introduction of crack in the mid-1980s made cocaine cheap and plentiful and brought illicit drug use and addiction into poverty-stricken communities that had heretofore succeeded in avoiding massive use of illicit drugs. Members of African American, Hispanic and Latino, native American, and Asian American communities organized to prevent drug use, drug addiction, and drug-related crime in their communities. The passage of the first Anti-Drug Abuse Act of 1986 assisted this effort. With it, Congress made demonstration grants available to local groups to prevent substance abuse among youth at high risk of becoming involved with illicit drugs.

The resulting movement mounted intensive efforts to confront the consequences of poverty and racism. One consequence was to have made poor communities more vulnerable to drug use and the health and social problems it created. Ethnic and cultural groups organized to confront these problems, helping addicts find treatment and reclaiming their communities from drug dealers. They also address other environmental factors, taking aim at the tobacco and alcohol industries' efforts to target ethnic communities for increased consumption of their products. They have defeated the introduction of new brands of cigarettes and alcoholic beverages

targeted to African Americans and Hispanics and Latinos. Campaigns to eliminate the disproportionately high numbers of alcohol and tobacco billboards located near schools and churches in inner-city neighborhoods have resulted in outright bans of such advertising in at least two American cities, Baltimore and Cincinnati.

COMMUNITY PARTNERSHIPS

In 1989, the Robert Wood Johnson Foundation invited communities to submit proposals to establish Community Partnerships, bringing together all segments of the community—parents, young people, schools, ethnic and cultural groups, religious institutions, businesses, local governing bodies, and social and civic organizations—to reduce substance abuse. So many communities responded to the foundation's invitation that the government arranged for $100 million in assets seized from drug smugglers to make even more funds available to communities to establish partnerships to prevent substance abuse and related problems.

As the community coalition movement grew, the Foundation funded Community Anti-Drug Coalitions of America (CADCA) to lead it and join together, to provide technical assistance to coalitions and others. The Foundation also funded the Center on addiction and Substance Abuse at Columbia University to conduct research on substance-abuse issues.

Most authorities credit the activities of this sustained grass-roots, drug–prevention effort, as well as strategies implemented by federal, state, and local governments, with contributing to the reduction in drug abuse, drug addiction, and drug- and alcohol–related deaths that have occurred since the late 1970s. While drug use has increased since 1992, these increases in most cases are still below the levels of 1979. Reductions since then include the following:

- The number of Americans who are current users of illicit drugs was cut in half, from 24.0 million in 1979 to 11.0 million in 1992. The 1999 National Household Survey on Drug Abuse shows that current drug use rose to 14.8

TABLE 1

Drug	*Age Group*	*1979*	*1992*	*1999*
Any Illicit Drug	Young Adults	38.0%	13.1%	18.8%
	Seniors	38.9%	14.4%	25.9%
	Youth	16.3%	5.3%	9.0%
Marijuana	Young Adults	35.6%	10.9%	16.4%
	Seniors	36.5%	11.9%	23.1%
	Youth	14.2%	3.4%	7.0%
Cocaine	Young Adults	9.9%	2.0%	1.9%
	Seniors	5.7%	1.3%	2.6%
	Youth	1.5%	0.3%	0.7%
Alcohol	Young Adults	75.1%	58.6%	60.2%
	Seniors	71.8%	51.3%	51.0%
	Youth	49.6%	20.9%	19.0%
Cigarettes	Young Adults	42.6%*	41.5%	41.0%
	Seniors	34.4%	27.8%	34.6%
	Youth	12.1%*	18.4%	15.9%

The significant reductions in drug abuse, drug addiction, and in drug-related deaths that have occurred over two decades suggest that prevention efforts should be continued and expanded and that private sector prevention efforts should be funded to increase positive gains.

*These figures are taken from the *Overview of the 1991 National Household Survey on Drug Abuse.* Final data were eliminated from later versions of the survey, and no information about cigarette use is available for youth or young adults for 1979.

million by the end of the decade. Current cocaine use, which peaked in 1985, dropped from 5.8 to 1.3 million in 1992, and rose to 1.5 million in 1999. Daily marijuana use by high school seniors dropped 500 percent: from 10.7 percent in 1979 to 1.9 percent in 1992, and rose to 6.0 in 1998. Alcohol-related traffic deaths have been reduced from 26,000 to 15,935 per year.

- Table 1 shows drug use among young adults (18–25), high school seniors, and adolescents (12–17) in the peak year (1979), the lowest year (1992), and in 1999.

SUE RUSCHE

Community Drug Resistance The fear and anger fueled by COCAINE use had already breathed new life into the community anticrime movement of the 1970s. Social scientists and policymakers had concluded that community anticrime programs were unlikely to arise spontaneously in poor, crime-ridden neighborhoods where they were most needed—and, indeed, could not even be implemented successfully by professional organizers (Rosenbaum, 1986). But research on citizen antidrug programs has found that, actually, they are most likely to arise in poor neighborhoods, where drug activity is most common (Davis et al., 1991).

TYPES OF COMMUNITY ANTIDRUG PROGRAMS

Weingart (1992) proposed a typology for understanding citizen antidrug organizations. He defines four types of programs: (1) Law-enforcement enhancement, (2) civil justice, (3) treatment and prevention, and (4) community building. Weingart argues that community antidrug efforts are overwhelmingly dominated by the first category of program—those that aim to complement the activities of law-enforcement agencies.

Law-Enforcement Enhancement. Block-watch programs train participants to observe drug activity from their homes and to report it—usually to a designated member of the block-watch organization—in as much detail as possible (descriptions of suspects, locations of drug caches, license numbers of buyers). That person relays the information periodically to a designated police-liaison officer and, in return, the police-liaison officer reports back to the organization on the form of action taken as a result of the complaints.

Citizen patrols are commonly used programs that enhance law-enforcement efforts. Patrols vary in the degree of confrontation they use with drug dealers. Some simply observe and call their base or the police when drug sales are spotted; others have gone as far as obviously photographing or otherwise harassing drug dealers. Experience suggests that just a few individuals patrolling can be effective in removing drug activity from a neighborhood, although it has proven difficult to maintain residents' commitments to participation over extended periods of time.

Civil-Justice Efforts. These involve bringing suits against drug dealers in civil court for actionable nuisances (the noise and violence that accompany drug activity) and bringing suits against property owners, demanding abatement of a nuisance (drug selling) at a particular location. These are by far the most common type of civil action, and many major cities have made enforcement of nuisance-abatement laws a priority of the city attorney's office. Civil actions against property owners seem to be highly effective in abating drug sales at the targeted location, usually through eviction of drug sellers (and other occupants of their apartments). Questions remain about whether these actions simply displace the problem to another locale and whether they violate the rights of property owners and (at times) innocent tenants (Smith, Davis, & Hillenbrand, 1992).

Treatment and Prevention Programs. Such programs often rely on the voluntary efforts of drug abusers, their families, and their neighbors to help one another. Community-treatment programs range in size from those that are part of national organizations-like COCAINE ANONYMOUS and NARCOTICS ANONYMOUS—to small grass-roots programs, which are often church affiliated. Local drug-education efforts are usually citywide rather than neighborhood based, and they often work through the schools. Other prevention programs offer neighborhood youths supervised recreational activities for self enhancement and as an alternative to the drug culture.

Operation Weed and Seed is a community based program designed to combat drugs, violence and gang activity in high-crime neighborhoods. First launched by the Department of Justice in 1991, the

program is administered by the Executive Office for Weed and Seed within the DOJ's Office of Justice Programs. It combines law enforcement efforts to target, apprehend and incapacitate violent street criminals (weed) with community policing, prevention and treatment, and neighborhood revitalization efforts (seed). Weed and Seed grew from three grant sites in 1991 to 200 sites across the United States in 1999. Approved sites receive an annual stipend which ranges from $35,000 to $500,000. In June, 1999, The National Institute of Justice published an impact evaluation of Operation Weed and Seed. The evaluation reported that the effectiveness of weeding and seeding varied across the eight sites surveyed. The most successful sites featured established community-based organizations, active community leaders and programs that concentrated on smaller population groups. Several evaluation sites encountered early community resistance to the program because residents were concerned about aggressive enforcement measures and targeted harassment. The 1999 report emphasized the importance of involving local residents early in the process to encourage interaction and trust between law enforcement personnel and community members.

Community Building. Finally, some community-building efforts try to unite local residents against drugs through vigils, rallies, and marches. These kinds of activities are common in major U.S. cities. Other community groups have fought back against drugs by eliminating signs of disorder—such as uncollected trash and graffiti; by enhancing the appearance and safety of the neighborhood—installing better street lighting or clearing refuse and planting flowers; or by demanding that local officials raze the abandoned buildings and clear the refuse-filled empty lots used by drug abusers and drug dealers.

LINKS TO POLICE ORGANIZATIONS

Some community antidrug efforts (most notably Black Muslim patrols in Washington, D.C., in Brooklyn, N.Y., and elsewhere) have been mounted without the involvement of the police. Such efforts are relatively rare, however. In the vast majority of programs, potential activists have found a willing ally in the police. At the time that citizens were attempting to organize against drugs, many local police departments were in the process of undergoing a conversion to a community-oriented approach to law enforcement, which invited citizen participation (Skogan, 1990).

Davis et al. (1991) report that the police played a critical role in the maintenance, and sometimes the formation, of community antidrug programs. Although police administrators are normally the first to state that they cannot mobilize a neighborhood against drugs, they often facilitate incipient organizations by donating space and speakers for meetings, by acting as advocates with other city agencies (sanitation, building inspection, etc.), by providing training and backup for patrols, and by bringing together leaders from different neighborhoods to cross-pollinate ideas.

RISKS OF VIGILANTISM

The same war on drugs that promotes the vigorous enforcement and prosecution of drug cases may also have adverse consequences, including the erosion of personal freedoms and the promotion of vigilantism. Since about 1980, the Supreme Court has responded to public outcries for tougher action against drug dealers by upholding cases that permit broader latitude in surveillance and search activities. Furthermore, very aggressive citizen efforts (such as the Black Muslim patrols and the Guardian Angels) have been criticized for harassment and violent assaults against drug dealers.

PROGRAM EVALUATION

Because community antidrug programs are new, the media have been the main source of information about their activities; however, a handful of studies have been undertaken to explore the implementation and impact of community antidrug programs. Davis et al. (1991) examined the kinds of communities that have spawned antidrug efforts and the effects the programs have had on residents' perceptions of crime and disorder. Contrary to extant theories of community organizing—which suggest that resident programs against crime can only be mounted successfully in middle-class areas—the investigators found that antidrug initiatives were more common in low-income neighborhoods, even after taking into account the fact that such neighborhoods had more drug activity. In addition, the study looked closely at four of these initiatives. Residents in neighborhoods served by

the programs reported lower fear of crime and greater neighborhood satisfaction than residents of comparable nearby areas without programs.

Rosenbaum and his colleagues studied the initiation of a national demonstration program called Community Responses to Drug Abuse (CRDA). Using federal funds, ten communities in nine U.S. cities implemented a variety of antidrug projects, including closing drug houses and creating drug-free zones. The researchers interviewed participants, observed program activities, and analyzed records at the ten sites. Rosenbaum et al. (1992) report that the local community organizations accomplished a great deal with limited funds. A crucial lesson learned by the organizations was that enforcement activities provide only a limited solution to the drug problem. The most effective strategies involved broader partnerships with other agencies and institutions, such as churches and schools.

Finally, the federal Community Partnership Demonstration Program, funded by the Office for Substance Abuse Prevention, provides assistance to more than 250 programs for the prevention of substance abuse and now the CENTER FOR SUBSTANCE ABUSE PREVENTION (CSAP) allows local organizations considerable discretion to shape their own initiatives in combating drug and alcohol abuse.

A 48-community study of the Community Partnership Program, released in December, 1999 by CSAP, reported a statistically significant reduction in drug and alcohol use among males in partnership-communities. The study selected a representative sample of 24 communities from the 251 funded by CSAP and compared substance abuse rates to 24 non-partnership communities over a period of 18 months. The researchers collected surveys from 83,473 randomly selected adults, 10th graders, and 8th graders in the 48 communities. The study found that community partnerships can be effective in decreasing alcohol and illicit drug use in males, but were less effective in decreasing alcohol and illicit drug use among females. The study also found that adults reporting less illicit drug use were more likely to live in a partnership community, be involved in substance abuse prevention activities, and live in a neighborhood perceived to have minimal illicit drug trading.

(SEE ALSO: *Crime and Drugs; Education and Prevention; Gangs and Drugs; Prevention Movement*)

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Prevention of Alcoholism: The Ledermann Model of Consumption The Ledermann model of alcohol consumption is an important concept for anyone who wishes to understand the underpinnings of modern policy efforts to prevent heavy drinking and alcoholism. The point of departure for this concept is a set of observations about how alcohol consumption is distributed in human societies.

Many have thought of this distribution as occurring in two parts. First, there is the great mass of "normal" drinkers; their drinking might be plotted as a bell-shaped curve, with a few people drinking no more than a sip in a year, an increasing number drinking greater amounts than a sip but less than the average amount, and then a declining number drinking more than the average amount, until the graph reaches the normal drinkers who drink much more than the average—and these are relatively few in number. Second, there is a much smaller number of "abnormal" drinkers; their drinking distribution also might be plotted as a bell-shaped curve, but this curve is shifted to the right of the distribution for normal drinkers. Figure 1 shows this two-distribution concept of normal and abnormal drinking, with the number of drinkers on the y axis and the amount consumed on the x axis.

Sully Ledermann, a French demographer, thought of this problem in relation to a single distribution that was not bell shaped or normal in its distribution. He imagined that drinking ought to be plotted in relation to a single curve, with a shape that is known as "lognormal" and without a categorical distinction between normal and abnormal drinkers. The shape is known as lognormal because the natural logarithms of individual consumption, rather than actual consumption values, are normally distributed. Assuming Ledermann is correct, the majority of individuals within a society will drink relatively modest amount of alcohol, and a small proportion will drink large quantities, but this will appear in an asymmetric or "skewed" distribution curve with a longer tail to the right of the average alcohol-consumption level (see Figure 2). To the right of the curve there should be no bump, which would be caused by the presence of an abnormal-drinkers category, distinct from the category of normal drinkers.

Perhaps the most important implication of Ledermann's thinking about alcohol consumption has to do with the prevention and the reduction of heavy drinking. Categorical distinctions between normal and abnormal drinkers make it possible to focus prevention and intervention efforts on the abnormal drinkers. In contrast, the Ledermann model suggests that efforts can be focused on the great mass of people who drink modestly as well as on the heavier drinkers: In so doing, reductions in the average amount of alcohol consumed should also result in significant reductions in the proportion of people who are very heavy drinkers. This difference in approach is part of an important ongoing debate about how societies can best organize to reduce the hazards of alcohol use.

BACKGROUND

Ledermann (1915–1967) first proposed his single-distribution hypothesis in a French publication entitled, *Alcool, Alcoolisme, Alcoolisation* (1956). In a second report published in 1964, he attempted to test and confirm the validity of his theory by using empirical data on drinking behavior from multiple studies. Born in Algeria, Ledermann spent most of his career in Paris, at the National Institute of Demographic Studies (INED) and the University of Paris. A prolific researcher, his interest in the distribution of alcohol consumption within societies developed out of a broader effort to identify the reasons for the lower average longevity of the people in France, in comparison to that of the people in other European countries. Increasingly, he came to believe that a close connection existed between the average, or per capita, level of alcohol consumption within a society and the prevalence of excessive drinkers at risk for alcohol-related injury or death, and that this relationship could be described mathematically.

Ledermann argued that the lognormal distribution of alcohol consumption resulted from the tendency of individuals to develop and change their drinking habits according to a "boule de neige" (snowball) mechanism driven by social pressures. The Norwegian scientist Ole-Jorgen Skog noted that, in general, lognormal distributions tended to result from the exponential (multiplicative) combination of behaviors (1985). On an individual level, this means that persons will tend to increase or decrease their frequency of a behavior by an amount proportional to the initial frequency with which they perform it. For example, we might expect that a person currently consuming 30 liters of

alcohol per year would perceive an increase of 6 liters as being comparable to an increase of 1 liter by an individual who currently consumes 5 liters. Such phenomena grow exponentially, in snowball fashion, and tend to distribute according to a lognormal function within populations. Ledermann believed that the snowball effect was caused by the operation of social pressures within drinking environments. This notion implies that the drinking behaviors of individuals within a particular social environment or "drinking culture" are tightly interrelated, such that changes in the alcohol consumption level of some individuals are very likely to induce changes in the consumption level of others. Skog and other scholars have elaborated upon this rudimentary social-interaction hypothesis in an effort to understand how shifts in the drinking habits of one sector may rapidly diffuse throughout the entire population.

The Ledermann model provides a simple formula for estimating the distribution of alcohol use in any homogenous population of drinkers (that is, any population in which the average consumption level does not vary significantly across subgroups). In addition to assuming lognormality with his model, Ledermann also hypothesized that the proportion of drinkers consuming more than 365 liters of absolute alcohol (ethanol) annually was small and invariant across populations, because such high consumption levels (1 liter per day) would quickly have lethal effects. With this constant determined, he could establish mathematically the full distribution of alcohol consumption within a population, knowing only the per capita or average consumption level. Knowledge of the distribution of alcohol consumption yields three important additional insights. First, one can estimate the proportion of heavy or excessive alcohol users in the population. This value is frequently defined as the percentage of drinkers consuming 10 centiliters or more of absolute alcohol per day. Second, the total amount of alcohol consumed by heavy users can be estimated. Third, and most important, the effect of changes in average consumption on the proportion of excessive drinkers in the population can be predicted. This final corollary of the model is perhaps the most controversial, because it indicates that the prevalence of excessive alcohol use within a society can be manipulated by restrictions on alcohol availability or other preventive efforts designed to reduce the general level of consumption in the population. The implications of the Ledermann model for alcohol-control policy and other public health efforts were carefully elucidated in a monograph by Finnish scholar Kettil Bruun and an international body of colleagues (1975).

Ledermann's hypotheses have been the object of intense scrutiny and debate in the half century since they were first proposed. Many researchers have examined the "fit" between the lognormal distribution and data obtained from actual populations of drinkers, with mixed results. Significant deviations from expectations of the model have been demonstrated in some cases; in other populations, the distribution closely approximated lognormality. Ledermann's assumption of constancy across populations of the proportion of heavy drinkers who consume 365 liters or more of alcohol annually has been severely challenged. In general, these critiques have weakened the deterministic character of Ledermann's original formulation, without challenging the basic assertion that there is a close connection between average alcohol consumption in the population and the prevalence of excessive or "at risk" drinkers. The debate over these issues is unresolved, but it is clear that Ledermann's ideas have served as a major stimulus in the effort to understand the relationship between the "drinking culture" of a society and the prevalence of excessive alcohol use.

Ledermann's thinking directly or indirectly underlies many current alcohol policies, especially those that control where, when, and how alcohol is consumed, and how much we pay for it. However, in the half century since his single distribution theory was first proposed, alcohol problem prevention research has continued to grow in sophistication, and modern efforts reflect a greater appreciation of the complexity of societal drinking patterns (Holder et al., 1999; Toomey and Wagenaar, 1999). The assumption of societal homogeneity in drinking behavior was a major tenet of Ledermann's first conceptualization of the single distribution theory. We now have a much greater understanding of the magnitude and significance of variation in drinking behavior, both within and between societies, based on age, gender, ethnicity, locale, and other aspects of culture (Holder and Reynolds, 1998). In addition to level of consumption, alcohol problem prevention efforts also focus on the pattern of drinking and the physical environments where alcohol is consumed. Particular at-

tention in both alcohol and drug abuse prevention studies has centered on such "harm reduction" efforts. This approach focuses on the promotion of safer use patterns rather than limitations on availability (Giesbrecht, 1999; Mosher, 1999). Alcohol server intervention programs, other alcohol education efforts, and early problem identification and intervention programs are examples of this targeted prevention approach. Ledermann's stature and influence in the field of alcohol problem prevention research are still marked, but modern alcohol problem prevention efforts are highly diverse and include a mix of individual and group-based strategies, recognizing that some approaches are appropriately directed at the societal level, but special populations and settings may require focused, specific efforts.

(SEE ALSO: *Addiction: Concepts and Definitions; Advertising and the Alcohol Industry; Alcohol: History of Drinking: Disease Concept of Alcoholism and Drug Abuse; Legal Regulation of Drugs and Alcohol; Prevention; Social Costs of Alcohol and Drug Abuse*)

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PREVENTION PROGRAM DIRECTORY

See Appendix, Volume 4.

PREVENTION PROGRAMS Prior to the 1980s, most schools around the country had courses in health education, tobacco education, alcohol education, or drug education. In these courses, students typically were taught that using tobacco, alcohol, MARIJUANA, or other drugs was bad for their health, and they may have found out how or why they were dangerous. Sometimes students were given detailed information about how these substances affected the body, how long the effects lasted, and even how people used them. Many tobacco-, alcohol-, and drug-education programs had tried deliberately to scare students by pointing out how many people die each year from drug abuse. It was widely believed that if students really knew how harmful smoking, drinking, or using drugs is, they would not do it. However, numerous studies found that teaching facts or using scare tactics often does not work. Therefore, programs designed to go beyond merely providing students with facts about the harmful effects of using drugs were implemented.

Many prevention programs did not work because they did not deal with the real causes of drug abuse (U.S. Public Health Service, 1986). Although we still need to learn more about what leads to drug abuse and how it develops, much is already known. This knowledge about the causes of drug abuse and the theories that researchers have developed to explain it provide the foundation upon which successful prevention programs are based. At this point, most drug-prevention experts agree that drug abuse does not have a single cause. Many different factors cause individuals to first try one or more drugs, and then they gradually become both

physically and psychologically dependent on them (Schinke, Botvin, & Orlandi, 1991).

Most people start using drugs during their early teenage years or slightly before (Schinke, Botvin, & Orlandi, 1991). This is the time when they are experimenting with a wide range of behaviors and life-style patterns as part of the natural process of growing up, becoming more independent, and discovering their own identity. Contrary to what some adults might think, more than half of all adolescents try one or more of these substances. Most individuals who try drugs do not use them more than a few times, but those who do run the very real risk of developing a compulsive pattern of use characterized by increases in both the frequency and amount of drug use and possibly development of drug dependence.

Many specific programs have been and are being carried out in schools and communities throughout the United States in the continuing effort to prevent experimentation with and use of alcohol and drugs. Included here is information on *"Here's looking at You"; Life Skills Training; Napa Project, Revisited; Ombudsman Program; PRIDE; Project SMART; Talking with Your Students about Alcohol; and the Waterloo Smoking Prevention Project.*

The overview entries on the *Parents Movement* and the *Prevention Movement* and the individual articles in the section entitled *Prevention* provide a framework for the articles in this section. For an extensive listing of other organizations engaged in similar efforts and of other programs now being used, see also *Education and Prevention* and the directory in the Appendix, Volume 4.

Prevention programs have undergone many physical changes throughout the years, from lecture-based to participation-based and from scare tactics to skill development, yet the programs do not always have positive effects. However, the outlook was becoming brighter in the late 1990s. According to Steven Schinke and Gilbert Botvin, writing in *Contemporary Pediatrics*, "Adolescents' penchant for risky behavior is no longer an impenetrable mystery, and there is now a body of scientific research on how attitudes and behavior can be changed. That research is beginning to pay off, yielding solid and empirically tested programs for preventing problems with tobacco, alcohol, and drugs among youth."

An early 1990s study evaluated the content and teaching strategies of ten school-based prevention programs: D.A.R.E., Health Skills for Living, Project Alert, Here's Looking at You 2000, Project I-STAR, Life Skills Training, Stanford Decide, QUEST, That's Life, and Teenage Health Teaching Modules. It was found that programs were becoming more similar, yet more training was necessary for the teachers.

Further studies are being done on some of these programs to see if they are still producing the desired results. For example, Drug Abuse Resistance Education (D.A.R.E.), an elementary and junior high school program, is the most widely-used prevention program in the nation (as of March 2000). Between 1999-2001, students receiving the curriculum and students receiving an addition of peer, parent, and community involvement will be evaluated and compared in the Minnesota D.A.R.E. PLUS Project.

"HERE'S LOOKING AT YOU"

The "Here's Looking at You" (HLAY) program grew out of the work done by Clay Roberts and Douglas Goodlett for their master's degrees in the 1970s. By 1978, Mr. Roberts and other health-education specialists at Seattle's Educational Service District No. 121 (ESD-121) had created a full alcohol-education curriculum for kindergarten through twelfth grades, designed mainly for delivery in fifteen to twenty class presentations each year, complete with multimedia support materials.

From the beginning, HLAY has been based on an educational theory involving both cognitive and affective elements: knowledge (information), attitudes, self-esteem, decision-making skills, and other social skills (Mooney et al., 1979). In subsequent versions of the program (HLAY-2 and HLAY-2000, with updates of the latter), strenuous efforts have been made to improve the educational strategy in light of ongoing psychosocial research and program evaluations. The program components fall into three basic categories: information, social skills, and "bonding." A two-pronged "inoculation" strategy—stressing both "risk factors" and "protective factors"—runs through these three categories. In both design and delivery, HLAY is one of the most thorough and sophisticated school-based programs in the United States, as well as one of the most widely used.

The underlying theoretical basis that has evolved for this program is recognizable by social

scientists as combining elements of both "rational choice" and "control" theories. In layman's terms, the program rests on the assumption that schoolchildren will be far less likely to use alcohol or other drugs if they are (1) given full and reliable information about the properties of chemical substances and the consequences of using them; (2) trained in self-control, decision making, and other social skills (including refusal); and (3) assisted in feeling positive about themselves and in bonding with friends, families, schools, and communities. Many of these outcomes would obviously be desirable in other arenas of youth and health as well.

One evaluation of HLAY-2000 has measured positive program impact on reported actual *use* of alcohol or other drugs (DuBois et al., 1989). This evaluation, covering grades 1 to 6, found evidence of positive impact on knowledge, self-esteem, and refusal skills but no evidence of impact on actual substance *use*, except in the case of chewing tobacco in grades 1 to 3. Other unpublished evaluations of HLAY-2000 (Bubl, 1988; Barrett, 1989) have not measured program impact on actual use of drugs or alcohol but have shown some evidence of impact at various grade levels on knowledge, self-esteem, coping, decision-making and refusal skills, and making friends.

LIFE SKILLS TRAINING

Toward the end of the 1970s, an approach to drug-abuse prevention called Life Skills Training (LST) was initiated. This approach differed from the type of information programs conducted by many schools until that time. Instead of students being taught a collection of facts about drugs and the dangers of using them, they were taught general skills for living happier and healthier lives. Studies testing the LST approach have been conducted since 1980, and they provide evidence that teaching life skills can help adolescents avoid becoming involved with drugs.

The main objectives of the LST program are: (1) to provide students with the information and skills they need to resist social pressures to use drugs; (2) to decrease potential motivations for using drugs by helping students develop greater autonomy, self-esteem, self-mastery, and self-confidence; (3) to enable students to cope effectively with anxiety, particularly anxiety induced by social situations; (4) to increase students' knowledge of the immediate negative consequences of drug use and provide them with accurate information concerning the prevalence rates of tobacco, alcohol, and marijuana use; and (5) to promote the development of attitudes and beliefs supportive of a lifestyle that excludes drug use.

The LST curriculum is a three-year program. It consists of fifteen class periods during the first year, ten booster sessions in the second year, and five booster sessions in the third year. The booster sessions, which are intended to reinforce the material taught in the first year of the program, focus on the demonstration and practice of the life skills that form the foundation of this prevention approach. The LST program contains the following five components, each of which consists of two to six sessions: Knowledge and Information (Four Sessions); Decision Making and Independent Thinking (Four Sessions); Self-directed Behavior Change (Two Sessions); Coping with Anxiety (Two Sessions) and Social Skills (Six Sessions).

During the 1980s, LST was tested by Botvin and his colleagues in eight separate studies that involved more than 25,000 students from over 150 schools in New York and New Jersey. Most of the studies focused on cigarette smoking, but several also examined the impact of LST on alcohol and marijuana use. The LST approach typically produced reductions of 50 percent to 80 percent in new smoking, drinking, and marijuana use after the first year of the program (Botvin & Tortu, 1988), but booster sessions appear to be necessary to maintain these initial prevention effects. Studies have demonstrated that the LST program can be effectively implemented by adult providers and peer leaders. Not surprisingly, it was found that the effectiveness of the LST program was related to how thoroughly it was implemented. Students whose teachers conducted the program carefully and completely demonstrated lower rates of drug use than did students whose teachers either deviated from the program or taught only part of it.

Research is currently under way to determine the extent to which the LST approach is effective in reducing risk for HIV infection. Studies are also being conducted to investigate the long-term effectiveness of this type of prevention strategy with tobacco, alcohol, and marijuana, as well as to determine the extent to which it is effective with other illicit drugs. In May 1999, students who had been through the LST program had approximately 50

percent lower incidences of drug abuse than students who had not been through any program.

NAPA PROJECT, REVISITED

The Napa Project was designed to demonstrate the promise of school-based affective and alternatives programs. It was oriented toward exemplary strategies for elementary and junior high school students because interventions in senior high seemed to be too late. The hope was to see each strategy implemented in high-quality fashion and in fertile circumstances, for periods measured in semesters and years, not days or weeks. The goal was to assess the strategies' effects on a range of student outcomes. Further, in addition to implementing and evaluating the strategies individually, there was reason to assess them in several combinations and sequences, in recognition that significant effects might not be attainable with any one strategy alone.

The Napa Project was conducted between 1978 and 1983, in close collaboration with the Napa Unified School District in northern California. All the studies were done in the Napa schools, which served a largely white, middle- and working-class community on the periphery of the San Francisco Bay area. The intervention and evaluation costs of the project were supported by a large, multiyear grant from the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA).

Underlying the selection of seven total prevention strategies for the Napa Project was a theoretical model linking them to improvements in classroom and school environments, and then to positive changes in students' competencies, values, and attitudes (see Figure 1). Derived from the work of the Jessors (Jessor & Jessor, 1975) and Fishbein (Ajzen & Fishbein, 1977; Schlegel et al., 1977), the causal model held that as students' satisfaction with self, peers, and school increased, and as they perceived their peers to have more positive attitudes toward school, their own attitudes toward drug use would become less accepting and they would perceive the norms of their peers to be similarly antidrug. This was intended to decrease both intentions to use drugs and actual drug use.

The four strategies and the grade levels at which they were implemented are listed below.

- Magic Circle—teachers were trained to lead structured class meetings designed to build a sense of connection and community, as well as to foster social and academic development (grades 3–4).
- Effective Classroom Management (ECM)-Elementary—teachers were taught communication skills, discipline techniques, and self-concept enhancement techniques for use throughout the school day (grades 4–6).
- Effective Classroom Management (ECM)-Junior High—communication, discipline, and self-concept enhancement skills were adapted for teaching in the junior high environment (grades 7–9).
- Jigsaw—teachers were taught to organize classrooms into cooperative learning groups of five or six students, in which each student was given the responsibility of teaching an essential piece of the regular curriculum to the other group members (grades 4–6).

Two alternatives strategies were offered as elective academic courses to junior high students. In these courses, students were taught skills and provided with opportunities for helping peers or younger children. The courses did not address the topic of drug use; instead, they sought to teach social competencies and to enhance self-esteem. The alternatives strategies were the following:

- Cross-age tutoring—students regularly tutored younger children in reading or other academic subjects (grades 8–9).
- Operating a school store—students ran a school store on their campus, selling school supplies and snacks, while learning relevant business skills in a related academic course (grades 8–9).

The final strategy was a drug education course that taught social competencies and drug information to seventh graders. In the course, students were taught Maslow's (1980) framework for understanding motivation; learned a systematic decision-making process; analyzed techniques used in commercial advertising; learned assertiveness skills for dealing with peer pressure; and practiced setting personal goals. Toward the end of the course, students were provided with information about tobacco, alcohol, and marijuana, in response to their

written questions. They also applied the social skills in considering drug-use issues.

The seven strategies were evaluated individually and in certain combinations in twelve separate studies. All studies assessed the implementation of the strategies, as well as their effects on students. Process and outcome evaluations were conducted by the project's full-time four-person research staff. General information about the studies can be found in Schaps et al. (1984), which lists twenty-eight publications describing the various studies.

None of the strategies was shown to be effective. The four in-service strategies and the two alternatives strategies had no systematic effects on students' perceptions of classroom climate; attitudes toward self, peers, or school; attendance; academic achievement; perceptions of peer group norms; or drug-related attitudes, intentions, or behaviors. Moreover, this lack of effects could not be readily explained by poor implementation of the strategies. Implementation of the alternatives strategies was generally satisfactory. Although implementation of the in-service strategies did vary greatly from teacher to teacher, and was found to be inadequate in many classrooms, no effects were found even for the subgroups of students had who the greatest exposure to the strategies, or who were in classrooms where the strategies were best implemented, or who received combinations of strategies over two or three years.

Nevertheless, the failings of Napa's strategies and theory may well have been inadequacies of scope and depth, not of direction. That is, Napa may well have been on a potentially fruitful track in seeking to promote socially constructive norms, attitudes, and competencies—and reduce substance abuse—by fundamentally altering students' experience of schooling. But those who designed Napa may have grossly underestimated the scope and substance of the needed changes, and also the resources and processes needed to enact those changes. Even the classrooms in which the best implementation of Napa's strategies was observed may not have differed much from "ordinary" ones.

In providing twelve workshops over a period of several months, supplemented by one or two individualized consultations with a trainer, Napa offered teachers more support than most prevention programs of its time. However, it is recognized that to change in meaningful ways, most teachers need several years of focused staff development; regular opportunities for planning, reflection, and problem solving with peers; congruent instructional and curricular materials; encouragement from school and central office administrators; supportive assessment practices; and protection from conflicting demands for change.

NATIONAL FAMILIES IN ACTION

In November 1977 a group of concerned citizens in Atlanta, Georgia, troubled by the emergence of commercial and environmental pressures that seemed to encourage people to use addictive drugs, formed National Families in Action (NFIA). These commercial and environmental pressures coincided with an escalation in drug use among children and young adults to the highest levels in the history of the world. The organization's founders—parents, doctors, law-enforcement officials, political leaders, educators, business leaders, and others—sought to replace the glamorization of drug use with accurate, reliable information based on scientific research about drug effects.

Initially, National Families in Action targeted the drug PARAPHERNALIA industry. If drugs such as MARIJUANA and COCAINE were illegal, the group reasoned, it made no sense to allow the sale of implements to enhance their use. Three months after its founding, National Families in Action got the Georgia legislature to pass the nation's first laws prohibiting the sale of drug paraphernalia. Publicity surrounding this event brought calls from people across the United States who wanted to organize similar groups to ban drug-paraphernalia sales in their communities. They also wanted to educate their families and communities about the harmful effects of drugs, to prevent drug use before it started, to help users stop, and to find treatment for those who couldn't stop using drugs. The organization published a manual, *How to Form a Families in Action Group in Your Community*, which helped many thousands of groups organize. In addition, members traveled throughout the United States to help families organize community-based, substance abuse prevention groups.

National Families in Action established a drug information center, collecting articles from medical and scientific journals about all aspects of substance abuse, including research about drug effects, prevention of use, intervention, and treatment. It also collects articles from newspapers and maga-

zines about drug policy and the emergence and growth of the grass-roots prevention movement in the United States (and, increasingly, abroad). In addition, the collection houses publications of the drug paraphernalia industry and organizations that advocate drug legalization. National Families in Action's drug information center contains more than 500,000 documents on substance abuse. The center answers questions from people throughout the world who call or write for information.

In 1982, National Families in Action began publishing *Drug Abuse Update*, a quarterly digest containing abstracts of articles collected at the center. In 1990, the organization introduced *Drug Abuse Update for Kids*, written for children in elementary and middle schools. It publishes other drug-education materials as well, including a curriculum about drugs and the brain titled *You Have the Right to Know*. From 1984 to 1990, National Families in Action's executive director, Sue Rusche, wrote a twice-weekly column on substance abuse that was syndicated by King Features to more than 100 newspapers throughout the nation.

In 1990, National Families in Action received a demonstration grant from the CENTER FOR SUBSTANCE ABUSE PREVENTION to help families who lived in two Atlanta public housing developments prevent substance abuse in their communities. Called Inner City Families in Action, this project was named one of eleven exemplary programs in the United States in 1993. The program trains parents to teach *You Have the Right to Know* to neighbors, friends, and children. It helps parents obtain needed skills to complete their education and enter the work force. It also helps parents form Families in Action groups to seek treatment for loved ones who are addicted to drugs, to engage children in productive activities, and to prevent substance abuse in their communities.

National Families in Action recently agreed to help support International Students in Action (ISIA), which was founded in 1999. ISIA's board members include students from educational institutions like the University of California, Berkeley, and Harvard, as well as international students from the United Kingdom and other countries. The group's goals include involving students in the drug prevention dialogue and creating a drug-education curriculum on campuses all over the world.

National Families in Action also developed an after-school program, Club HERO (Helping Everyone Reach Out), which provides a positive environment for youths and rewards them for school performance and good behavior. NFIA introduced the *You Have A Right to Know* course into the program, and gives youths the chance to listen to and interact with local community role models.

NFIA has always been a leader in the fight against the drug-legalization movement. They stepped up these efforts in 1999 when they joined two other organizations in condemning a reality-based drug education program that teaches children that it is possible to have "positive relationships" with marijuana, cocaine, and other illicit drugs. A proposed conference in October 1999 called *Just Say Know: New Directions in Drug Education*, sponsored by The Lindesmith Center-West, aimed to instruct parents and students that drug use among some kids is inevitable and something that should not be stopped or prevented. The director of The Lindesmith Center-West suggested that "successful" drug users be sent into classrooms to serve as good examples for children.

Sue Rusche, NFIA executive director, condemned the conference and said its major goal was to use children as pawns in the drug-legalization crusade. "In the 1970s," she remarked, "this approach to drug education helped drive adolescent drug use to the highest levels in history."

The Lindesmith Center and its supporters believe that the "Just Say No" policies of the 1980s have not worked and a new approach to drug education is necessary. Drug prevention advocates such as NFIA believe that parents who teach and discipline their children can make a difference. They suggest keeping a continuous dialogue with kids, setting limits, and enforcing consequences if rules are broken. Recent data has suggests that kids whose parent instruct them about the dangers of drug use are 36 percent less likely to use marijuana and 56 percent less likely to use cocaine (Office of National Drug Policy, 1998). NFIA continues to fight numerous organization and movements whose major goal is the legalization of drugs or reality-based drug education.

Throughout its history, National Families in Action has developed numerous networks and national coalitions to advance the field of substance abuse prevention. These include the Prevention, Intervention and Treatment Coalition for Health (PITCH), an association of community-based prevention organizations that serve many different

ethnic and cultural groups throughout the nation. Through its advocacy efforts, PITCH helped bring about the creation of a new federal agency, the Substance Abuse and Mental Health Services Administration, to further develop the prevention field. National Families in Action is increasingly called upon to help citizens from other nations develop prevention groups.

Along with other national prevention organizations, National Families in Action has played a pivotal role in driving drug use down since 1979.

NATIONAL FEDERATION OF PARENTS FOR DRUG-FREE YOUTH/NATIONAL FAMILY PARTNERSHIP (NFP)

A large number of parent-group leaders, who had previously organized drug-prevention groups in their states and local communities, formed the National Federation of Parents for Drug-Free Youth in the spring of 1979. With the assistance of national organizations in Atlanta, Massachusetts, and elsewhere, these leaders had organized prevention groups of parents in response to the greatest escalation in drug use by American adolescents in the history of the world. They organized to protect children by striving to prevent drug use before it began, by helping young drug users to stop, and by obtaining treatment for those who couldn't stop by themselves.

During the 1970s, legislatures in eleven states decriminalized MARIJUANA. During this same period, an explosion of head shops proliferated throughout the United States. These places, which sold PARAPHERNALIA to enhance drug use, targeted their products to children and teenagers. The national decriminalization discussion produced rhetoric that ignored or played down the harmful effects of drugs, and this rhetoric spilled over into drug-education materials, which counseled the "responsible use" of drugs that were both dangerous and illegal. Song lyrics and films in the adolescent culture tended to reinforce the popularity and acceptance of drug use. These factors appeared to contribute to, if not actually drive, the astonishing escalation in adolescent drug use throughout the 1970s.

Parent groups organized to prevent children from entering the drug culture and to rescue those who already had, taking aim at the drug-paraphernalia industry and fighting decriminalization. By 1979, however, it had become clear that action at local and state levels was not enough. Representation at the national level was critical particularly in light of the fact that a federal bill to decriminalize marijuana was gaining support from members of Congress. Parent-group leaders formed the National Federation of Parents for Drug-Free Youth to represent their interests in Washington.

The first order of business was to defeat the pending federal decriminalization bill, which would have removed criminal penalties for the possession of up to an ounce of marijuana. Federation volunteers bought 1-ounce jars of parsley to demonstrate that an ounce was not an insignificant amount, and to reinforce the fact that an ounce of marijuana can yield from forty to sixty "joints." They delivered these jars to each member of Congress, educating senators and representatives about the high levels of marijuana and other drug use that decriminalization in some states appeared to have produced among young people, and asking them to vote against the federal decriminalization bill. The Federation succeeded in this effort. Congress voted the bill down and rejected decriminalization for good.

Shortly afterward, the Federation led a letter-writing campaign to newly elected President Reagan, asking him to place leaders sympathetic to parent-groups' concerns in important drug-policy roles in his administration. In addition, the Federation brought parent-group leaders from communities across the United States to Washington to brief First Lady Nancy Reagan about their efforts. As a result, Mrs. Reagan became an informal spokesperson for the Federation and its work.

When Drug Enforcement Administration agent Enrique Camarena was brutally murdered while on duty in MEXICO, the Federation's Virginia chapter conceived a Red Ribbon campaign to honor the slain agent. The chapter wanted to express support for law-enforcement officers nationwide who put their lives on the line every day to enforce the nation's drug trafficking laws. The initial campaign developed into Red Ribbon Week, held annually each October. During this week, schools and communities across the nation celebrate the Red Ribbon campaign for drug-free communities.

In 1993, the Federation refocused its mission and scope, reincorporating under the name of National Family Partnership (NFP).

The NFP conducts training for parents, youth. and community leaders to help them organize prevention groups. Along with other national prevention organizations, the NFP has contributed to the reduction in drug use that has occurred since 1979.

OMBUDSMAN PROGRAM

Ombudsman is a word of Swedish origin that can be loosely translated as "a helping person." The Ombudsman program is a drug-abuse prevention program geared to students in grades five through nine. The program was developed by the Drug Education Center, located in Charlotte, North Carolina, and is based on the assumption that the most effective way to prevent adolescent alcohol and other drug (AOD) abuse is through the promotion of individual personal growth. This is attempted via enhancements of self-esteem, social skills, and the empowerment of students in a group project that seeks to help others. Students meet once or twice per week (depending on the course module chosen) during regular classroom hours. In addition, the program activities are designed to be integrated into academic subject areas. Either a trained facilitator or a classroom teacher who has been trained by a certified Ombudsman trainer directs the program.

The Ombudsman program was one of the first drug-abuse prevention programs in the United States to be funded by the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) in 1977. The purpose of the NIDA grant was to fully develop and evaluate the outcome of this program.

The program has three phases. The first, Self-Awareness, involves a series of exercises that encompass activities for building self-esteem. The purpose of these activities is to foster the development of self-worth and respect for others. The second phase, Group Skills, gives students an opportunity to foster communication, positive group interaction, and refusal/resistance decision-making skills. Information on the effects of drugs is taught in this phase. During the third and last phase, students apply the knowledge and skills they have gained in the program by planning and carrying out a project that helps others within their own community or at school. Ombudsman program activities are experiential, utilize cooperative learning techniques, and appeal to a variety of learning styles.

Ombudsman program outcomes have been evaluated by using the Student Attitudinal Inventory (Kim, 1981c). Short-term evaluation results indicate that the program can affect seven high-risk student attitudinal factors closely related to adolescent drug-using behavior: negative social attitude, rebelliousness, low valuing of school, poor student-teacher relationship, perception of incohesive family relationship, low self-esteem, and attitudes favoring drug use. It has also been learned that the program is more effective among younger than among older students. Finally, data from one long-term evaluation suggest that there is a greater proportion of students who no longer use drugs (i.e., who gave up experimenting with drugs) among the students trained in the Ombudsman program than among those who have not participated in the program.

PRIDE (NATIONAL PARENTS RESOURCE INSTITUTE FOR DRUG EDUCATION)

Thomas "Buddy" Gleaton, Ed.D., and Marsha Keith Manatt Schuchard, Ph.D., founded PRIDE in Atlanta, Georgia, in 1978. PRIDE's purpose is to help parents form groups to protect their children from becoming involved with MARIJUANA and other drugs. The organization is based on the following fundamental principles: (1) drug abuse is a health issue; (2) the family is the greatest bulwark against adolescent drug use; (3) families need help from the rest of the community to steer young people safely through the many temptations and dangers that confront them every day.

Initially, PRIDE based its group model on parent peer groups initiated by Dr. Schuchard and her family. A parent peer group encourages parents to get to know and link up with the parents of their children's friends. They establish social guidelines for their children to which they all agree to adhere, and they try to create positive alternatives for young people to prevent them from engaging in unhealthy and destructive behaviors during adolescence. Dr. Schuchard's handbook, *Parents, Peers and Pot*, published and distributed by the National Institute on Drug Abuse, outlines how to form parent peer groups.

PRIDE later expanded its parent peer-group model to include larger groups of parents who wanted to work for change throughout their com-

munities to prevent drug abuse among young people. The organization offers training to parents across the nation. PRIDE also added a youth component, training junior high and high school students and encouraging them to take a stand against drug use. In both cases, the essence of the PRIDE philosophy is to help parents and young people reverse adolescent peer pressure that encourages negative behaviors, and use it as a force to persuade young people to adopt positive behaviors.

The PRIDE Drug Use Survey has helped thousands of local school systems determine the extent of ALCOHOL, marijuana, and other drug use among students in elementary, middle, and high school. A large data base allows PRIDE to spot early trends in the rise or fall of various drugs used by students. A systemwide survey of Atlanta public school students in 1994 demonstrated a shocking correlation between drug use and possession of guns and other weapons. The more involved a student is with drugs, the more likely he or she is to possess a weapon. If this early indicator holds true for students in other school systems, it will provide even more reason to intensify efforts to prevent drug use among students, in order to free them from violence as well as drug abuse and addiction.

As the United States devoted more resources to developing the discipline of substance-abuse prevention, and as grass-roots organizations such as PRIDE and others increasingly demonstrated that PREVENTION reduces drug use and abuse, European and other nations became intensely interested in learning about the American prevention experience. PRIDE has done much to foster this interest, and the PRIDE conference increasingly draws participants from other nations to learn about American grass-roots prevention techniques and processes.

PROJECT SMART

Project SMART was started in 1981 in Los Angeles by Drs. C. Anderson Johnson, Brian R. Flay, William B. Hansen, and John W. Graham as a pioneering effort to scientifically test programs for preventing experimental and habitual use of multiple substances. Originally the name stood for Self-Management and Resistance Training, but since then, it has come to stand more generally for the programs created by this University of Southern California research team, and for the programs used in many of their projects.

It was the goal of Project SMART researchers to interrupt the usual pattern of experimentation and habituation by presenting innovative programs that provided students with skills for overcoming situations that might promote use. Project SMART provides instructions for classroom teachers on how to prevent experimentation with and regular use of alcohol, tobacco, marijuana, and other drugs. Originally, there were two sets of Project SMART materials. One set focused on teaching students self-management skills. The other set provided students with social pressure resistance skills for dealing with peer pressure to use substances as well as skills for avoiding pressure from television, movies, music, adults, and ADVERTISING that might make substance use attractive. Both sets of materials included information about the consequences of alcohol, tobacco, and marijuana use.

These two curricula represented two different ways of thinking about what causes young people to experiment with substances. Self-management training came from the idea that young people use drugs to help them handle the challenges of growing up. This approach was based on the hypothesis that young people who lack the ability to manage their lives may experiment with alcohol or drugs as an alternative to handling difficult situations. The goal was to increase the skills that are important to being successful so that substance use would not be seen as a practical alternative. The training was applied to making decisions, handling STRESS, improving self-esteem, and increasing a person's ability to set and achieve goals. Students were taught how to identify and manage their stress through relaxation training; how to increase their self-esteem through finding positive qualities about themselves and others; and how to make well-thought-out decisions by mastering a process for identifying problems, thinking of alternatives, and weighing consequences. They learned how to set and achieve goals through practicing personal goal setting.

Training in resistance skills came from the idea that experimentation with alcohol, tobacco, and other drugs was related to social pressure. It was hypothesized that young people had to deal with offers, threats, and dares to use drugs and that they experimented with alcohol and other drugs in order to fit in with their peer group. Thus young people who had the skills necessary for refusing offers in a

way that allowed them to still be accepted were thought to be able to avoid experimentation. In this program, students learned to identify different types of peer pressure to use drugs; they were then taught some simple but effective strategies for refusing offers and resisting pressure, such as saying "No, thanks." Students practiced these techniques in role plays. The program also informed students about the real rates of use among their peers, which were nearly always lower than what students originally expected.

Both programs included a session in which students were videotaped making a personal commitment to use the skills they had been taught in order to remain drug-free. Students who had been trained in self-management skills described what they would do instead of using drugs, while students who had received resistance training described how they would respond if someone offered them a drug. Finally, in both programs, admired and respected classmates were used as peer facilitators. The peer facilitators helped conduct both programs' small-group activities and were trained to demonstrate the skills that students needed to master.

In 1982 and 1983, the two programs were presented to two groups of students in the Los Angeles Unified School District (Hansen et al., 1988). A third group of students received no special Project SMART program. Students who participated in the project filled out surveys that asked them to report on their use of alcohol, tobacco, and marijuana. After being pretested, students were given the program in the seventh grade and then completed surveys in the eighth and ninth grades. The students who had not been given any special program also completed surveys at the same time. At the end of this project, it was found that students who had received training in how to resist pressure were less likely to use all three substances.

The program materials for Project SMART have continued to evolve. Numerous research projects have added variations to what students are taught, and the actual methods for teaching the resistance skills have been revised and refined as the people who created and delivered the programs learned more about how young people think and found better ways to get the message of the program across.

In 1985, Drs. William B. Hansen and John W. Graham started a new project that emphasized a new strategy for prevention termed normative education. This program component was designed to establish a social norm that was intolerant of alcohol, tobacco, and other drug use. Normative education was based on that part of the original Project SMART that gave young people feedback about the rates of substance use among their peer group. In addition, the program encouraged young people to discuss openly with their parents and their friends the appropriateness of drinking in a number of situations.

A test was conducted that compared students who had participated in the normative-education program with students who had participated in the program that taught resistance to peer pressure. The two groups had received their program in the seventh grade and results tallied in the eighth grade were analyzed and published (Hansen & Graham, 1991). The results showed that each group of students had benefited from the program it received. Students who were given normative education expected greater intolerance of alcohol and drug use among their peers than did the other group. Students who had been taught how to resist pressure were much more capable than the students of the other group of refusing when tested in a situation in which a fellow student pretended to offer them a can of beer. However, the students who had established conservative group norms were less likely to drink alcohol, get drunk (see Figure 1), develop problem behaviors in relation to alcohol, use marijuana (see Figure 2), and/or smoke tobacco.

Ultimately, Project SMART became a curriculum guide that included the best components of all the research projects that contributed to it. The program now consists of two parts: the basic program that is delivered to students in the first year of middle or junior high school and a booster program that is given the following year. Some Project SMART program materials have been given different names, such as Project STAR and Project I-STAR. Except for being based on the most up-to-date versions of the curriculum, these programs are identical to Project SMART. The success of the curriculum was reproduced in a large study conducted in the greater Kansas City area. In this region, students from schools that received the program exhibited reduced rates of alcohol, tobacco, and marijuana use compared to students from schools that received no special program (Pentz et al., 1989).

TALKING WITH YOUR STUDENTS ABOUT ALCOHOL (TWYSAA)

TWYSAA is concerned with influencing the students' drinking behavior not just in the present (during childhood and adolescence) but throughout their entire lives. The authors realized that, once children are no longer in school, their opportunities to receive in-depth education about alcohol would be very limited. TWYSAA teaches children how to estimate their own personal biological risk of developing ALCOHOLISM—based on their family history and individual physiological factors. Students also learn how factors such as age, gender, fatigue, illness, pregnancy, menstrual period, and medication must be considered in making drinking choices. Safety issues, such as DRUNK DRIVING are discussed, and information is given about alcohol's negative effects on the cognitive skills needed for success in school. Students are encouraged to honor the parental and religious values of their households as well as the legal prohibition (in the U.S.) against purchasing alcohol or drinking before age 21.

This curriculum has three levels available: Level One for grades five and six; Level Two for grades seven and eight; and Level Three for grades nine and ten. TWYSAA is designed to be presented once at each level, so students receive instruction every other year. The major goals of the program are to increase the number of students who choose to abstain from drinking ALCOHOL, to delay the age at which students begin to drink alcohol (if they do begin drinking), and to reduce high-risk drinking.

TWYSAA is part of a series of alcohol-education programs designed by the Prevention Research Institute in Lexington, Kentucky. The original program, Talking With Your Kids About Alcohol, was a ten-hour course for parents—aimed at educating them about how to make low-risk drinking choices for themselves and giving them guidance on how to teach their children about alcohol. TWYSAA was the second program developed for the series; it is meant to be used as a complement to the course for parents. Schools that wish to implement the TWYSAA curriculum are first required to make Talking With Your Kids About Alcohol available to parents.

The TWYSAA course is usually taught as a part of the school's health-education curriculum. Teachers prepare for course instruction by attending a three- or four-day training program. The Prevention Research Institute provides these training sessions at various locations; it will also bring the training program to a location if a sufficient number of teachers want to be trained. At the training session, teachers receive all the course materials including slides to use in classroom presentations, a detailed teacher's guide with lesson plans, and printed materials that may be reproduced for students.

An evaluation of the curriculum was conducted by the Prevention Research Institute over a three-year period in nine schools in Kentucky and Ohio. Drinking behaviors and attitudes were measured before students took the TWYSAA course, immediately after the course, then one and two years later. A group of students (a control group) who did not take the course were also studied for comparison. The findings from this study indicated that TWYSAA had successfully achieved each of its major goals. Compared to students who had not taken the course, more of the TWYSAA students chose to abstain from alcohol; those who began drinking started later; and fewer TWYSAA students drank heavily.

WATERLOO SMOKING PREVENTION PROJECT

In 1979, the Waterloo Smoking Prevention Project represented one of the first rigorous efforts to evaluate a "social influences" approach to smoking prevention. Based in Waterloo, Canada, this project made use of a school-based curriculum to help students become aware of the social pressures to smoke and to practice ways of resisting those pressures.

The first curriculum component of the Waterloo Project consisted of two sessions in Grade 6 that were intended to provide information on the consequences of SMOKING. This was done with a method pioneered by the ancient Greek philosopher Socrates, who posed questions and then used the answers and discussion to shed light on difficult problems. In the Waterloo sessions, the Socratic method was used to stimulate the development in students of beliefs, attitudes, and intentions regarding smoking. Information obtained during the discussion was repeated in later work by the instructors and also via videotapes, poster making, and class

discussions, so as to increase students' understanding and recall of the material.

The second and probably most important component of the project was the focus on the social influences that cause one to smoke (e.g., family, media, peers) and the development of skills to resist such pressures. Ideas were again elicited from students and repeated in a variety of ways. Students then practiced using the skills by role-playing what they could do when someone wanted them to smoke.

The third component of the program concerned decision making and public commitment. Students were asked to pull together the information learned earlier and to consider the social consequences of smoking in their own social environment. Each student then made a decision about smoking and announced it to the rest of the class, along with the reasons for the decision. "Booster" sessions were used to strengthen the students' skills. After the sixth-grade curriculum, students in Waterloo schools were given two booster sessions in Grade 7 and one booster session in Grade 8. All curriculum sessions were delivered by advanced graduate-school students who were on the research staff.

The Waterloo Project research team completed a very rigorous experiment to evaluate the short-term and long-term impact of this smoking-prevention curriculum and its booster sessions in grades six to eight. Out of twenty-two participating schools, eleven were designated at random to receive the Waterloo Project curriculum; the other eleven schools did not use any social-influences curriculum.

After tracking virtually all of the students in the participating schools, the research team used questionnaires to ask them whether and when they had started to smoke tobacco. There seemed to be a beneficial impact of the program before students reached Grade 9: Students who had received the curriculum were less likely to have started smoking. These early effects were not maintained during the high school years, however: The smoking levels of students who had received the curriculum were just as high as those of students who had not received it.

The value of the social-influences approach in preventing the onset of regular smoking by the end of high school needs further study. Results from the Waterloo Project and from other studies suggest that program effects obtained in junior high school might gradually decay during the following years and totally disappear by Grade 12. This kind of outcome may mean that high school booster sessions are necessary.

The apparent lack of effects of social-influence programs in preventing students from smoking by the time they reach Grade 12 should not be overinterpreted. First, it is possible that boosters in early high school years might help to maintain substantial early effects. Second, there is a much better understanding in 2000 than there was in the late 1970s and early 1980s of the essential components of effective prevention programs (Glynn, 1989). These improvements might well mean that current versions of social-influence programs might produce more durable effects. Third, society at large has changed since 1979, and social values are now more supportive of nonsmoking.

OVERALL SIGNIFICANCE

Youth drug prevention programs are seen as a vital, though often needing improvement, resource in the attempt to help today's society. Continuing evaluation of such programs is crucial since the programs need funding and are expensive. In May 2000, House and Senate subcommittees proposed further cuts (as compared to the cuts of 1999) to the substance abuse prevention budget for development and application grants. [paa]It is inherently difficult, of course, to prevent or change undesirable behavior through any classroom curriculum—given the wider and emotionally powerful influences of home, peers, and community—which create a countervailing mode, especially in the case of alcohol. Even where classroom programs might have a beneficial impact, it is difficult to measure with much sensitivity, given the present stage of evaluation technology and especially when such measurement depends on the self-reports of children.

Efforts are being made, however. For example, The Bureau for At Risk Youth published a booklet in 2000 with research-based information on establishing prevention programs. Another report in 2000 was prepared by the National Association of State Alcohol and Drug Abuse Directors detailing successful prevention program models. If continuing studies can concretely show essential and effective program elements, it is likely that ineffective programs could be enhanced to further help youth.

(SEE ALSO: *Adolescents and Drug Use; Advertising and the Alcohol Industry; Advertising and Tobacco Use; Coping and Drug Use; Education and Prevention; High School Senior Survey; Marihuana Commission; Parents Movement; Partnership for a Drug-Free America; Prevention; U.S. Government Agencies*)

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PRISONS AND JAILS Prisons serve as a principal form of punishment in the United States. In 1997, federal prison facilities held 99,000 inmates, while state prisons held just over one million inmates. Local jails held another 567,000 prisoners. These figures represent a constant and dramatic rise in prison population since the early 1990s, when federal prisons held 56,000 inmates and state prisons held 533,000 inmates. (Lipton,

Falkin, & Wexler, 1992). These increases in prison population are largely due to the public outcry against drug-related crimes and the resultant tougher sentencing practices that have been enacted against the committers of these crimes and against repeat offenders (Wexler et al., 1992). State and federal sentencing guidelines impose mandatory minimum sentences for drug crimes and these sentences are often lengthy. Repeat offenders in some jurisdictions, including New York, can be sentenced to life imprisonment. Most states have chosen to respond to prison crowding by accelerating the construction of new prisons rather than by diverting offenders into community treatment programs and increasing the emphasis on preventative measures.

The costs of incarceration in the United States are high. In 1997, the Federal Bureau of Prisons calculated the average yearly cost to incarcerate an inmate at $23,542. The average yearly cost for a state prisoner was $19,800 and the cost for those housed in local jails was $20,225. In light of these costs, states have begun to establish drug courts that use drug treatment programs rather than incarceration as the preferred remedy. States have also tried boot-camp prisons as a way to reduce the recidivism rates—the rate of repeat criminal activity—of juvenile and adult offenders, yet by 2000 states began to abandon or scale back such approaches because they proved no more effective than traditional forms of incarceration.

Especially since the advent of CRACK use in the mid-1980s, drug-dependent offenders have been responsible for a disproportionate amount of crime as compared to nonusers. In 1995, drug offenders constituted 23 percent of state prison population and 60 percent of the federal population. Many persons arrested were actively engaged in the use of drugs around the time of their arrests. Current urinalysis surveys of persons arrested in twenty-two major U.S. cities indicated that roughly two-thirds of adult arrestees and more than half of juvenile arrestees tested positive for at least one illicit drug. One-third of state prisoners and about 20 percent of federal prisoners said that they committed their offenses while under the influence of drugs. Therefore, it is clear that drug-related behavior takes up a significant part of corrections budgets.

It has become imperative to find ways of keeping offenders from reverting to crime, thereby reducing the amount of money devoted to new jails. Intensive substance-abuse treatment programs have become an important part of the corrections approach in prisons because of accumulating evidence that treatment is capable of reducing recidivism rates (Wexler, 1994). Although drug and alcohol counseling is available in nearly 90 percent of state and federal prisons, only 10 to 20 percent of prison inmates participate in treatment during their incarceration. The failure of inmates to take advantage of treatment options is troubling, especially when state corrections officials have estimated that from 70 to 85 percent of inmates need some type of substance abuse treatment.

The majority of jails also provide some form of drug treatment or counseling. Of local jails that offered drug programs in 1997, 50 percent provided detoxification, 78 percent provided drug education, 68 percent had individual counseling, 85 percent had group counseling and 87 percent provided community referrals.

(SEE ALSO: *Crime and Drugs; Prisons and Jails: Drug Treatment in; Shock Incarceration and Boot Camp Prisons; Treatment Alternatives to Street Crime; Treatment in the Federal Prison System*)

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PRISONS AND JAILS: DRUG TREATMENT IN Several public policies converged with, and the completion of important research documenting the efficacy of drug-abuse treatment for incarcerated offenders, to make the 1990s a significant decade for treatment in jail and prison settings. In regard to policy, the crack cocaine epidemic that began in the mid-1980s led state legislatures and Congress to pass drug laws which attempted to reduce crack distribution at the street level. As a consequence the arrests, convictions, and incarceration of drug offenders increased dramatically. Determinate and mandatory minimum sentences for drug-related offense resulted in overcrowded jails and prisons. More jails and prisons were built to address this growing inmate population crisis, and the composition of jail and prison inmates changed. The percent of Hispanic and African-American inmates, particularly those from the inner city, increased dramatically; late in the late 1990s, there was a substantial increase in the number of African-American women entering jails and prisons. Most inmates were seriously involved in drug use, particularly cocaine, and relatively few had received previous treatment. There was increased urgency to develop interventions that would reduce the swelling inmate population as in state and federal jails, detention centers, and prisions, as well as the related escalating costs. Interest in effective drug-abuse treatment grew.

Based on the results of federally funded efforts in the 1980s and Early 1990s, data showed that drug-abuse treatment was effective in reducing drug abuse and crime among incarcerated offenders. Among the treatment modalities that had been tried (e.g. Alcoholics Anonymous or other twelve step programs), the most comprehensive data on treatment impact existed for therapeutic communities (TCs). TCs are self-help, group-based residential treatment programs that through repetition and reinforcement try to aid addicts in developing a drug free life style. Drug abuse and crime are seen as reflecting a disorder of the whole person, not just a result of using drugs. By committing oneself to the values and activities of the TC, with its emphasis on work ethic, social productivity, and responsibility to the community, clients develop better values and the skills for right living (DeLeon, 1999; Pearson & Lipton, 1999).

PRISON BASED DRUG TREATMENT PROGRAMS

The 1990s witnessed the growth of prison TCs throughout the United States. The spread of these programs was fueled by the results of evaluation studies in New York State, Delaware, California, and Texas, which confirmed the effectiveness of this modality. These studies also emphasized that TC treatment needs to be of sufficient duration (9 to 12 months) to be maximally effective, with inmates recruited within 12 to 15 months of release eligibility. Further, a continuum of TC treatment linked to the inmate's changing correctional status (prison→work release→parole/other community supervision) was found most likely to lead to long-term success (Martin et al., 1999).

JAIL BASED DRUG TREATMENT PROGRAMS

Drug-abuse treatment in jails setting has a more limited history, and evaluation studies generally reflect a lower level of methodological rigor, than prison studies. Jail treatment outcome studies completed in Chicago and Hillsborough County, Florida nonetheless arrived at some related positive conclusions (Peters & Matthews, in press; Swartz & Lurigio, 1999). Mirroring the experience of prison studies, they indicate that length of treatment and aftercare services increased greatly the chances of long-term success. The Chicago jail program used a modified TC lasting 6 months, whereas the Florida program involved a number of treatment components (e.g., relapse prevention, stress manage-

ment). Jail-based treatment programs face a number of unique challenges:

1. devising effective programming of shorter duration than prison programs,
2. logistical problems relating to controlling client flow through the program's phases,
3. coordination with the courts to permit (wherever possible) a client to remain in program for a sufficient period to benefit from its services and follow-up.

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PRISONS AND JAILS: DRUG USE AND HIV/AIDS IN From the beginning of the epidemic in the early 1980s, HIV/AIDS has seriously affected correctional inmate populations. The first AIDS cases among inmates were reported in New York State in 1983. As the overall face of the epidemic has changed, with the virus first infecting mostly white homosexuals, to increasing predominance among African American and Hispanic intravenous drug users and their sexual partners, prisons and jails have become epicenters for HIV/AIDS, STDs, tuberculosis, and hepatitis. Nevertheless, the prevalence of HIV among inmates, although disproportionate to rates found in the total U.S. population, has probably declined in recent years.

In 1997, the most recent year for which data are available, there were about 9,200 U.S. prison and jail inmates with AIDS, representing a prevalence rate of 0.5 percent or five times that found in the total U.S. population. In addition, there were between 26,000 and 36,000 inmates with HIV infection (non-AIDS), representing a prevalence of 1.45 to 2.03 percent or five to seven times the rate in the total population. Perhaps more significantly for the public health, somewhere between 151,000 and 197,000 people with HIV infection and AIDS passed through a U.S. correctional facility and were released to the community during 1997, between 20 and 26 percent of all people in the country living with HIV and AIDS in that year (Hammett et al., 2000).

HIV prevalence rates among inmates vary widely by geographic region, with the highest rates found in the Northeast, particularly in New York State and New York City (about 10-13 percent among men and 18-20 percent among women). More than half of all HIV-infected inmates are found in correctional facilities in the Northeast. HIV prevalence rates are also typically higher among women inmates than among male inmates. The higher rates among women are generally thought to result from female inmates' generally higher rates of drug involvement, either through their own drug use or sexual relations with drug users, as well as prostitution (Hammett et al., 1999).

There is a very close relationship between substance use and HIV/AIDS among inmates. In New York State, it is consistently estimated that over 90 percent of inmate cases of HIV/AIDS are related to drug use. Although HIV transmission among inmates has been documented, it has been at quite low rates. The vast majority of inmates with HIV disease are believed to have been infected while in the community (Hammett et al., 1999).

The vast majority of correctional inmates have at least some history of substance abuse. The National Center on Addiction and Substance Abuse estimates that 81 percent of State inmates, 80 percent of Federal inmates, and 77 percent of city/county jail inmates are "substance-involved". This is defined as having one or more of the following characteristics: used an illegal drug regularly; incarcerated for a drug offense, driven under the in-

fluence, or another alcohol-related offense; under the influence when committing a crime; committed a crime to obtain money for drugs; or has a history of alcohol abuse. Despite these high rates of substance abuse among inmates, the availability of substance abuse treatment in correctional facilities is falling farther and farther short of the need (CASA, 1998). In 1997, the U.S. government's Substance Abuse and Mental Health Services Administration estimated that there were more than 865,000 state and federal inmates in need of drug treatment, but only about 111,5000 (or 13%) receiving treatment (SAMHSA, 2000).

In general, prisons and jails offer tremendous opportunities to provide substance abuse treatment, medical and mental health care services, and public health interventions such as HIV prevention programs to an extremely high-risk and underserved population. To date, however, the opportunity has by no means been fully explored.

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PROBATION *See* Coerced Treatment for Substance Offenders

PROBLEM DRINKING *See* Addiction: Concepts and Definitions

PROCESSES OF CHANGE MODEL Historically, changing an addictive behavior was assumed to be the same as taking action. People with addictions were viewed as changing when they quit drinking, smoking, or abusing other substances. Action-oriented therapies were readily available but only a small percentage of addicted individuals entered therapy, only about 50 percent completed therapy and only 25 to 35 percent were successful in overcoming their addiction following therapy. Action-oriented therapies impacted on a small percentage of addiction problems on a population basis.

In the late 1970s one-thousand ordinary people attempting to stop smoking taught us that change is a process which unfolds over time and involves progress through six stages of change: precontemplation, contemplation, preparation, action, maintenance, and termination. Creating therapies that match the needs of people at each stage of change has permitted us to reach, retain, and impact on more people than we ever imagined possible. How can therapy help people progress across the stages?

In precontemplation, people to not intend to take action in the foreseeable future. Individuals in this stage may be unaware or under-aware of their problems. Families, friends, or employers, however, are often well aware that precontemplators have problems. When precontemplators present for psychotherapy, they often do so because of pressure. Often, they feel coerced into counseling by spouses, employers, parents, or courts that threaten to punish them.

These clients are at risk of dropping out of therapy quickly and prematurely. So the first therapeutic strategy is drop-out prevention: "How can I help you to stay in therapy long enough to have it make a significant difference in your life?" Fortunately, if therapists match interventions to the client's stage, precontemplators will complete therapy at the same rate as those in preparation.

Stage matching begins by setting realistic goals. If therapists pressure precontemplators into immediate action, they will keep clients away or drive them away from counseling. Historically, thera-

pists labeled such clients as unmotivated, noncompliant, resistant, or not ready for therapy. But, it was therapists who were not ready for them, nor motivated to match clients' needs, and were resistant to changing their paradigms and practices.

The goal is to help precontemplators progress to contemplation. This initial goal produces success early in treatment. Consciousness-raising is used to help clients become aware of how they defend against pressures to quit when they are not ready. "How do you react when someone tries to pressure you to quit drinking or smoking?" Common responses include, "I get angry," "I withdraw," "I tell them to mind their own business," "I change the topic," or "I minimize the problem."

As precontemplators become aware of their defenses and start to drop them, they can process more of the pros of therapy. "We're not here just to help you understand your substance use. Therapy can help you be less defensive and happier, raise your esteem, improve your relationships, and help you make more money." As the pros of changing increase, we know that clients are progressing into contemplation.

Contemplators intend to take action in the next 6 months. Awareness of the pros of changing increase, but the cons also increase. Once clients intend to stop substance abuse, they confront the costs or cons. "Am I ready to give up my substance of choice that has been a good friend? Am I prepared to pay the price of time, effort, emotion and the risk of failure?"

A delicate balance between the pros and cons produces a profound ambivalence that causes some people to procrastinate. The love–hate relationship with their "good friend" can fool therapists into assuming that these clients are ready for immediate action. In fact, their rule of thumb is, "When in doubt, don't act!"

The goal for these clients is to progress to preparation. Their perception of the cons of quitting must change. They may need anticipatory grief counseling during which they mourn the loss of a good friend. They need to reevaluate how they think and feel about themselves as an addict and how they imagine themselves free from addiction.

Their cons have to decrease only about half as much as their pros increase, so in stage-matched treatments we place twice as much emphasis on the benefits of changing. Typically, there are more than forty scientific benefits to becoming free from an addiction. One way to enhance motivation is to become aware of how much of one's body, self, social relations, and society benefit from such major changes.

People in preparation are convinced the pros of changing outweigh the cons. They are ready to take immediate action. But, they need to be prepared for how long action will last. Many clients think the worst will be over in a matter of days or weeks. Biologically, the worst is over that quickly as they go through withdrawal. Behaviorally, however, people have to be prepared to work the hardest for about 6 months.

Clients are encouraged to think of such action as the behavioral equivalent of a life-saving surgery: "Would you inform people that recovery has to be your top priority for 6 months; that you can't be at your best and that you will need their support to get through this toughest of times?"

After 6 months, clients progress into the maintenance stage where they do not have to work nearly as hard but they still have to work to prevent relapse. How long does maintenance last? Some people believe it is a lifetime: Addicts are always in recovery and never recovered. Evidence suggests maintenance lasts 4 to 5 years. With smoking, for example, the national data in the 1990 Surgeon General's Report indicated that after 12 months of not a single puff, the percent of smokers who resume regular smoking is about 40 percent. After 5 years of total abstinence the relapse rate drops to 5 percent. When is cancer cured? Cures are counted after 5 years of no symptoms or remission. Some of the most common cases of cancer, and chronic diseases take five years to be cured.

Therapy will not continue indefinitely. But, clients will need to be prepared to cope with the most common causes of relapse. Across addictions, the most common cause is emotional distress: times of anxiety, anger, depression, boredom, loneliness, and stress. How do average Americans cope with such distress? They drink more, smoke more, eat more, and take more over-the-counter drugs and illicit drugs.

What are healthy alternatives during times of temptation? Three choices are:

(1) talking or social support;
(2) relaxing via yoga, meditation, prayer, or some other form of releasing stress or distress; and

(3) exercise or physical activity as an excellent way to manage moods, stress, and distress.

Clients need to develop a plan for how they will cope in the face of inevitable distress that will hit when therapy has stopped.

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PRODUCTIVITY: EFFECTS OF ALCOHOL ON ALCOHOL is the most commonly used and abused drug in the United States. In 1991, approximately 50 percent of all 125 million employed workers in the United States had taken at least one drink, and about 6 percent reported they had been drinking heavily (five or more drinks on five or more occasions) during the past month. Heavy drinking is more than four times as prevalent among male workers than it is among female workers, and it is most prevalent in male-dominated, semiskilled, transient occupations such as construction and transportation.

Alcohol can affect productivity in various ways. The relevant physiological effects of alcohol include intoxication, hangovers, WITHDRAWAL (abstinence syndrome) after long-term heavy use, and residual physical, mental, or social disabilities due to abuse or chronic dependence. The most important effects of intoxication—clumsiness, sleepiness, difficulty in processing new information or communicating ideas—impair physical safety and cognitive capability. Both effects can lead to poor performance, absenteeism, and job loss. Hangovers or periods of withdrawal can have similar results. Liver and heart damage, stroke, and irreparable injuries are the most common physical and mental disabilities. The most common social disability is withdrawal of trust by associates.

The consequences for economic productivity are measured not by taking them individually but by statistically estimating the overall loss of wage-earning capacity attributable to alcohol abuse and dependence. These losses are computed in two forms. *Morbidity cost* is the annual loss of earnings by individuals who are impaired by alcohol compared to the earnings of unimpaired people with similar demographic characteristics. According to the most recent estimate of this loss in the United States, one fourth of working-age men and one twentieth of working-age women were so impaired, thus averaging a 4 percent loss of earnings potential, or a total of a 35-billion dollar loss in income reduction in 1993. *Mortality cost* is the present value of the lost lifetime earnings of the nearly 100,000 individuals (two thirds of them male) who are estimated to die annually because of alcohol use—one fourth in traffic crashes, one fifth from liver disease, one eighth from homicide or suicide, one tenth from other accidents, and the remainder from esophageal cancer and a wide variety of other toxic effects. The average expected value of future earnings lost was about 33 billion dollars in 1993.

Morbidity and mortality costs account for well over half the estimated economic burden of alcohol-related illness. However, morbidity and mortality cost estimates involve complex econometric modeling procedures and use survey data from many sources. Model results have differed by as much as 200 percent for morbidity costs and 25 percent for mortality costs.

(SEE ALSO: *Accidents and Injuries from Alcohol; Complications: Medical and Behavioral Toxicity Overview; Economic Costs of Alcohol Abuse and Alcohol Dependence; Industry and Workplace; Drug Use in; Social Costs of Alcohol and Drug Abuse*)

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KARGER, J. (1997). Lax liquor rules can hurt business, careers. *Dallas Business Journal, 20*, 23.

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PRODUCTIVITY: EFFECTS OF DRUGS ON Concern about drug use in the U.S. workforce has focused on the most common illicit drugs—COCAINE and MARIJUANA—although also common is the nonmedical use of TRANQUILIZERS, SEDATIVES, and STIMULANTS. In 1990, about 7 percent of employed workers had used an illicit drug in the past month, according to national surveys. Illicit drugs are used at higher rates by men than by

women and also at higher rates by low-paid workers in transient occupations than by other workers.

Laboratory studies show that typical single doses of marijuana effect small temporary impairments in performing complex tasks, whereas typical single doses of cocaine may effect small temporary enhancements—especially when the performance of subjects is impaired by fatigue. To the extent that generalization is possible, sedatives and tranquilizers are similar in their effects to marijuana. Using illicit drugs during off hours is much more common than doing so while on the job. The effects of hangovers, post intoxication fatigue, or withdrawal from a chronic run of use may be significant for productivity, as may also be the potential accumulation of longer term disabilities, including social mistrust.

Productivity loss due to drugs is estimated by comparing the earnings of problem users with those of other people with similar demographic characteristics. The total income losses are now estimated at about 10 billion dollars annually, with a large fraction of this estimate being attributable to the nonmedical use of sedatives and tranquilizers. Productivity losses account for about one sixth of the total estimated economic burden of drug problems.

(SEE ALSO: *Industry and Workplace, Drug Use in; Productivity: Effects of Alcohol on; Social Costs of Alcohol and Drug Abuse*)

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PROFESSIONAL CREDENTIALING A host of health-care professionals provide treatment for substance-abuse disorders. They include, but are not limited to, physicians, psychologists, social workers, nurses, pastors, and addiction or drug-abuse counselors. Institutions and programs that train these professionals are accredited, and the individuals, after undergoing the training, may obtain credentials from a professional or state body. In this context, one must define the terms *accreditation* and *credential* and examine the role of each in protecting the interests of the consumer of substance-abuse treatment.

In the United States, there are two forms of educational accreditation—institutional accreditation, which began in the late 1790s in New York State, and professional accreditation, which began in the first years of the twentieth century. Accreditation is a voluntary, self-regulating process designed to evaluate the strengths and weaknesses of an educational institution. Institutional accreditation and professional accreditation have a pattern in common. It involves: (1) preparation of a detailed and objective self-study by the institution or professional program that outlines and evaluates objectives, activities, and achievements; (2) an on-site visit by a team of peers that provides expert evaluation and offers suggestions for improvement; and (3) a subsequent review and decision by a central governing commission or board to award or deny accreditation. The location of the institution determines which one of the six regional accreditation organizations will accredit it. An exception to this regional pattern is made for institutions with programs of a specialized nature, such as trade and technical education, rabbinical and Talmudic education, and the like. National accreditation bodies accredit these programs.

The U.S. Secretary of Education and the Commission on Recognition of Post-Secondary Accreditation (CORPA) recognize both regional and national accreditation organizations—that is, they accredit the accreditors. Professional accreditation is carried out, in the main, by organizations formed by members of the profession. For example, the American Psychological Association accredits doctoral programs in psychology. These specialized accreditation bodies operate nationally. Within each field, one accrediting agency is recognized by the Committee on Post-Secondary Education. These recognized agencies come together to form the Assembly of Specialized Accrediting Bodies, which works on issues of common interest to those involved in professional education. The counseling function is recognized within several professions that undergo accreditation, but the subspecialty of substance-abuse or addictions counseling is not at present independently recognized within this framework (as of 1995).

Although accreditation applies to programs or institutions and does not cover substance-abuse counseling, *credentials* apply to individuals and do cover this subspecialty. Institutions that offer training in substance-abuse counseling design their programs to meet the requirements outlined by the state or by potential employers so that graduates can obtain *certification*. Graduates must then pass tests certifying that they have a specific level of

proficiency in the theoretical and practical aspects of substance-abuse treatment. For example, in Michigan the Department of Public Health and other interested organizations initiated a program for the professional development of counselors that is based on education, experience, supervised practical training, professional recommendation, testing and review, ethics, and residence. Michigan requires that persons undergo a three-tier testing process covering the theoretical and practical aspects of substance-abuse treatment to become certified addictions counselors (CACs). The first test covers fundamental knowledge of substance-abuse counseling; the second, applications to specific populations; and the third, the oral presentation of a case. Certification is for a specific term and renewal requires additional education. Once certified, a person may provide addiction treatment in states other than the one that awarded certification, through a reciprocity agreement that covers states with membership in the International Certification Reciprocity Consortium.

In addition to certification by the state, certification may also be obtained through professional organizations. For example, the American Society of Addiction Medicine, under the auspices of the American Medical Association, certifies physicians who wish to treat substance abuse. The association offers courses that review topics in addiction theory and practice, examines candidates who wish to obtain credentials, and certifies their advanced knowledge and skills in this area. Other professional associations such as the American Psychological Association are currently developing procedures and mechanisms for providing substance-abuse-treatment credentials to their members who supply mental health services in this area.

Both accreditation and certification work to improve the quality of the education and specialty training that individuals receive and to assure the quality of the services provided. As a safeguard, consumers of substance-abuse services may determine whether the professional delivering the services was trained in a program accredited by the appropriate professional organization in a university or college accredited by the appropriate regional accrediting board. Consumers may also determine whether the professional holds credentials as a substance-abuse counselor, since these credentials certify that a person has met certain educational requirements and displayed the level of knowledge and skill deemed necessary in the profession.

(SEE ALSO: *American Society of Addiction Medicine*)

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PROHIBITION OF ALCOHOL The Eighteenth Amendment to the Constitution of the United States prohibited the "manufacture, sale and transportation of intoxicating liquors." The amendment, passed by Congress in 1917, was written to become effective one year after its ratification by the states. The amendment outlawed only the manufacture, transport, and sale of liquor; it did not criminalize the possession of ALCOHOL for personal use, nor did it make purchase of liquor from bootleggers a criminal offense, nor did it define what was meant by "intoxicating" liquors. To implement the amendment, Congress passed the National Prohibition Act, better known as the Volstead Act. The Volstead Act was crafted to allow supplies of alcohol to be produced and transported for scientific and other commercial purposes. It also defined an intoxicating liquor as any beverage containing more than 0.5 percent alcohol. It could have set the permissible level higher and allowed, for example, the production, transportation, and sale of BEER, but it did not. Prohibition became effective in 1920. A Prohibition Bureau was established within the Treasury Department to carry out the provisions of the law. Under the Volstead Act, Treasury agents could obtain a search warrant only if they could prove that alcohol was being sold, thus precluding searches of individual homes no matter how much liquor might be there. Some wealthy people, given the ample notice that Prohibition was coming, laid in enough alcoholic beverages to last them through most of the following decade. The law also had the effect of allowing manufacture for personal use. Such home production sometimes became part of a cottage industry contributing to the supplies distributed by bootleggers. Even committed Prohibitionists appeared to believe that the public would not tolerate any effort to criminalize the act of drinking itself. The Volstead Act, unlike some state laws, permitted the manufacture of beer

as long as the beer contained no more than 0.5 percent alcohol (near beer).

Given the common belief that Prohibition failed utterly to alter the consumption of alcohol or its adverse effects on health, it is appropriate to ask, To what extent did the law reduce alcohol use in the United States? First, there is no question that it succeeded in eliminating 170,000 saloons, even if it did not change the attitudes of most Americans about the morality of drinking. And, while some writers have asserted that drunkenness actually increased during Prohibition, most available records point to the opposite conclusion (Aaron & Musto, 1981; Lender & Martin, 1987). The most consistent findings on the impact of Prohibition come from statistics on medical problems known to be linked to alcohol consumption, especially excessive alcohol consumption. Among these problems were hospital admissions for alcoholism and admissions to state mental institutions for alcoholic dementia and alcoholic psychosis. Striking decreases were observed in New York and Massachusetts, two states that did not have restrictions on alcohol consumption prior to 1920. Massachusetts state mental hospital admissions for alcoholic psychosis fell from 14.6 per 100,000 in 1910, to 6.4 in 1922, and were 7.7 in 1929; in New York, such admissions fell from 11.5 in 1910, to 3.0 in 1920, rising again to 6.5 in 1931 (Aaron & Musto). Deaths from alcohol-related diseases also fell. National statistics showed that the number of deaths from cirrhosis, about 14.8 per 100,000 in 1907, were only 7.9 in 1919, 7.1 in 1920, and did not rise above 7.5 during the 1920s. There were decreases in arrests for drunkenness and in the costs of jailing public inebriates. Commander Evangeline Booth of the Salvation Army asserted that not only had drinking fallen off sharply, especially among the poor, but there were fewer broken homes because of wages lost to drinking or violence related to drinking.

Aaron and Musto state, "Observers . . . have been unanimous in concluding that the greatest decreases in consumption occurred in the working class. . . . In large measure, intoxicants priced themselves out of the market" (Aaron & Musto 1981, p. 165). A quart of beer or a quart of gin were five to six times more expensive in 1930 than they were prior to Prohibition. Prohibition defenders asserted that instead of purchasing liquor in saloons, workers were putting their earnings into cars and refrigerators. Admittedly, the impact on alcohol consumption was greatest in the early years of Prohibition. As bootlegging increased in the late 1920s, medical problems linked to alcohol use began to rise again, but they did not reach the high levels experienced before 1920. Other data on per capita alcohol consumption immediately after repeal in 1934 indicated that there must have been a drastic decline in average alcohol consumption during the Prohibition years. Undoubtedly, crime associated with bootlegging increased. Many bootleggers became quite wealthy. Some who were involved in illegal activities prior to Prohibition used the wealth flowing from bootlegging to extend and further develop organized criminal enterprises, some of which later became involved with trafficking in illicit drugs. One of the most notorious of the figures associated with organized crime was Al Capone, who came to national attention as a result of his Chicago-based criminal activities. Aaron and Musto point out, however, that organized rackets existed in large cities before Prohibition and that the homicide rate increased most sharply between 1900 and 1910.

Patrons of a speakeasy enjoy a drink illegal under the Volstead Act. Undated photograph. (© Bettmann/CORBIS)

Unquestioned, also, is the unreliable quality of bootlegged liquor, much of which was produced by diverting or hijacking industrial alcohol. Some industrial alcohol could simply be flavored and sold as scotch, gin, or bourbon. Much of it, however, had been mixed with METHANOL (methyl alcohol) or other chemicals to render it undrinkable—denatured. Bootleggers hired chemists to remove the denaturants by redistillation ("washing"). Inadequate processing, which was not uncommon, produced a liquor that could be toxic or even lethal. The liquor produced in England and Canada and smuggled in by ship or truck was of a higher quality. One smuggler who brought in such quality liquor, Bill McCoy, has given us a term still used to describe an authentic product—the "real McCoy."

The continued criticism of Prohibition and the frustration of enforcing the Volstead Act led many of their advocates to become increasingly defensive and hostile to those not seen as supporters. Concern for the drunkard sharply diminished. According to Lender and Martin, "Many crusaders began labelling rehabilitation as nothing more than a waste of time and energy; prohibition, they promised would make such work unnecessary" (Lender & Martin 1987). Groups interested in treatment declined. The Association for the Study of Inebriety dissolved in the mid-1920s. Volstead Act advocates became more hostile toward alcoholics as criticism of Prohibition increased. Some suggested amending the Act to make drinking itself a criminal offense. One such suggestion came from an official in the Prohibition Unit of the Treasury Department, Harry J. ANSLINGER, then the Assistant Commissioner of Prohibition. Thus the nineteenth-century concerns of the TEMPERANCE MOVEMENT for the physical and spiritual health of alcoholics turned, in the 1920s, to calls for stiffer jail terms, or even exile, for chronic alcoholics. In the context of these attitudes, the harsh penalties that were then being meted out under the leadership of the Treasury Department for mere possession of illicit drugs become somewhat more comprehensible.

The enforcement of the Volstead Act had been vested in the Treasury Department's Prohibition Unit within the Internal Revenue Bureau. The first National Prohibition Administrator, the head of the Prohibition Unit, was John F. Kramer. The Narcotics Division, headed by Levi G. Nutt, a pharmacist by training, was part of the Prohibition Unit. The Narcotics Division became an independent unit in the Treasury Department in 1930 when the Prohibition Unit was transferred to the Department of Justice. Harry J. Anslinger was appointed first Commissioner of Narcotics.

Despite growing criticism, Prohibition, according to Aaron and Musto, was still alive and well when Herbert C. Hoover was elected president by a large margin in 1928. An overwhelming majority of both houses of Congress and nearly all the state governors supported the Eighteenth Amendment. Even opponents of Prohibition did not realistically expect to see it repealed. But the onset of the Great Depression in 1929 dramatically changed the situation. Opponents of Prohibition no longer argued for its repeal because of its demoralizing effects on civil liberty but argued instead that the revival of the liquor industry would provide jobs and tax revenue. In the 1932 campaign for the presidency, Franklin D. Roosevelt promised to repeal Prohibition. Almost immediately after his inauguration, he had changes introduced in the Volstead Act to legalize the sale of beer.

In 1933, the Twenty-First Amendment to the Constitution was ratified. It was brief and to the point: "Section 1. The Eighteenth Article of Amendment to the Constitution of the United States is hereby repealed." The federal government, however, retained responsibility to regulate and tax beverage alcohol and to prevent its illegal production. Section 2 of the Amendment allowed the states to continue Prohibition under state laws if they so desired. Some states did so; many states adopted alcohol beverage control laws (ABC laws). These were intended to curb the abuses that had characterized the production and sale of alcohol prior to prohibition. Among other provisions, ABC laws restricted the hours when alcohol could be sold (to make taverns and bars less attractive) and banned liquor sales on Sundays and election days. Some ABC laws created state-operated monopolies for the sale of packaged beverages. The various federal laws dealing with control of alcohol remained the responsibility of various federal agencies. It was not until 1972 that they were brought together and responsibility lot overseeing them was assigned to a single agency—the Bureau of Alcohol, Tobacco, and Firearms (BATF) in the Department of the Treasury.

(SEE ALSO: *Alcohol: History of Drinking; Harrison Narcotics Act of 1914; Legal Regulation of Drugs*

and Alcohol; Tax Laws and Alcohol; Temperance Movement; Treatment, History of)

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PROHIBITION OF DRUGS: PRO AND CON *See* Policy Alternatives

PROJECT RETURN FOUNDATION, INC. *See* Treatment Programs/Centers/Organizations: An Historical Perspective

PROJECT SMART/STAR *See* Prevention; Prevention Programs

PROPOXYPHENE *d*-Propoxyphene (Darvon®) is an OPIOID drug that is structurally related to METHADONE. It is used clinically to produce analgesia when the level of PAIN is not severe. Its popularity rests largely on the belief that propoxyphene is less likely to cause addiction than CODEINE, a drug that is also used for relief of moderate levels of pain. Propoxyphene is typically used in combination with aspirin or acetaminophen. Its ANALGESIC effects are synergistic with those of aspirin and other nonsteroidal anti-inflammatory agents.

When it was introduced into clinical medicine in the early 1960s, propoxyphene was not subject to special narcotic regulatory control. This fact may explain its early popularity, which was probably due to clinicians' unrealistic fears about the addictive potential of codeine and to the inconvenience of prescribing it under the narcotic regulations that were in effect before the the CONTROLLED SUBSTANCES ACT of 1970 was passed.

Although propoxyphene has only one-half to two-thirds the potency of codeine, it has been used to control symptoms of the opioid WITHDRAWAL syndrome. It is not commonly abused because it produces unpleasant toxic effects at high doses.

(SEE ALSO: *Opiates/Opioids*)

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PSILOCYBIN This is an indole-type HALLUCINOGEN, found naturally with another hallucinogen in a variety of mushrooms—the most publicized being the Mexican or MAGIC MUSHROOM, *Psilocybe mexicana*, as well as other *Psilocybe* and *Conocybe* species. These mushrooms have long been consumed by Native Americans, especially in Mexico and the southwestern United States, as part of religious rites.

Psilocybin produces effects similar to LYSERGIC ACID DIETHYLAMIDE (LSD), but it is less potent and is metabolized in the body to form psilocin, another hallucinogenic compound. Both of these compounds have been synthesized in clandestine laboratories and made available on the streets.

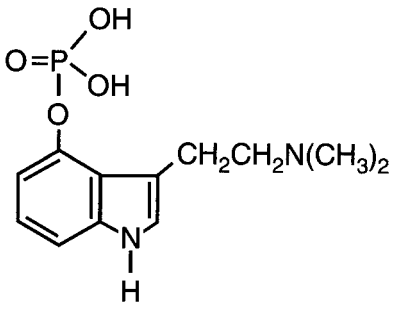

Figure 1
Psilocybin

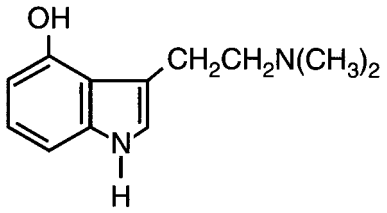

Figure 2
Psilocin

(SEE ALSO: *Hallucinogenic Plants; Peyote; Plants, Drugs from*)

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PSYCHEDELICS *See* Hallucinogens; Lysergic Acid Diethylamide (LSD) and Psychedelics

PSYCHOACTIVE *Psychoactive* is a general term that came into use about 1961. It describes a substance that affects the central nervous system, producing changes in mental activity and/or behavior. A psychoactive substance or process may affect the way an individual thinks or the manner in which the environment is perceived or experienced; it may change the behavior of an individual in a given situation.

(SEE ALSO: *Psychopharmacology*)

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PSYCHOACTIVE DRUG Any of a group of drugs (also called psychotropic drugs) that act upon the central nervous system, producing changes in mental activity and/or behavior. Psychoactive drugs are among the most widely used group of pharmacologically active agents, with extremely important clinical applications, including anesthesia for surgery and analgesia for relief of pain. They are also used for nomedical purposes, such as to alter consciousness, improve performance, and as elements in cultural and religious rituals (alcohol and peyote are examples). Some psychoactive drugs produce an effect in those who suffer from a mental or medical disorder, but no effect on normal individuals. The antidepressants, fo example, have little or no effect on normal individuals other than side effects. Other psychoactive drugs, such as the sedative-hypnotics, produce effects in all individuals.

Psychoactive drugs are used to suppress disorders of movement and to treat anxiety disorders, depression, bipolar disorder (manic-depression), and schizophrenia, among other mental illnesses. In addition, drugs used primarily to treat disorders in peripheral organs can also affect the central nervous system (e.g., beta-blocking agents, used to treat high blood pressure or disorders of heart rhythm, or steroid hormones used to control inflammation). The psychoactive effects of these drugs are generally considered side effects, although some are used for their psychoactive properties as well.

Culturally approved non-medical psychoactive drugs include alcohol, nicotine (tobacco), and caffeine. Psychoactive drugs that have been determined to have a high potential for harm and little medical benefit include heroin, hallucinogens, and some older sedative-hypnotics such as methaqualone. Marijuana has traditionally been placed in this category, but recent research has demonstrated potential effectiveness for medical problems including glaucoma, nausea, and weight loss associated with cancer or AIDS.

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PSYCHOANALYSIS Psychoanalysis is an analytic technique originated by Sigmund Freud (1856–1939), an Austrian neurologist. It has been altered by his students and their students, in turn, throughout the twentieth century. Psychoanalysis is a theory of the way the mind works: (1) Sequences of thoughts are determined—they do not occur by chance; (2) Much of our thinking takes place out of awareness—it is unconscious and not easily recovered; (3) The experiences of early childhood, particularly those with important caretakers, continue to have an impact (often uncon-

sciously) on our daily lives; (4) Feelings, both sexual and aggressive, are present at birth and affect behavior. The theory helps us understand something of the addicts' complex motivations and of their inner experience and behaviors.

Psychoanalysis is also a method: It attempts to understand mental processes by free association (following thoughts wherever they lead without selection or censoring) and by the analysis of dreams, fantasies, and behaviors. Psychoanalysts apply this method as a therapy or treatment for certain forms of mental disability.

(SEE ALSO: *Causes of Substance Abuse: Psychological [Psychoanalytic] Perspective*)

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PSYCHOLOGICAL DEPENDENCE *See* Addiction: Concepts and Definitions

PSYCHOLOGICAL TREATMENT FOR SUBSTANCE ABUSE *See* Treatment; Treatment Types

PSYCHOMOTOR EFFECTS OF ALCOHOL AND DRUGS Alcohol and other drugs of abuse can alter normal behavior in a deleterious way. Epidemiological studies have shown that 50 percent or more of all single-vehicle traffic fatalities in the United States are associated with the use of ALCOHOL. The risk of a driver causing an accident increases progressively the more that BLOOD ALCOHOL CONTENT (BAC) increases past 0.4 grams per liter (g/l). At BACs of 1.0 g/l, the risk is tenfold, and with BACs of 1.5 g/l, the risk is almost thirtyfold compared to nonalcohol conditions. The same phenomenon applies to accidents in which pedestrians are killed by drunk drivers.

PSYCHOMOTOR PERFORMANCE

Most behavioral tasks are complex processes in which information sampling and its processing, motor responses, and sensorimotor coordination are involved. A decrement in any part of this system leads to impaired performance. Numerous studies describe techniques used to assess the psychomotor functions of people under the influence of chemicals with the potential for impairing performance. The vastness of the range of behavioral activities, however, makes it unlikely that any one, or even a small number, of tests could completely describe the impairing properties of alcohol and other drugs under all conceivable circumstances.

A way to approach this problem is to isolate the main variables of performance into smaller entities and measure the effects separately with a set of relevant tests. Since psychomotor behavior consists of external stimuli and a rational response to them, a simplified chain of events can be divided into a sensory part (detection of stimulus), a central part (complex processing of the sensory information), and a motor part (overt behavior or motor reaction to the stimulus).

It is sometimes difficult to select the most sensitive, accurate psychomotor test for various agents that impair performance. Sets of tests have been used—for example, in studies on the likelihood of bus drivers to have traffic accidents. The capabilities that best characterized the drivers with low accident records were constant and keen attention, adequate information processing, and the absence of hasty reactions. Eye-to-hand coordination was less important, and simple reaction times represented the poorest correlation to safe driving. Although it is logical to choose a set of tests that cover the most important variables, in most tests there is an overlap among several skills. Alcohol, drugs, and their combinations, moreover, may impair these integrated variables to a varying extent in different individuals. Because of this, one cannot predict or give exact numerical data for the amount of impairment associated with a single variable of the system affected. Nor does impairment in one sensitive test mean that the overall performance is severely impaired. In practice, it may not be important to know whether the accident of a drunken driver resulted from impaired attention rather than from poor motor coordination or slowed reactions, when all these skills were more or less affected.

CONFOUNDING FACTORS IN PSYCHOMOTOR TESTING

Substance abuse is commonly, but not necessarily, associated with an acquired TOLERANCE; this means that after repeated administration, a given dose of a drug produces a decreased effect and larger doses become needed to obtain the effects observed with the original dose. Deleterious psychomotor effects are usually easy to detect when large single doses are taken by people who have not yet acquired tolerance to the effects of drugs. The question becomes more complex when the user who takes small doses acquires significant tolerance to them because of regular use.

For any skilled performance, a large variation is observable among individuals. Thus some people may, by nature, have slower reactions or poorer information-processing capacities, and their best performance in the respective tests may be clearly worse compared with that of more capable subjects—even when the more capable are under the influence of performance-impairing drugs. The decremental drug effect can be similar in both cases, but the more capable subjects can afford it because of their better reserves. It is consequently difficult to define safe and unsafe doses of any agent.

Other factors that may influence psychomotor behavior include motivation, learning, adaptation to the task, and drowsiness. Paying the subjects according to how well they perform might improve motivation and performance, and this might skew the test results. (Such a motivational enhancer is not always mentioned in the research reports.) Impairment of performance may not be detected in tasks of short duration in a stimulating environment, whereas deleterious effects can be documented in monotonous tasks of long duration. Transposed to normal life situations, this observation may explain why an inebriated driver can get through a difficult driving test without any significant errors but cannot handle a surprising event after several hours of monotonous driving on a highway.

ALCOHOL

An alcohol dose affects the central nervous system (CNS)—the predominant effect being a depression of central functions. This means that the higher the dose of alcohol, the more the CNS is depressed. The most highly integrated brain functions are involved first; when the brain cortex is released from its functions of integrating and control, processes related to judgment and behavior occur in a disorganized fashion and the proper operation of behavioral tasks becomes disrupted.

The effects of alcohol are biphasic, and the phases depend on the dose and the rate of administration. With higher alcohol concentrations, central depressant effects dominate. Low concentrations seem to stimulate various functions by inhibiting the control mechanisms. This is seen in animal studies as decreased motor activity with large doses of alcohol and increased activity with small doses. In humans, very small doses of alcohol do not necessarily impair performance, and the tension-relieving effects of alcohol can sometimes be seen in some tests of short duration. However, there is no reason to overestimate this occasionally observed pseudostimulant effect of alcohol; in actuality, alcohol impairs various skills that are needed to cope with everyday routines.

Several investigators have demonstrated that alcohol does induce a larger decrease in test performances requiring hand-eye coordination, whereas simple tests of cognitive ability show less of a decrease. When more complex cognitive functions are studied, however, low to moderate BACs (0.3–1 g/l) impair sensory tasks and sensorimotor skills less than they do complex cognitive behavior, such as performing two tasks simultaneously (“divided attention”). It thus seems that alcohol impairs the rate of information processing by slowing the ability to switch attention from one to another sensory input to motor control, without significantly impairing sensory motor functions as such. In fact, moderate BACs (less than 1 g/l) are not associated with dramatic changes in such basic neurophysiological mechanisms as neuromuscular transmission or the conduction velocity of motor nerves. Alcohol effects are thus better seen in situations where the information load is increased and highly integrated functions are needed for the task.

It is well known that the muscles of the eye and eye movements often easily reflect the CNS depression caused by alcohol. One of the most sensitive signs is the appearance of lateral nystagmus; small twitches or vibration in the position of the eye are seen when the person looks to the side. The angle of the gaze at which the nystagmus appears correlates

with the alcohol dose: On average, a BAC of 0.5 g/l induces nystagmus at a 45-degree angle of deviation, whereas a BAC of 1 g/l produces nystagmus even at the 35-degree angle. Also, saccadic eye movements (from one fixation point to another) become slower with BACs of 0.8 g/l to 1 g/l. All this indicates that people who are drunk have a narrower sector of intact vision than people who are sober. Visual information becomes disrupted if eyes must be turned to the side to detect stimuli, or if eyes must be moved quickly from one point to another.

Several types of tests measure skilled performance in tasks related to driving behavior. *Tracking tasks* involve hand-to-eye coordination, and the task is to keep an object on a prescribed path by controlling its position through turning a steering wheel. Impairment of performance is seen at BACs of as little as 0.7 milligrams per milliliter (mg/ml). *Choice reaction task* refers to a situation where aural or visual stimuli (or both) need response according to rules that necessitate mental processing before giving the answer. In traffic, driving requires a division of attention between a tracking task and surveillance of the environment. When a driver must process information from more than one source concomitantly—by adding sudden reaction tasks to the tracking task—very low BACs are sufficient to produce significant impairment of performance.

Clinical tests for drunkenness include many simple tasks that are easy to measure even in field conditions. These can be divided into three subtests. (1) Motor subtests consist of measuring a person's ability to walk along a straight line with eyes open and closed; maintain a steady turning gait; fit the tips of index fingers together with eyes closed, and collect small objects (e.g., matches) from the floor. (2) *Vestibular* subtests assess the person's body sway, with eyes open and closed, and nystagmus. (3) *Mental* subtests assess the driver's ability to subtract backward, orientation as to time, and overall behavior. The performance in each subtest is graded from 0 to 3, but these clinical tests are not very sensitive to small BACs (nystagmus exlcuded), and there is great individual variation. The use of these clinical tests for drunkenness in field conditions has greatly diminished since portable BREATHALYZERS became available. The tests are most useful in situations where one has to decide whether to take a blood test for detection of other drugs when no alcohol is found in the driver's breath. Unfortunately, tests developed to detect alcohol effects are less sensitive to the effects of BENZODIAZEPINES and other CNS depressant drugs.

DRUG-ALCOHOL INTERACTIONS

It is well known that large doses of CNS-active drugs impair various of the functions and interact at least additively with alcohol, thereby resulting in heavy sedation or unconsciousness. This effect suggests that even small doses of alcohol may impair performance when taken together with correctly prescribed CNS-active drugs such as anxiolytics, ANTIPSYCHOTICS, ANTIDEPRESSANTS, and OPIOIDS. The deleterious interaction is most obvious when single doses are taken. The issue becomes more complex in chronic alcohol abuse when acquired tolerance of varying extent has developed. Such an adaptation often decreases the expected pharmacological actions of other psychoactive drugs (an effect termed *cross tolerance*).

ALCOHOL AND BENZODIAZEPINES

Taken orally, benzodiazepines have a low acute toxicity. Low doses taken with alcohol (ethanol) may impaire skilled performance. A specific benzodiazepine antagonist (flumanzenil) effectively cancels the share of benzodiazepines in mixed intoxications.

Although the risk of a driver having an accident while under the influence of alcohol increases progressively as the BACs increase, a study of the epidemiology and psychomotor effects of benzodiazepines and alcohol are not clear in this respect. One might expect their combined action to be potent, but this has not been documented. Under experimental conditions, a person's tolerance to the drug has been found to minimize or cancel the expected enhanced action of the benzodiazepine in combination with alcohol.

ALCOHOL AND CANNABIS

With chronic (long-term) use of *Cannabis* (MARIJUANA), a person may acquire a tolerance to its effects. However, tests show the combined effects of ethanol and cannabis to be detrimental to skilled performance. This interaction is potentiative and multidimensional, resulting partly from the fact that *Cannabis* shows a peculiar increase of

effect with time that is unrelated to plasma-*Cannabis* levels.

ANTI-ALCOHOL DRUGS

This category generally covers both drugs used to diminish motivation for drinking plus those that cancel (as antagonists) alcohol intoxication. Although the list of possible antagonists is long—and includes AMPHETAMINES and CAFFEINE—no convincing antagonism has been documented. Therefore, no pharmacological agent exists to cancel out the psychomotor effects of alcohol to allow sober performance.

(SEE ALSO: *Accidents and Injuries from Alcohol; Addiction: Concepts and Definitions; Blood Alcohol Concentration; Driving, Alcohol, and Drugs; Driving Under the Influence; Drunk Driving*)

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PSYCHOMOTOR STIMULANT This term is used to describe drugs that act as central nervous system (CNS) stimulants. Such drugs generally are appetite suppressants, decrease sleep and fatigue, increase energy and activity, and at higher doses can cause convulsions and death.

Ingestion typically results in increased wakefulness and a decreased sense of fatigue, increased speech and motor activity, alertness, and, frequently, elevation of mood. Many of the drugs in this class have a potential for abuse, with reports of euphoria at higher doses. Although users often report improved performance on physical and mental tasks, this is rarely the case, but they do restore performance that has been impaired by fatigue.

Prolonged use of most of these drugs can result in tolerance to many of their effects. Repeated high doses can result in distorted perception and overt psychotic behavior.

(SEE ALSO: *Amphetamine; Cocaine; Tolerance and Physical Dependence*)

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PSYCHOPHARMACOLOGY Psychopharmacology is that branch of science that involves the study of the effects of interactions between drugs that affect the central nervous system (i.e., psychoactive drugs) and living systems. Behavioral and neurobiological effects as well as the mechanisms of actions and side effects of drugs are often examined. Pre-clinical studies of psychoactive drugs using animal models and tissue preparations are an important aspect of psychopharmacology, contributing to our understanding of the mechanisms involved in disorders of the central nervous system and mental illness. Clinical psychopharmacological investigations include examining the effects of drugs used in treating psychiatric disorders (such as anxiety, depression, schizophrenia, and mania), as well as other dysfunctions within the central nervous system (such as movement disorders, Alzheimer's disease). Also included is study of the effects of psychoactive drugs used non-medically to induce altered states of consciousness, to improve mood, or to otherwise affect the mental status and/or behavior of the individual. A growing area of research in psychopharmacology addresses disorders of addiction or dependence to some of these drugs. New treatments for alcoholism (naltrexone), opioid dependence (burprenorphine), and smoking cessation (bupropion) have resulted from these efforts, and many more treatments are under development. Some of the drugs used for treatment of depression and anxiety are also being investigated for potential usefulness in treating substance dependence, since it is often accompanied by these comorbid conditions.

Psychopharmacology is an interdisciplinary field of science. Psychopharmacologists may be

physicians trained in psychiatry or neurology, psychologists with extra training in pharmacology, or pharmacologists with special training in psychology and behavior.

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PSYCHOSTIMULANT *See* Drug Types; Psychomotor Stimulant

PSYCHOTHERAPY *See* Treatment; Treatment Types

PSYCHOTROPIC SUBSTANCES CONVENTION OF 1971 The 1971 Convention on Psychotropic Substances extended the international drug control system to cover mood-altering substances such as stimulants (e.g., AMPHETAMINES), SEDATIVE-HYPNOTICS (e.g., BARBITURATES), and HALLUCINOGENS (e.g., LSC and MESCALINE). It limited the use of these substances to medical and scientific purposes, and it did not cover ALCOHOL or TOBACCO. As of November 1994, 132 governments were party to the convention.

GENERAL PROVISIONS

The manufacture, trade, and distribution of psychotropic substances are subject to licensing, record keeping, and reporting. The convention generally permits governments great flexibility in applying the provisions to meet their particular needs, because it recognizes that psychotropic substances are widely used in medical practice to treat mental and physical disorders. In addition, the convention includes provisions for the prevention of abuse and for the treatment and rehabilitation of drug addicts. Because of the convention, a substance abuser may receive treatment, education, aftercare, and rehabilitation as an alternative or in addition to punishment.

A patient may not obtain any of the substances regulated under the convention without a medical prescription, although exceptions are allowed under certain circumstances, when licensed pharmacists may supply small quantities of the substances that are less likely to be abused. In addition, the convention set forth precautions to be taken to ensure that the distribution of psychotropic substances conformed to sound medical practice. An example of such practice is the proper labeling of retail packages to include adequate directions for use and warnings, if necessary.

A party to the convention may prohibit exportation of psychotropic substances from its country. It may also notify other parties, through the United Nations secretary-general, that it prohibits the import of schedule II, III, or IV substances into its country.

All signatories must provide detailed annual statistical reports on the production, trade, and consumption of psychotropic substances to the International Narcotics Control Board (INCB), the central authority that was established to coordinate control of the illegal manufacture and use of narcotics. The reports for substances in schedules I and II must be more detailed than those for substances in schedules III and IV, which are not as rigidly regulated.

SCHEDULES OF PSYCHOTROPIC SUBSTANCES

A psychotropic substance is assigned to one of four schedules by balancing the drug's potential for abuse and the threat it poses to the public health against its therapeutic behefits. The placement of a drug in one of the schedules affects its trade, manufacture, distribution, and use. Hallucinogens and other drugs that are of no—or severely limited—medical use are placed in schedule I. Schedule I substances, the most stringently regulated of the four schedules, may only be used for scientific and limited medical purposes in government-operated licensed establishments. The manufacture, trade, distribution, and possession of these substances require special licensing or authorization from the government. The amounts of these substances that

may be supplied, imported, and exported are limited, even for authorized uses, and records of their use must be kept.

Schedule II drugs, such as METHAQUALONE and amphetamines, possess a high potential for abuse and limited medical usefulness, and therefore they are subject to tighter controls over their production and trade than substances in schedule III and schedule IV. Governments must issue special import and export authorizations before these drugs can be traded internationally. Experience has shown that placement of a substance in schedule II severely reduces its use.

Schedule III and schedule IV have been assigned to such drugs as depressants, sedative hypnotics, anxiolytics, barbiturates, and minor tranquilizers. Individuals and businesses involved in the manufacture, trade, and distribution of schedule III and schedule IV psychotropic substances must have licenses from the government. They must maintain records of the manufacture and wholesale trade, import, and export of these substances. The World Health Organization (WHO) has designated several drugs in schedule IV, including BENZODIAZEPINES such as diazepam (Valium®) and alprazolam (Xanax®), "essential drugs" that governments must assure are available for medical purposes.

ROLE OF THE WORLD HEALTH ORGANIZATION

The Convention on Psychotropic Substances allows the United Nations Commission on Narcotic Drugs to add substances to its schedules, and also to transfer or remove them. WHO recommends what it considers to be the appropriate placement of drugs within schedules. A party to the convention may ask the United Nations secretary-general to recommend that WHO place other drugs under control. WHO reviews substances to determine whether they have the "capacity to produce a state of dependence and central nervous system stimulation or depression resulting in hallucination or disturbances in motor function or thinking or behavior or mood" and whether they pose a risk to public health. WHO must make known in great detail the criteria it applied in evaluating a psychotropic substance for control.

The evaluations WHO makes are based on scientific and medical criteria, but in deciding whether to accept or reject WHO's recommendations, the United Nations Commission on Narcotic Drugs may consider social, economic, and political issues. A two-thirds majority vote is required, however, before the Commission may alter or amend a schedule. If, because of exceptional circumstances, a party cannot apply the provisions of the convention to a newly added substance, it may notify the United Nations secretary-general and obtain permission to satisfy only minimal control requirements.

SIGNIFICANCE OF THE CONVENTION

The 1971 Convention on Psychotropic Substances recognized that the abuse of mood-altering substances, like the abuse of narcotic drugs, could have harmful effects, at the same time that it acknowledged that psychotropic drugs provide important medical and scientific benefits. Through the treaty drawn up at the Convention, the international community took another step in the cooperative effort to curtail drug abuse while preserving the availability of psychotropic substances for legitimate medical use.

(SEE ALSO: *International Drug Supply Systems; Single Convention on Narcotic Drugs; WHO Expert Committee on Drug Dependency*)

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ROBERT T. ANGAROLA

PUBLIC INTOXICATION Before the seventeenth century, public intoxication was not, by itself, a crime in England. Drunkenness was punishable as a criminal offense only if it resulted

in some form of breach of the peace or disorderly conduct. In 1606, however, in England, simple public intoxication was first made a criminal offense. This English precedent was reflected in some laws in the American colonies as well as in the United States in the city, county, and state laws enacted after the American Revolution. By the early 1960s, about two million arrests occurred annually for simple public intoxication, representing about 33 percent of all arrests in the United States.

Since then, remarkable changes have occurred in the handling of public intoxication. Through efforts initially in the courts and later through federal and state legislation, important steps have been taken to transfer the handling of public intoxication from the criminal-justice system to more humane and effective public-health care. The major stumbling blocks to further progress have been the lack of adequate funding and uncertainty about the most effective way of treating alcohol abuse and alcoholism.

INITIAL COURT CHALLENGES

Beginning in 1964, lawyers argued that derelict alcoholics could not lawfully be punished for their public intoxication on two independent grounds. First, they argued that these derelict alcoholics did not have the *mens rea* (Latin, guilty mind or intent) required for conviction of a crime, because their public intoxication was a symptom of the disease of alcoholism. Second, they argued that punishing an alcoholic for exhibiting the symptoms of that disease in public was cruel and unusual punishment, prohibited by the U.S. Constitution.

In lower court cases, these arguments prevailed. In 1968, however, in the case of *Powell v. Texas*, the U.S. Supreme Court handed down a split decision on this issue. Four justices found that it would be cruel and unusual punishment to convict Powell, an admitted alcoholic, for simple public intoxication. Four other justices determined that the matter should be left to the states and should not be decided on a constitutional level. The ninth and controlling justice determined that, because Powell had a home, he could properly be held responsible for being intoxicated in public and thus was appropriately convicted. This left open the question of whether a derelict alcoholic, without a home, could also be convicted.

A drunk man lies passed out near the celebration at the Berlin Wall on New Year's Eve 1989. (© Owen Franken/CORBIS)

ENACTMENT OF FEDERAL STATUTES

Faced with a stalemate in the Supreme Court, advocates for reforming the public-intoxication laws turned to Congress. In spite of a large number of federal public health statutes, none referred explicitly to the problems of intoxication and ALCOHOLISM. Congress responded by enacting the Alcoholic Rehabilitation Act of 1968, which recognized alcoholism as a major health and social problem, and recommended handling public intoxication as a health problem rather than as a law-enforcement matter. This was followed by the Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment, and Rehabilitation Act of 1970, which created the NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM to administer all alcoholism programs and authority assigned to the U.S. Department of Health, Education, and Wel-

fare (now the U.S. Department of Health and Human Services). These new federal laws for the first time provided a national focus for handling public intoxication on a public-health basis.

CHANGES IN THE STATE STATUTES

Before the court cases and the federal statutes, simple public intoxication constituted a criminal offense throughout the United States. Following the dramatic legal developments in the courts and in Congress, state and local laws rapidly began to change. Initially in the District of Columbia and in Maryland, and subsequently throughout other parts of the country, the criminal statutes prohibiting simple public intoxication were repealed and replaced with new laws establishing detoxification programs for intoxicated persons and rehabilitation programs for chronic alcoholics. By the early 1990s, more than 67 percent of the states had revised their laws to reflect this change in approach.

THE CURRENT STATUS

Existing federal and state laws now provide a firm foundation for handling public intoxication as a public-health problem rather than as a matter for the criminal-justice system. Relatively little additional change can be accomplished solely by further litigation or legislation.

With these legal and legislative hurdles overcome, two additional obstacles have arisen to impede further progress. First, the competition for federal and state health funds has become intense. Other important health needs, including basic health care for the needy and treatment for people with acquired immunodeficiency sydrome (AIDS), have made it very difficult for public officials to devote adequate resources for the expansion of public-health programs to include public intoxication and alcoholism. The problem has been compounded by a lack of any clearly effective method for the prevention or treatment of intoxication and alcoholism. A low rate of rehabilitation has led many public health officials to conclude that scarce public resources are more effectively devoted to other illnesses, especially communicable diseases. Unless there is additional investment, the police will remain deeply involved in identifying and responding to intoxicated individuals, and their response will not necessarily be limited to transporting the individual to a sobering-up station.

Progress in the prevention and treatment of intoxication and alcoholism has therefore been slow, in spite of the major changes made in the courts, the Congress, and state and local legislative bodies. Unless and until the American public places a higher priority on the handling of public intoxication as a public-health matter or medical science finds more effective methods to prevent and treat this problem, this situation is unlikely to change.

Two developments in the last decade of the twentieth century illustrate the public concern and frustration with the continuing problems of public intoxication and alcoholism. First, publicity about the substantial death and destruction caused by people driving under the influence of alcohol has led to more stringent penalties and more strict enforcement against this behavior. Second, tragic death caused by binge drinking on college campuses have led to an increase in the drinking age from 18 to 21, and stricter enforcement in college towns throughout the country.

(SEE ALSO: *Alcohol: History of Drinking; Detoxification; Homelessness, Alcohol, and Other Drugs; Temperance Movement; Treatment: Alcohol; Treatment, History of*)

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PETER BARTON HUTT

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Volume 3

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R – Z

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RACIAL PROFILING "Profiles," formal and informal, are common in law enforcement, particularly in narcotics law enforcement. They consist of general characteristics and features that might make a law enforcement officer suspicious. In some instances, law enforcement agencies formulate and disseminate formal profiles to officers to guide their investigative actions. Even when profiles are not formally maintained, however, officers inevitably rely on their past experience to generate informal profiles for whom to follow more closely, approach, stop, or question. There is nothing wrong with profiling as a general practice, but when race becomes a factor in a profile, serious constitutional and ethical issues arise.

Racial profiling is the use of racial generalizations or stereotypes as a basis for stopping, searching, or questioning an individual. Racial profiling received a great deal of attention in the United States in the late 1990s as a result of a series of prominent incidents and the release of data on police practices from several jurisdictions. The data consistently showed that African Americans and Hispanics are disproportionately targeted by law enforcement for stops, frisks, and searches. Court records showed, for example, that in Maryland African Americans made up 70 percent of those stopped and searched by the Maryland State Police from January 1995 through December 1997, on a road on which 17.5 percent of the drivers and speeders were African American. A 1999 report by the New Jersey Attorney General found that 77 percent of those stopped and searched on New Jersey highways are African American or Hispanic, even though, according to one expert, only 13.5 percent of the drivers and 15 percent of the speeders on those highways are African American or Hispanic. An *Orlando Sentinel* analysis of 1,000 videotapes of Florida state trooper traffic stops in 1992 showed that on a road where 5 percent of the drivers were African American or Hispanic, 70 percent of those stopped and 80 percent of those searched by the Florida state police were African American or Hispanic.

Racial targeting need not be expressly invited by a profile. Consider, for example, the U.S. Drug Enforcement Agency's (DEA) drug courier profile for airports. All the factors listed below have been identified by DEA agents in court testimony as part of the DEA's drug courier profile:

arrived late at night
arrived early in the morning
arrived in afternoon
one of first to deplane
one of last to deplane
deplaned in the middle
bought coach ticket
bought first-class ticket
used one-way ticket
use round-trip ticket
paid for ticket with small denomination currency

paid for ticket with large denomination currency
made local telephone call after deplaning
made long-distance telephone call after deplaning
pretended to make telephone call
traveled from New York to Los Angeles
traveled to Houston
carried no luggage
carried brand-new luggage
carried a small bag
carried a medium-sized bag
carried two bulky garment bags
carried two heavy suitcases
carried four pieces of luggage
overly protective of luggage
disassociated self from luggage
traveled alone
traveled with a companion
acted too nervous
acted too calm
made eye contact with officer
avoided making eye contact with officer
wore expensive clothing and gold jewelry
dressed casually
went to restroom after deplaning
walked quickly through airport
walked slowly through airport
walked aimlessly through airport
left airport by taxi
left airport by limousine
left airport by private car
left airport by hotel courtesy van
suspect was Hispanic
suspect was African-American female

Even without the last two factors, this profile describes so many travelers that it does not so much focus an investigation as provide DEA officials a ready-made excuse for stopping whomever they please. A Lexis review of all federal court decisions from January 1, 1990 to August 2, 1995, in which drug courier profiles were used and the race of the suspect was discernible, revealed that of sixty-three such cases, all but three suspects were minorities: thirty-four were African-American, twenty-five were Hispanic, one was Asian, and three were white. While this is not a scientific sampling—it does not include cases in which the race of the suspect could not be discerned, and it does not include cases that did not result in judicial decisions (either because there was no arrest or indictment, or because the defendant pleaded guilty)—the statistics are so one-sided as to raise serious questions about racial targeting.

Although statistical data alone do not conclusively establish that officers are engaged in "racial profiling," they provide strong circumstantial evidence. Many police officers, moreover, admit that all other things being equal, they are more suspicious of, for example, young African-American men than elderly white women. Nor is such thinking irrational. Criminologists generally agree that young African-American men are more likely to commit crime than elderly white women, because at least with respect to some crime, young people commit more crime than old people, men commit more crime than women, and African Americans commit more crime than whites. Indeed, it is precisely because the use of race as a generalization is not irrational that racial profiling is such a widespread phenomenon.

In some areas, however, there is evidence that the use of racial profiles is irrational. The strongest evidence is with respect to drug law enforcement. Much of the racial profiling that occurs on the nation's highways is conducted for drug law enforcement purposes. Officers use the pretext of a traffic infraction to stop a car and then ask for consent to search the car for drugs. This tactic has been expressly approved by the U.S. Supreme Court.

Yet studies show that officers get virtually the same "hit rates" for whites and African Americans when they conduct traffic stops for drugs. In other words, officers are no more likely to find drugs on an African-American driver than a white driver. Consistent with these results, the U.S. Public Health Service has found, based on confidential self-report surveys, that African Americans and whites use illegal drugs in rough proportion to their representation in the population at large. In 1992, for example, 76 percent of illegal drug users were white and 14 percent were African American. Since most users report having purchased drugs from a dealer of the same race, drug dealing is also likely to be fairly evenly represented demographically. Thus, the supposition that African Americans are more likely to be carrying drugs is sharply contradicted by the data.

In any event, even where demographic data suggests that the practice of racial profiling may not be irrational, it is both unconstitutional and unwise.

Because of the pernicious history of racial classifications in the United States, the Supreme Court forbids official reliance on racial generalizations—even accurate ones—except when there is no other way to achieve a compelling government end. The usual argument police officers advance in defense of profiling is that it recognizes the unfortunate fact that minorities are more likely than whites to commit crime. But while this may be true with respect to *some* crimes, the generalizations are hopelessly overinclusive even as to those crimes. The fact that African Americans are more likely than whites to engage in violent crime, for example, does not mean that most African Americans commit violent crime. Most African Americans, like most whites, do not commit any crime; annually, at least 90 percent of African Americans are not arrested for anything. On any given day, the number of innocent African Americans is even higher. In addition, when officers focus on minorities, they lose sight of white criminals. Race is a terribly inaccurate indicator of crime.

Most important, relying on race as a factor for suspicion violates the first principle of criminal law: individual responsibility. The state's authority to take its citizens' liberty, and in extreme cases, lives, turns on the premise that all are equal before the law. Racial generalizations fail to treat people as individuals. As a result, policies that tolerate racial profiling undermine the criminal law's legitimacy. As any good leader knows, and many criminologists have confirmed, legitimacy is central to getting people to follow the rules. If people believe in the legitimacy and fairness of the system, they are much more likely to abide by the rules than if they see the system as unjust. Thus, racial profiling may indeed contribute to crime by corroding the legitimacy of the criminal law.

Efforts to halt racial profiling are now in place in many American jurisdictions. In 1999, President Clinton ordered all federal agencies to study their law enforcement practices to root out racial profiling, and several states and cities—including North Carolina, Connecticut, Florida, Houston, and San Diego—have required reporting on the racial patterns of law enforcement. Such reporting is the first step toward ending the practice, because as long as records of police practices are neither kept nor made public, the nature and extent of the problem will be hidden. The second step requires clear statements by law enforcement officials stipulating that racial profiling is impermissible: Precisely because racial profiling is deeply embedded in the culture and not always irrational, police officers are likely to continue to do it unless the practice is clearly prohibited. And the third step will require effective monitoring and discipline. It remains to be seen whether racial profiling can be halted effectively.

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DAVID D. COLE

RATIONAL AUTHORITY Drug addicts are reported to have a low tolerance for ANXIETY. As a result, few are able to voluntarily sustain an extended period of drug treatment, which is necessary for meaningful intervention. Instead, they tend to disengage themselves from treatment programs once the anxiety has been brought to the surface (Brill & Lieberman, 1969). "Rational authority," a late 1960s euphemism for mandatory (but not necessarily punitive) treatment, became a basis for holding addicts in a long-term treatment program.

The philosophy behind rational authority justifies the development of coercive mechanisms or strategies that permit assigning to treatment those addicts who ordinarily would not voluntarily seek assistance. Rehabilitation programs based upon this philosophy derive their legitimate coercive powers through the *authority* of the courts. The authority is considered *rational* because it is utilized in a humane and constructive manner, and it does this by relating the means of authority to the ends of rehabilitation.

This conceptualization represents an evolutionary change from the emphasis on the use of authority as a punitive end in itself. Rational authority also suggests combining the authority of the probation or parole officer with the techniques of social

casework. As such, authority becomes a means for the officer or associated rehabilitation worker to implement desired behavioral changes. In addition to being required to obey the usual conditions of probation, addicts can be involuntarily held in a therapeutic setting until they have acquired a tolerance for abstinence and the conditioning processes thought to maintain addiction have been reversed. Evaluations of programs in New York, California, and Pennsylvania that are based upon rational authority indicate that when addicts are thus supervised, they are often less likely to relapse into addictive behavior (Brill & Lieberman, 1969).

(SEE ALSO: *California Civil Commitment Program; Civil Commitment; Coerced Treatment for Substance Offenders; Contingency Management; New York State Civil Commitment Program; Treatment Alternatives to Street Crime Treatment/Treatment Types*)

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HARRY K. WEXLER

RATIONAL RECOVERY (RR) Rational Recovery (RR) is one of a number of self-help movements that have emerged as alternatives to ALCOHOLICS ANONYMOUS (AA) for those with drug and alcohol problems. Rational Recovery began with the publication of *Rational Recovery from Alcoholism: The Small Book* by Jack Trimpey in 1988. The program is based on Rational Emotive Therapy, a mental-health treatment with a cognitive orientation developed by the psychologist Albert Ellis. It is premised on the assumption that psychological difficulties are caused by irrational beliefs that can be understood and overcome, not by existential or spiritual deficits. The emphasis is on rational self-examination rather than on religiosity.

An RR "coordinator" leads a group of five to ten members, who meet once or twice weekly for ninety minutes. Each coordinator maintains contact with an adviser, a mental-health professional familiar with the RR program. RR emphasizes cognitive devices for securing abstinence, such as discussion of "the Beast," a term used to personify the compulsive thoughts that drive an individual to drink. Members use a "Sobriety Spreadsheet" on which they write out irrational beliefs that activate their desire to drink. They also read Trimpey's *The Small Book* to develop the proper attitude toward abstinence. These devices are used in RR meetings as well as outside to examine vulnerability to drinking and to overcome it. At meetings these issues are also addressed in a less formal way in "cross-talk," an open, face-to-face exchange among participants.

RR differs from AA in that it does not encourage supportive exchanges and phone calls between meetings, nor does the enrollee solicit a sponsor among established members. Also in contrast to AA, there is no equivalent of "working" the TWELVE STEPS, and a spiritual or religious orientation to treatment is explicitly eschewed. Like SECULAR ORGANIZATIONS FOR SOBRIETY (SOS), RR encourages study of its methods and outcome. One such study by Galanter and coworkers sent follow-up questionnaires to seventy RR groups in nineteen states and received sixty-three responses. Ninety-seven percent of participants in the responding groups filled out questionnaires. They were mostly men about forty-five years old, each with about a twenty-five-year history of alcohol problems. The majority were employed, had attended college, and had heard about the program through the media or by word of mouth. A majority had used marijuana, a substantial minority had also used cocaine, and a small minority had used heroin.

At the time of the study (the early 1990s), RR was a much younger organization than AA. Most of the coordinators had been members for only nine months, most groups had been meeting for about a year, and the implementation of the movement's specific techniques (use of the Sobriety Spreadsheet and discussion of "the Beast") was not consistent. Nevertheless, the members' commitment to the central tenet of the movement, sobriety, was considerable. Although 75 percent had previously attended AA meetings, the majority (82%) rated RR

principles higher than AA principles in helping them achieve sobriety. However, it seems quite likely that RR benefits considerably from the experience these former AA members bring with them. A sizable percentage of RR participants who returned questionnaires were involved with mental-health care as well as with RR. Thirty-six percent had seen a psychotherapist the week before the survey, and 21 percent were currently taking medication prescribed for psychiatric problems. Many group coordinators had formal mental-health training, and 24 percent had graduate degrees or certificates in mental health. It is likely that, just as AA derives some legitimacy from its spiritual roots, RR derives some of its influence from the credibility of the professional psychology with which it is associated. Without carefully controlled studies that adjust for differences in patient backgrounds, it is hazardous to compare outcome studies from RR to studies of AA and other self-help groups. The data that do exist, however, tentatively suggest that RR may do at least as well.

An RR group can be formed at no cost by a recovering substance abuser in consultation with the executive office of the Rational Recovery movement (Box 800, Lotus, California 95651).

(SEE ALSO: *Sobriety*; *Treatment Types: Self-Help and Anonymous Groups*)

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MARC GALANTER

RAVE A rave is a large, typically overnight dance party with a focus on techno and related forms of music. The rave provides a venue for innovative musical forms and fashions as well as for the use and abuse of a variety of drugs known collectively as CLUB DRUGS. Raves and the "ravers" who attend them have been a part of youth culture since the late 1980s when all-night parties and Detroit techno music sprang up in the United Kingdom to form the phenomenon that is still a social concern today. Raves are held in a variety of locales, from traditional nightclubs to warehouses to open pastures (sometimes without the knowledge of the owners). A major part of the attraction of raves is the permissive, underground atmosphere. Ravers, who are more often than not in their late teens and early twenties, enjoy the freedom from supervision that is common at raves.

Dancers take to the crowded, smoky dance floor at an all-night rave at Groove Jet in Miami Beach, September 24, 1999. (AP Photo/Greg Smith)

Hedonism or "pleasure seeking" is also of central value in rave culture, and this correlates with a high incidence of drug use. Many ravers freely admit to the presence of various club drugs on the rave scene, particularly METHAMPHETAMINE (meth, crank, crystal, speed or whizz) and MDMA (E, X, ecstasy, or rolls) although others such as ROHYPNOL, GHB, LSD, and KETAMINE have recently gained more attention in the media as club drugs. In truth, polydrug abuse is common enough on the rave scene that no list of drugs can be regarded as comprehensive. Ravers tend to regard the drugs they use as newer and safer than "older" drugs like HEROIN and PCP. This is rarely true insofar as safety is concerned. Raves have certainly seen their share of drug casualties, and are cause for concern because of the high incidence of drug problems among ravers.

RICHARD G. HUNTER

RECEPTOR, DRUG A receptor is a molecular site, specific for a drug or its class, with which the drug must combine to produce its effect. If a drug is in the body but cannot bind to the receptor, then there is no effect. A receptor can be thought of as the button or switch that the drug must activate in order to produce a physiologic effect.

Receptors for drugs are the same receptors used in the brain by naturally occurring compounds referred to as neurotransmitters. NEUROTRANSMITTERS are chemical signaling messengers in the brain that work by binding to specific receptors; a wide variety of drugs of abuse bind to these same receptors. In this sense, drugs of abuse insert themselves into natural and normal systems found in the brain take over normal pathways in abnormal ways. Receptors are essential for normal functioning of the body and are, therefore, of great interest and importance in physiology and medicine.

Receptors can be stimulated by compounds called AGONISTS, or blocked by compounds called ANTAGONISTS. Antagonists prevent the action of agonists. For example, NALTREXONE, an antagonist, will prevent MORPHINE, an agonist, from having any effect.

A major achievement of research in drug abuse over the past thirty years has been the identification and study of almost all receptors for drugs of abuse. Receptors are generally classified into two types: an ion channel type and a coupled type receptor or "G protein". NICOTINE acts at one of the former and morphine at one of the latter. However, sometimes the initial molecular site that a drug acts at is not one of these two classical types of receptors. For example, COCAINE acts at another kind of molecule called a transporter for DOPAMINE; after cocaine binds at this site, dopamine transport in the brain is blocked, which then results in increased actions at the dopamine receptor. Since receptors are the initial, molecular sites of binding of drugs, they are clearly of interest in understanding how drugs produce their effects and how we might develop medications for drug abuse treatments.

NICK E. GOEDERS
REVISED BY MICHAEL J. KUHAR

RECEPTOR: NMDA (N-METHYL D-ASPARTIC ACID) The NMDA receptor is a protein on the surface of neurons (nerve cells). When the major excitatory NEUROTRANSMITTER, GLUTAMATE, binds to this protein, the central pore of the NMDA receptor channel opens—then cations (the ions of sodium, potassium, and calcium) are able to cross the cell membrane. The movement of cations through the pore results in neuronal excitation.

The NMDA receptor is one of several cell receptor surface proteins activated by glutamate. The HALLUCINOGEN PHENCYCLIDINE (PCP) blocks the open channel of the NMDA receptor preventing cation flow. It is believed that overactivation of the NMDA receptor could be responsible for the neuronal cell death observed following some forms of stroke; it may even be involved in the cell death associated with neurodegenerative diseases.

(SEE ALSO: *Neurotransmission; Receptor; Drug*)

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RECIDIVISM *See* Relapse; Relapse Prevention

RECREATIONAL DRUG USE *See* Addiction: Concepts and Definitions; Policy Alternatives; Safer Use of Drugs

REINFORCEMENT Although the term reinforcement has many common uses and associated meanings, its meaning is precise when used by behavior analysts and behavior therapists. The act or process of making a reinforcer contingent on behavior is termed positive reinforcement, and a reinforcer is any object or event that, when delivered following some behavior, *increases* the probability that the behavior will occur again. A typical example might evolve from a laboratory experiment with

rats. A rat is placed in a small plastic chamber. The rat can press a lever located on one wall of the chamber. When the rat presses the lever, a small food pellet drops into a dish. If the rat returns to the lever and continues to press it would be said that the food pellet functions as a reinforcer that the behavior is maintained by positive reinforcement.

There is often confusion between positive reinforcement and negative reinforcement. Negative reinforcement occurs when a behavior results in terminating an aversive stimulus. In the case of the rat, the negative stimulus might be a loud noise. A lever press turns off the stimulus. If the rat continues to press the lever, it would be said that loud noise functions as a negative reinforcer and the behavior is maintained by negative reinforcement. Thus, both positive and negative reinforcement refer to increases in behavior, but differ in whether a pleasant stimulus is presented as the result of some behavior (positive reinforcement). Negative reinforcement is also referred to as escape (if the response turns off the stimulus each time it appears) or avoidance (if the response can postpone presentation of the stimulus).

It is important to note that reinforcement is a concept that refers to the relationship between behavior and its consequences. Stimuli or events are not assumed to have inherent reinforcing effects. For example, although most people like money and will continue to exhibit behavior that results in obtaining money, it cannot be assumed that money functions as a reinforcer for everyone. For example, money might not serve as a reinforcer for a monk devoted to an ascetic lifestyle. The defining characteristic of reinforcement depends on how a behavior is changed and not on the types of things that serve as reinforcing events (Morse & Kelleher, 1977). Factors that help determine whether a given object or event is reinforcing or punishing for a given individual include that individual's previous experiences and other features of the environment that coexist and are associated with the object or event. The upshot is that different things may function as reinforcers for different people.

DRUGS can serve as reinforcers that maintain drug-seeking and drug-taking behaviors. This can be observed in the prevalence of drug use among humans and has also been shown in laboratory research with animals. In a typical laboratory experiment, the animal such as a rat or monkey has a catheter placed in a vein and connected to a pump-driven syringe. The animal can press a lever to activate the pump, and this results in a dose of a drug such as COCAINE, HEROIN, NICOTINE, or ALCOHOL being infused into the vein. If the animal continues to press the lever to obtain the drug, then the drug is said to serve as a reinforcer. Interestingly, those drugs which lead to ADDICTION in humans also serve as reinforcers in animals. The only exception is MARIJUANA (THC),which is used fairly extensively by humans but does not function as a reinforcer in animals. It should be noted that drugs that serve as reinforcers under one condition may not serve as reinforcers under other conditions. For example, nicotine serves as a reinforcer only at low doses and when doses are properly spaced. Nevertheless, the observation that drugs of abuse generally function as reinforcers in experimental animals has brought the study of drug-seeking behavior and drug abuse into a framework that allows carefully controlled behavioral analyses and the application of well-established and objective behavioral principles (Schuster & Johanson, 1981).

The acquisition of drug use in humans predominantly involves positive reinforcement, whereas the maintenance of drug use can involve both positive and negative reinforcement. The ability of a drug to serve as a positive reinforcer is usually associated with its pleasurable subjective effects (e.g. a "rush", a "high", or other feelings of intoxication). But again, given the definition of reinforcement, it is not necessary for a drug to be subjectively reinforcing or pleasurable in order for it to maintain behavior. Many drugs are also associated with symptoms of WITHDRAWAL when abstinence is initiated following a period of regular use. In this case, taking the drug again may terminate the aversive state of withdrawal; in this way, drug use is maintained by negative reinforcement. Drug use can also be influenced by sources of reinforcement other than the direct effects of the drug. For example, social encouragement and praise from a peer group can play an important role in the development of drug use by teenagers. Biological factors may also come into play. For example, some individuals may be more or less susceptible than others to feeling and recognizing the pleasurable effects of drugs. When drug use is viewed as a behavior maintained by the reinforcing effects of drugs, it suggests that this behavior is not amoral or uncontrolled but rather that it is the result of normal behavioral processes.

(SEE ALSO: *Addiction: Concepts and Definitions: Causes of Substance Abuse: Learning; Research, Animal Model: Intercranial Self-Stimulation; Wikler's Pharmacologic Theory of Drug Addiction*)

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RELAPSE An individual who has recovered from an illness or has entered a period of stability in a chronic illness and who subsequently suffers a recurrence of symptoms is said to have experienced a relapse. In the addictions, there has been some controversy over whether the term relapse can be used to indicate any use following a period of abstinence, or whether it should be reserved for more significant episodes of substance use that might indicate a return to problematic use or in some cases dependence. At the present time, there is some consensus in the field that the term *lapse* should be used for minor episodes of use following a period of abstinence, whereas *relapse* should be used to connote major episodes of use, such as drinking five or more drinks on two or more consecutive days.

Among the addictions, rates of relapse are relatively high among individuals who achieve abstinence with or without formal treatment. For example, up to 60 percent of alcoholics, heroin addicts, and smokers relapse within three months of the end of treatment. Although relapse episodes are common, most substance abusers do experience substantial reductions in the frequency and severity of use for extended periods after treatment. Addictions are now thought to be chronic, relapsing disorders in which afflicted individuals cycle through periods of heavy use, treatment, abstinence or reduced use, and relapse.

A number of models have been proposed to explain the relapse process. One of the more influential and widely accepted of these is the cognitive-behavioral model. According to this model, individuals experience an increased risk of relapse when they encounter so-called *high-risk* situations, which are situations that have been associated with substance use in the past. The model postulates that one of two processes occurs when a substance abuser encounters a high-risk situation. If the individual has high self-efficacy, or the belief that he or she can manage the situation without using alcohol or drugs (i.e., relapsing), a coping response is performed and relapse is avoided. However, if the individual has lower self-efficacy, a coping response is not performed and relapse ensues. Therefore, in this model relapse is seen largely as a function of whether one (1) encounters high-risk situations, and (2) is able to mount an effective coping response. Other cognitive features of the model include outcome expectancies (i.e., what will happen as a result of either substance use or the exercise of a coping behavior) and attributions for one's behavior.

Related models of relapse, which encompass enduring personal characteristics and background variables, in addition to immediate precipitants and coping responses, have also been proposed. According to these models, individuals with characteristics such as a family history of substance abuse, concurrent psychiatric problems, and more severe substance-use histories are at increased risk for relapse during periods of abstinence. Risk for relapse is further increased by factors such as major life events, protracted life stressors, low social support, and low motivation for self-improvement. When individuals with these characteristics encounter a high-risk situation, they are less likely to be able to mount an effective coping response.

Other models of relapse place much less emphasis on conscious, cognitive processes. For example, one classical conditioning model proposes that sudden urges to use, or cravings, are triggered when an individual encounters a situation or experience that has been frequently paired with substance use in the past. For example, a former substance abuser might suddenly experience craving for cocaine when he encounters someone with whom he used to smoke cocaine. Another model postulates that relapses are frequently governed by ingrained, automatic processes that occur below the level of conscious thought. This might explain why in some cases, substance abusers appear to have very little insight into the factors that led them to relapse. A

third model is focused on the importance of WITHDRAWAL symptoms in the onset of relapse. This last model would seem to better account for relapses that occur within a few days of the onset of abstinence than relapses that occur after months of abstinence. However, there is some evidence that individuals who have been abstinent for significant periods of time could have experiences that trigger the onset of withdrawal-like feelings through classical conditioning processes described above.

Although the models briefly described here tend to focus on particular factors or mechanisms that are hypothesized within each model to play important roles in relapse, it is widely believed that the process of relapse is actually determined by a host of factors, including motivation, mood states, craving, and coping behaviors, as well as other cognitive, biological, and interpersonal factors. Moreover, individuals probably differ with regard to the relative importance of various factors in the onset of their relapse episodes. It is also possible that the processes which bring about relapses that occur relatively quickly differ to some degree from those that lead to relapse after long periods of abstinence or nonproblematic use.

One of the problems in developing a valid model of the relapse process is that it is very difficult to study. It is usually not possible to interview or observe substance abusers immediately prior to relapse, so researchers have often had to rely on accounts of events leading up to relapse gathered at some point after the episode to obtain information on relapse precipitants. Unfortunately, there is considerable evidence that retrospective reports such as these can be inaccurate or biased because substance abusers are either unaware of what brought on a relapse or their memory is distorted. Recently, researchers studying NICOTINE relapse have begun to use palm-sized, portable computers to systematically record in near real time information about the mood states, cognitions, and situations that smokers experience, and to link these factors to the onset of smoking relapse, which are also recorded on the computers. It is not clear whether this new technology will work adequately with abusers of other substances, such as ALCOHOL and COCAINE. Final determinations of the validity of various models of relapse will likely have to await the development of better technologies with which to study the process.

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RELIGION AND DRUG USE Drug use and religion have been intertwined throughout history, but the nature of this relationship has varied over time and from place to place. Alcohol and other drugs have played important roles in the religious rituals of numerous groups. For example, among a number of native South American groups, TOBACCO was considered sacred and was used in religious ritual, including the consultation of spirits and the initiation of religious leaders. Similarly, wine, representing the blood of Christ, has been central in the Holy Communion observances of both Roman Catholic and some Protestant churches. Considered divine by the Aztecs of ancient Mexico, the PEYOTE cactus (which contains a number of psychoactive substances, including the psychedelic drug MESCALINE) is used today in the religious services of the contemporary Native American church (Goode, 1984).

Although tobacco, ALCOHOL, peyote, and other drugs have been important in the religious observances and practices of numerous groups, many religious teachings have opposed either casual use or the abuse of psychoactive drugs—and some religious groups forbid any use of such drugs, for religious purposes or otherwise. Early in America's history, Protestant religious groups were especially prominent in the TEMPERANCE MOVEMENT. Many of the ministers preached against the evils of drunkenness, and well-known Protestant leaders, such as John Wesley, called for the prohibition of all alcoholic beverages (Cahalan, 1987). The Latter-day Saints' (Mormons) leader Joseph Smith prohibited the use of all common drugs, including

alcohol, tobacco, and caffeine (no coffee or tea), as did other utopian groups founded during the Great Awakening of the early 1800s. Religious groups and individuals were also active in America's early (1860s–1880s) antismoking movement (U.S. Department of Health and Human Services, 1992). In contemporary American society, certain religious commitments continue to be a strong predictor of either use or abstinence from drugs, whether licit or illicit (Cochran et al., 1988; Gorsuch, 1988; Payne et al., 1991). For example, Islam forbids alcohol and opium use but coffee, tea, tobacco, khat, and various forms of marijuana were not prohibited, because they came into the Islamic world after the prohibitions were laid down. Indulgence in any debilitating substance is, however, not considered proper or productive. Christianity, Judaism, and Buddhism may not prohibit specific drugs, but they and most other widespread, mainstream religious traditions also caution against indulgence in most substances. In our society, many who have indulged have sought the help of ALCOHOLICS ANONYMOUS (AA) or NARCOTICS ANONYMOUS (NA)—both self-help groups founded on strong spiritual underpinnings.

This discussion is limited to recent conditions in the United States, focusing on potentially dangerous, abusive, and/or illicit patterns of drug use. Since such drug use is widely disapproved by most religious teachings and leaders, it is not surprising to find that those with strong religious commitments are less likely to be drug users or abusers. Moreover, research findings clearly show that religious involvement has been a protective factor, helping some adolescents resist the drug epidemics of the 1970s and 80s.

Because religion has been found to be a protective factor against drug use and dependence and because our society is concerned with drug use among young people, much of the research linking religion with drug use focuses on adolescents and young adults. This age range is particularly important for several reasons. First, it is the period during which most addiction to NICOTINE begins; the majority of people who make it through their teens as nonsmokers do not take up the habit during their twenties or later (Bachman et al., 1997). Second, ADOLESCENCE and young adulthood is the period during which abusive alcohol consumption is most widespread. Third, recent EPIDEMICS in the use of illicit drugs have been most pronounced among teenagers and young adults. Fourth, during this portion of the life span, many changes, opportunities, and risks occur; thus, the structures and guidelines provided by religious commitment may be especially important in helping young people resist the temptation to use and abuse drugs. Finally, evidence that religious conversion is most likely to occur during adolescence (Spilka, 1991) makes this period particularly appropriate for research on the link between religion and drug use.

THE RELATIONSHIP BETWEEN RELIGIOUS COMMITMENT AND DRUG USE

Research investigating the relationship between religious commitment and drug use consistently indicates that those young people who are seriously involved in religion are more likely to abstain from drug use than those who are not; moreover, among users, religious youth are less likely than nonreligious youth to use drugs heavily (Gorsuch, 1988; Lorch & Hughes, 1985; Payne et al., 1991).

Examples from 1979, 1989, and 1999. Figure 1 shows how drug use was related to religious commitment among high school seniors in 1979, 1989, and 1999. Individuals with the highest religious commitment were defined as those who usually attend services once a week or more often and who describe religion as being very important in their lives; individuals with low commitment are those who never attend services and rate religion as not important. Figure 1 clearly indicates that those with low religious involvement were more likely than average to be frequent cigarette smokers, occasional heavy drinkers, and users of MARIJUANA and COCAINE; conversely, those highest in religious commitment were much less likely to engage in any of these behaviors. Other analyses have shown that similar relationships exist for other illicit drugs (Bachman et al., 1986) and for other age groups (Cochran et al., 1988; Gorsuch, 1988).

Recent Trends in Drug Use and Religious Commitment. Figure 1 presents data from three points in time, separated by ten-year intervals. It is obvious in the illustration that between 1979 and 1989, the proportion of high school seniors using the illicit drugs marijuana and cocaine declined markedly; also during that decade, the proportion reporting instances of heavy drinking declined appreciably, as did the proportion of frequent

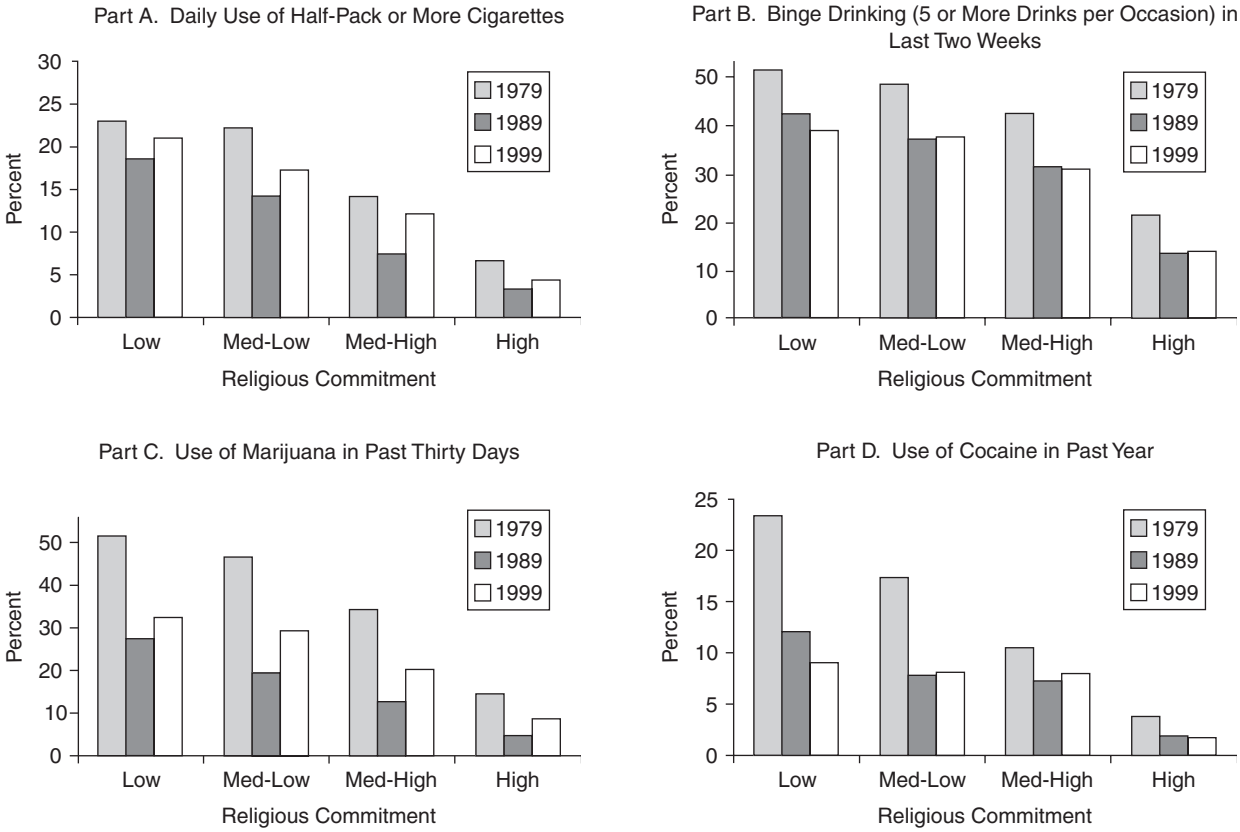

Figure 1
Drug use among high school seniors shown separately for four levels of religious commitment: 1979, 1989, and 1999.

smokers. Between 1989 and 1999, the proportion of cigarette users and marijuana users rose somewhat; for year-to-year changes in substance use, see Johnston et al. (2000). For the present purposes, the most important finding in Figure 1 is that religion was linked to drug use at all three times, although the relationships appear a bit more dramatic during periods of heavier use.

Because high religious commitment is associated with low likelihood of drug use, it is reasonable to ask whether any of the decline in illicit drug use during the 1980s could be attributed to a heightened religious commitment among young people during that period. The answer is clearly negative, as illustrated in Figure 2. The same annual surveys that showed declines in drug use also indicated that religious commitment, rather than rising during the 1980s, was actually declining among high school seniors. It thus appears that other factors accounted for the declines in illicit drug use, factors such as the increasing levels of risk and the heightened disapproval associated with such behaviors (Bachman et al., 1988, 1990; Johnston, 1985; Johnston et al., 2000). Moreover, Figure 2 shows that religious commitment—especially ratings of importance—actually rose slightly during the 1990s, so it does not appear that the rise in use of some drugs during the 1990s is attributable to any further drop in religiosity.

Religion as a Protective Factor. The most plausible interpretation of the relationship between religion and drug use during recent years, in our view, is that religion (or the lack thereof) was not primarily responsible for either the increases or the subsequent decreases in illicit drug use. Rather, it appears that those with the strongest religious commitment were least susceptible to the various epidemics in drug use. Figure 3 (adapted from Bach-

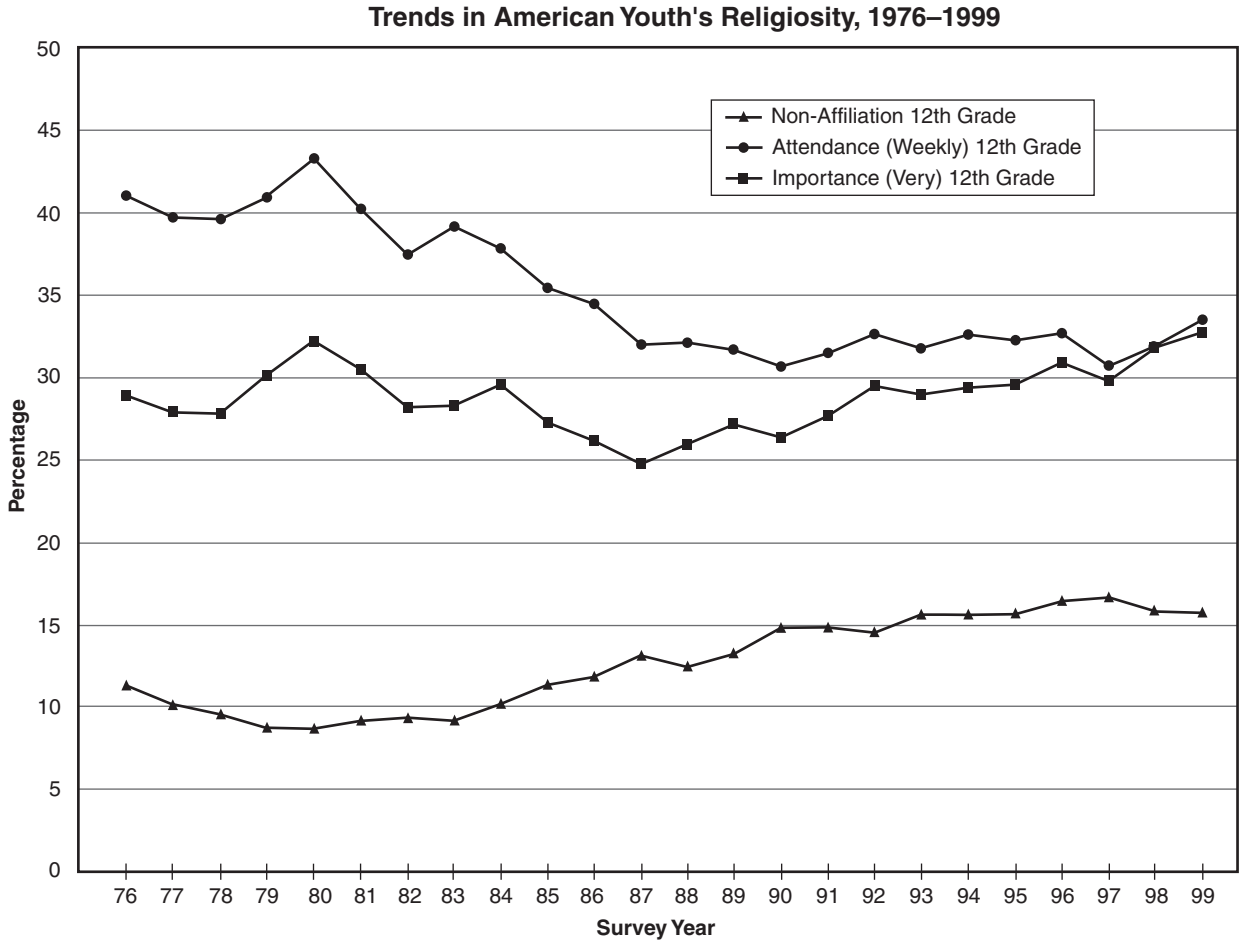

Figure 2
Trends in American youth's religiosity: 1976–1999.

man et al., 1990) provides one example in support of that interpretation. The figure illustrates trends in cocaine use from 1976 through 1988, distinguishing among the four different degrees of religious commitment. Cocaine use roughly doubled between 1976 and 1979 among high school seniors and began to decline sharply after 1986. But the most important pattern in the figure, for the present purposes, is that these historical trends in cocaine use were much more pronounced among those with little or no religious commitment. Put another way, it seems that strong religious commitment operated as a kind of protective factor, sheltering many youths from the waves of drug use sweeping the nation.

Denominational Differences. There are important differences among religious groups in the emphasis placed on drug use (Lorch & Hughes, 1988). In particular, the more fundamentalist Protestant denominations, as well as Latter-Day Saints (Mormons) and African American Muslims, rule out the use of alcohol and tobacco and disdain illicit drug use. Research examining differences in drug use among young people finds that those who belong to fundamentalist denominations are more likely to abstain from drug use than are youth who belong to more liberal denominations (Lorch & Hughes, 1985). Analyses of the data on high school seniors (Wallace & Forman, 1998) corroborate the findings of earlier research; the number of young people strongly committed to fundamentalist denominations (e.g., Baptists) who use drugs is much lower than average and lower than the percentages for

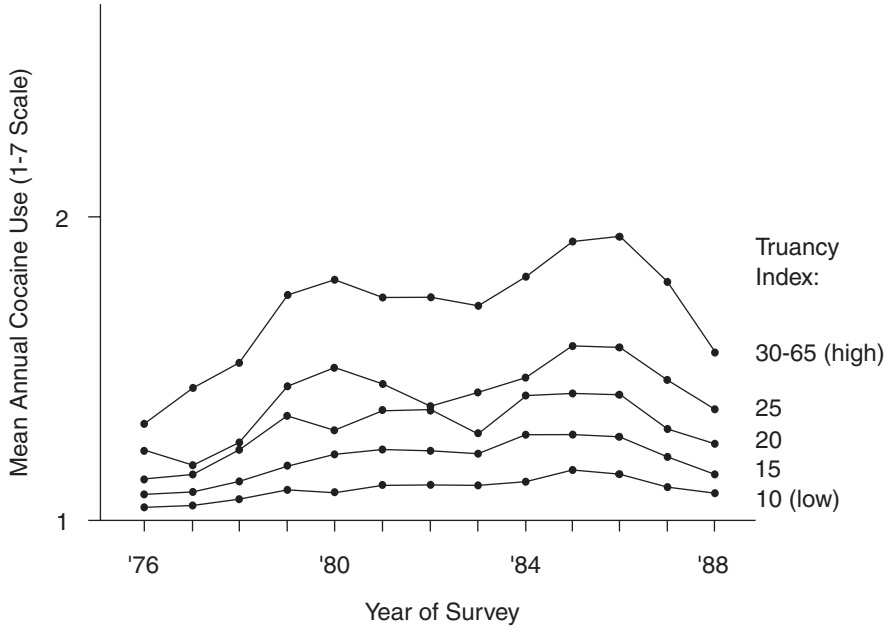

Figure 3
Trends in annual cocaine use shown separately for five levels of truancy, high school seniors: 1976–1988.

those strongly committed to other religious traditions.

Changes During Young Adulthood. Panel surveys that followed high school seniors up to fourteen years after graduation revealed that substance use often increases in response to new freedoms such as leaving high school and moving out of parents' homes, whereas use often decreases in response to new responsibilities such as marriage, pregnancy, and parenthood (Bachman et al., 1997). Additional analyses of these data reveal that religion continues to be strongly related to various forms of drug use during the late teens, twenties, and early thirties. These analyses reveal that religious attendance and importance change rather little for most individuals, but when changes in religiosity occur, there tend to be corresponding changes in substance use. Specifically, increases in religious commitment are correlated with declines in the use of alcohol and illicit drugs. Smoking behavior, on the other hand, is linked with religiosity during high school and thus also during young adulthood. However, after high school, smoking behavior is relatively little affected by changes in religiosity—presumably because by the time of young adulthood, most individuals who continue to smoke have become dependent on nicotine and find it very difficult to quit.

POSSIBLE CAUSAL PROCESSES

Since religious commitment is negatively related to drug use, it becomes important to understand the possible causal processes underlying that relationship. Wallace and Williams' socialization influence model (1997) specifies a number of possible mechanisms through which religious commitment might operate to influence adolescent drug use. The model postulates that health-compromising behaviors like drug use are the result of a dynamic socialization process that begins in childhood and extends throughout the course of life. According to the model, the family is the primary and first socialization influence, and a continuing source of socialization into the norms and values of the larger society. The model hypothesizes that religion, peer networks, and other contexts in which young people find themselves (e.g., schools) operate as key secondary socialization influences that impact drug use, primarily indirectly, through their influence on key socialization mechanisms, including social control, social support, values, and individual and group identity. Below, we describe some of the

ways in which religion, parents, peers, and other potential causes might overlap to influence adolescent drug use. The socialization influence model further suggests that key aspects of adolescent religiosity, particularly denominational affiliation and religious attendance, are often under the control of parents and reflect the types of doctrinal beliefs, teachings, and adult and peer models to which parents want their children exposed.

Content of Religious Teaching. One possible causal process seems obvious: Most religious traditions teach followers to avoid the abuse of drugs. Restrictions vary, of course, from one tradition to another, and the greater emphasis on prohibition in fundamentalist denominations seems the most likely explanation for the lower levels of use among adherents. But even in traditions that do not explicitly or completely ban drug use, there is still much teaching ranging from respect for one's own body to family responsibilities to broader social responsibilities, all arguing against the abuse of drugs. Because all drugs, including cigarettes and alcohol, are illicit for minors, young people who are strongly committed to religion may abstain from drug use simply in obedience to the laws of the nation; but even more important, they are likely to act in obedience to what they perceive to be God's laws.

Parental Examples and Precepts. In addition to the direct teachings associated with attendance at religious services, young people raised in religious traditions are likely to be exposed to parents and other relatives who follow such teachings. Thus, part of the explanation for less drug use among religiously involved young people may be that their families reinforce the religious structures against use and abuse. A further factor may simply be availability; religious parents who do not drink, smoke, or use drugs will not have these substances in their homes, thus reducing the opportunity for young people to experiment with them.

Peer Group Factors. The dynamics operating within the family probably have their parallel in broader social contacts. That is, those who are strongly committed to religion probably associate with others holding similar views. Thus, the strongly religious are less likely to belong to peer groups that encourage experimentation with cigarettes, alcohol, and other drugs and more likely to participate in peer networks and activities that do not involve drugs. Given the strong relationship between drug use by peers and an adolescent's own drug use, the norms of the peer group are especially important as predictors of whether a particular teenager will start using drugs (Jessor & Jessor, 1977).

Overlaps with Other Causes. Religious commitment among young people is correlated with a number of other factors known to relate to drug use. In particular, students who achieve good grades, who plan to go to college, and who are not truant are also less likely to use drugs, as well as more likely to display high levels of religious commitment. These various factors are closely interrelated in a common syndrome (Dryfoos, 1990; Jessor & Jessor, 1977), and thus it is difficult to disentangle causal processes. Indeed, it could be argued that religious commitment is probably one of the root causes, contributing to both educational success and the avoidance of drug use. Analyses of possible multiple causes of drug use (or abstention) have shown that religious commitment overlaps with other predictors, but only partially. In other words, although religious commitment may be part of a larger syndrome, it also appears to have some unique (i.e., nonoverlapping) impact on drug use.

CONCLUSION

The relationship between religion and drug use among young people is not completely straightforward. On the one hand, a considerable amount of research indicates that young people who are strongly committed to religion are less likely than their uncommitted counterparts to use drugs. On the other hand, data presented here and elsewhere suggest that religion has had relatively little impact on recent national declines in drug use among young people. Further examination of this relationship reveals that America's drug epidemic occurred primarily among those not affected by religion; highly religious youth were relatively immune to the plague that infected a significant portion of the nation's youth. Accordingly, we conclude that religious commitment has been, and continues to be, an effective deterrent to the use and abuse of licit and illicit drugs.

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(SEE ALSO: *Ethnic Issues and Cultural Relevance in Treatment; Jews, Drug and Alcohol Use Among; Prevention Movement; Vulnerability: An Overview*)

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JOHN M. WALLACE, JR.

REMOVE INTOXICATED DRIVERS (RIDUSA, INC.) Founded in 1978, this volunteer grass-roots organization (P.O. Box 520, Schenectady, NY 12301; 518–372–0034) is devoted to efforts to deter impaired driving, help victims seek justice and restitution, close loopholes in DWI (driving while impaired) laws, and educate the public on the scope of impaired-driving tragedies. RID activists have played a key role in the passage of reforms of the impaired-driving laws in many

states, enabled passage of more than 500 anti-DWI laws, and monitored more than 15,000 court cases.

RID's victim-support activities, which are free, include providing long-term emotional support to victims of drunk-driving crashes and to their families; counseling victims and accompanying them throughout all phases of criminal prosecution of the offender; assisting victims in obtaining compensation; and referring victims and their families to appropriate supportive agencies. Court monitoring and research activities include monitoring the efforts of police, prosecutors, magistrates, and judges in drunk-driving cases through research and analysis of local court records, and reporting these findings to the public. RID's public awareness and education activities are extensive. Members organize public meetings; present educational talks to community and religious organizations; participate in forums, exhibits, and media events; supplement high school driver-education classes; and support SADD (STUDENTS AGAINST DRIVING DRUNK) and other similar student groups. They study and report on alcohol-related vehicle and traffic laws; support concepts such as designated-driver and alcohol-server education, and promote SNAP (a Sane National Alcohol Policy), which advocates raising taxes on alcohol, curbing campus beer promotions, and airing public-service advertising to counter all broadcast alcohol commercials.

RID is organized into autonomous chapters, with more than 150 chapters in at least forty-one states in the United States and a national group in France. Financial support comes from member dues, government and corporate grants, charitable contributions, and memorial gifts. Information on how to organize a RID chapter is available from the national office in Schenectady, New York.

(SEE ALSO: *Accidents and Injuries from Alcohol, Dramshop Liability Laws; Drunk Driving; Mothers Against Drunk Driving*)

FAITH K. JAFFE

REPEAL OF PROHIBITION *See* Prohibition of Alcohol

RESEARCH This section is devoted primarily to detailed explanations of the ways in which behavioral psychologists and psychopharmacologists explore the interactions between drug actions and behavior in laboratory settings. The section begins with an overview article, *Aims, Description, and Goals.* The article *Developing Medications to Treat Substance Abuse and Dependence* ties basic research directly to clinical applications. The articles on *Drugs as Discriminitive Stimuli; Measuring Effects of Drugs on Behavior; Measuring Effects of Drugs on Mood*; and *Motivation* describe these general research techniques and concepts and their applicability to understanding drug abuses.

Research in the field of drug dependence, however, is much broader and more diverse than the topics included in this section. In fact, research is conducted on most of the topics contained in this encyclopedia—from epidemiological studies to new methods for detecting drug smuggling; from herbicides that can target specific plant sources of illicit drugs to how to target prevention messages to subgroups within the population; from how certain drugs produce their toxic effects to developing new drugs to reduce drug craving or prevent relapse; from how the interactions of environment and genetics make certain individuals more vulnerable to drug use to the relative effectiveness of different treatment programs. Many of these research issues are touched upon in such diverse articles as those on controlling illicit drug supply; on TREATMENT; or PREVENTION; and on VULNERABILITY AS A CAUSE OF SUBSTANCE ABUSE.

Clinical, behavioral, epidemiological, and basic research is carried out primarily by researchers at universities, government research centers, and research institutes. It is funded both publicly and privately. The work of a representative few of these centers is described elsewhere in the encyclopedia (see *Addiction Research Foundation (Canada); Addiction Research Unit (U.K.); Center on Addiction and Substance Abuse (CASA); Rutgers Center of Alcohol Studies; U.S. Government/U.S. Government Agencies (SAMHSA, NIAAA, NIDA, CSAP, CSAT)*. In 1992, worldwide, there were more than eighty research centers devoted to problems of drugs and alcohol. Fifty-eight of the centers were in the United States; thirteen were in Europe and the U.K.; the others were in Central and South America, Asia, Australia, and New Zealand.

For more information on research, see also *Imaging Techniques: Visualizing the Living Brain;*

Pain: Behavioral Methods for Measuring the Analgesic Effects of Drugs; Research, Animal Models.

Aims, Description, and Goals In a Chinese book on pharmacy, which dates to 2732 B.C., references are found to the properties of MARIJUANA (a type of Old World HEMP, *Cannabis sativa* of the mulberry family). In an Egyptian papyrus from about 1550 B.C., there is a description of the effects of OPIUM (a product of the opium poppy, *Papaver somniferum*). In almost every culture, the uses of ALCOHOL are documented in both oral and written tradition, often going back into antiquity—the Bible, for example, mentions both the use and abuse of wine. Although people have made observations on PSYCHOACTIVE substances for thousands of years, much remains to be learned about both alcohol and drugs of abuse; much research remains to be done before these substances and their effects can be fully understood.

WHAT WE NEED TO KNOW

Most substance-abuse research carried out today is a consequence of public health and social concerns. With millions of people using and abusing many different substances, and because of the close association between AIDS and drug abuse, it is imperative to know just how dangerous—or not dangerous—any given drug is to public health and safety. For economic as well as medical reasons, it is essential to find the most effective ways to use our health resources for preventing and treating substance abuse. So many questions still exist that no one scientific discipline can answer them all. The answers must be found through studies in basic chemistry, molecular biology, genetics, pharmacology, neuroscience, biomedicine, physiology, behavior, epidemiology, psychology, economics, social policy, and even international relations.

From a social standpoint, the first question for research must be: How extensive is the problem? Surveys and other indicators of drug and alcohol usage are the tools used by epidemiologists to determine the extent and nature of the problem, or to find out how many people are abusing exactly which drugs, how often, and where. As the dimensions of the problem are defined, basic scientists begin their work, trying to discover the causes and effects of substance abuse at every level, from the movement of molecules to the behavior of entire human cultures. Chemists determine the physical structure of abused substances, and then molecular biologists try to determine exactly how they interact with the subcellular structures of the human body. Geneticists try to determine what components, if any, of substance abuse are genetically linked. Pharmacologists determine how the body breaks down abused substances and sends them to different sites for storage or elimination. Neuroscientists examine the effects of drugs and alcohol on the cells and larger anatomical structures of the brain and other parts of the nervous system. Since these structures control our thoughts, emotions, learning, and perception, psychologists and behavioral pharmacologists study the drugs' effects on their functions. Cardiologists and liver and pulmonary specialists study the responses of heart, liver, and lungs to drugs and alcohol. Immunologists examine the consequences of substance abuse for the immune system, a study made critical by the AIDS epidemic. The conclusions reached through these basic scientific inquiries guide clinicians in developing effective treatment programs.

In considering drug abuse, people have long wondered why so many plants contain substances that have such profound effects on the human brain and mind. Surely people were not equipped by nature with special places on their nerve cells (called RECEPTORS) for substances of abuse—on the off chance that they would eventually smoke marijuana or take COCAINE or HEROIN. The discovery in the late 1960s that animals would work to obtain injections or drinks of the same drugs that people abuse was an important scientific observation; it contributed to the hypothesis that there must be a biological basis for substance abuse. These observations and this reasoning led scientists to look for substances produced by people's own bodies (endogenous substances) that behave chemically and physiologically like those people put into themselves from the outside (exogenous substances)—like alcohol, NICOTINE, marijuana, cocaine, and other drugs of abuse. When receptors for endogenous substances were discovered—first for the OPIATES in the 1970s and only recently for PCP, cocaine, marijuana, and LSD—their existence helped establish the biological basis for drug abuse. So did the discovery of a genetic component for certain types of ALCOHOLISM. These discoveries by no means negate the extensive behavioral and so-

cial components of substance abuse, but they do suggest a new weapon in dealing with the problem—that is, the possibility of using medication, or a biological therapy, as an adjunct to psychosocial therapies. Asserting a biological basis for substance abuse also removes some of the social stigma attached to drug and alcohol addiction. Since drug dependence is a disorder with strong biological components, society begins to understand that it is not merely the result of weak moral fiber.

Armed with information that was derived from basic research, clinical researchers in hospitals and clinics test and compare treatment modalities, looking for the best balance of pharmacological and psychosocial methods for reclaiming shattered lives. Finding the right approach for each type of patient is an important goal of treatment research, since patients frequently have a number of physical and mental problems besides substance abuse. The development of new medications to assist in the treatment process is an exciting and complex new frontier in substance-abuse research.

The best way to prevent the health and social problems that are associated with substance abuse has always been a significant research question. Insights gained from psychological and social research enable us to design effective prevention programs targeted toward specific populations that are particularly vulnerable to substance abuse for both biomedical and social reasons. Knowing the consequences of substance abuse often helps researchers to formulate prevention messages. For example, the identification of the FETAL ALCOHOL SYNDROME (FAS), a pattern of birth defects among children of mothers who drank heavily during pregnancy, was a major research contribution to the prevention of alcohol abuse. Drug-abuse-prevention research has assumed a new urgency with the realization, brought about by epidemiologists and others, that the AIDS virus is blood-borne—spread by sexual contact and by drug abusers who share contaminated syringes and needles. HIV-positive drug users then spread the disease through unprotected sexual intercourse. Public education about drug abuse and AIDS must use the most powerful and carefully targeted means of reaching the populations at greatest risk for either disease, and these means can be determined only by the most careful social research and evaluation methodologies.

Substance-abuse research is no different from any other sort of scientific endeavor: The process is not always orderly. Critical observations by clinicians frequently provide basic researchers with important insights, which guide the research into new channels. Observations in one science often lead to breakthroughs in other areas.

METHODS

The range of methods employed by scientists studying substance abuse is as wide as the range of methods in all the biological and social sciences. One important method is the use of animal models of behavior to answer many of the questions raised by drug and alcohol use. Animal models are used in biomedical research in virtually every field, but the discovery that animals will, for the most part, self-administer alcohol and the same drugs of abuse that humans do, meant that there was a great potential for behavioral research uncontaminated by many of the difficult-to-control social components of human research. The results of animal studies have been verified repeatedly in human research and in clinical observation, thus validating this animal model of human drug-seeking behavior.

Research Personnel. Drug- and alcohol-abuse research is conducted by many different types of qualified professionals, but mostly by medical researchers (MDs) and people with advanced degrees (PhDs) in the previously mentioned sciences. They work with animals and with patients in university and federally funded laboratories, as well as in privately funded research facilities, in offices, and in clinical treatment centers. Other sites include hospitals, clinics, and sometimes schools, the streets, and even advertising agencies when prevention research is under way.

FUNDING

Who pays for substance-abuse research has always been an important issue. In the late 1980s and early 1990s, most of the drug- and alcohol-abuse research in the world was supported by the U.S. government. One of the federally funded National Institutes of Health—the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA)—funds over 88 percent of drug-abuse research conducted in the United States and abroad. In 1992, this amounted to over 362 million dollars, which supported NIDA's own intramural research at the Addiction Research Center and the research done in universities under grants

awarded by the institute. NIDA's sister institute, the NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM (NIAAA), plays a parallel role in funding alcohol-abuse research. In 1992, it funded 175 million dollars in alcohol-research grants. Many other U.S. government agencies also have important roles in sponsoring and conducting substance-abuse research. For the most part, state and local governments do not sponsor substance-abuse research, although they do much of the distribution of funds for treatment and prevention programs.

Other countries, most notably Canada, sponsor basic clinical and epidemiological substance-abuse research within their own universities and laboratories, but none does so on a scale that is comparable to that of the United States. Private foundations and research institutions like the Salk Institute for Biological Studies, Rockefeller University, and the Scripps Clinic and Research Foundation use their own funds, as well as federal grant support, to pay for their research endeavors. Pharmaceutical companies also support some substance-abuse research—mostly clinical work related to medications that might be used as part of treatment programs for drug and alcohol abuse. Again, much of this work is sponsored, in part, by the U.S. government.

(SEE ALSO: *National Household Survey; Substance Abuse and HIV/AIDS; Research, Animal Model; U.S. Government/U.S. Government Agencies*)

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CHRISTINE R. HARTEL

Clinical Research In the process of developing new drugs, pharmaceutical companies must perform rigorous studies in the laboratory, in animals, and then, if the drug looks promising, in humans. Carefully designed research into the safety and effectiveness of a drug in humans is called CLINICAL RESEARCH (or CLINICAL TRIALS). Research resulting from new surgical techniques, medical devices, and other medical treatments also fall under this heading.

To conduct research in humans, approval must be obtained from the Food and Drug Administration (FDA). The research sponsors (usually the pharmaceutical company) submit a detailed application termed an *Investigational New Drug Application* that summarizes the drug characteristics, manufacturing process, and results of any laboratory and animal studies. In addition, this application provides detailed information regarding proposed studies in humans, including the research protocol, data collection documents, and informed consent form. If the drug is proven to be safe and effective, the sponsors can submit a voluminous application called a *New Drug Application* to the Food and Drug Administration. This application contains the material in the Investigational New Drug Application as well as the data, analyses, and conclusions of all of the clinical trials conducted.

Clinical trials of drugs or medical devices progress through four phases. Phase I studies are conducted on healthy volunteers to assess the safety of the drug or device. Phase II studies are conducted on a relatively small group of patients with the target disease to assess effectiveness as well as safety. Phase III studies are conducted on a large group of patients with the target disease to confirm effectiveness, observe side effects, and to compare the test treatment to the standard treatment. Phase IV studies are performed for a variety of reasons after the drug or device has been on the market. Reasons for conducting phase IV studies include: to test the treatment in different populations (e.g., in children or the elderly), to assess the effects of long-

term use of the treatment, or to use the treatment on a different target disease.

STUDY DESIGN

Study design is a crucial determinant of the strength, validity, and subsequent usefulness of clinical research results. Study design is the methodology used to conduct the clinical research. Many different types of clinical research studies exist. The strength of the data depends upon the conditions used during the conduct of the trial. Also, these conditions help to eliminate bias by the investigator, patient, or others who are involved in the collection and analysis of the data. The most important conditions are blinding, randomization, and controlling. The randomized, controlled, double blind study is considered to be the clinical research ideal.

Blinding refers to the process in which the patient does not know whether he or she is receiving the test treatment or a placebo treatment. In the single blind design, the patient does not know which treatment he or she is receiving. The investigator knows, however, and this may lead to bias. Ideally, studies should be double blind, a condition in which neither the patient nor any of the other people who are actively involved in the study have knowledge of the treatment.

Randomization refers to the process in which the patients are randomly assigned to the various treatments. This insures that the test treatment and controls are allocated to the patient by chance, and not the choice of the investigator. Randomization eliminates the possibility that an investigator could sway study results.

Clinical research studies can be either controlled or uncontrolled. Controls can be either the standard treatment for the target disease (*active controlled*) or a placebo (*vehicle controlled*). Many diseases have a natural tendency to wax and wane so study results can be misleading without a control group to serve as a comparator to the treatment group. Because controlled studies are a more reliable indicator of a treatment's effectiveness, uncontrolled studies are considered as preliminary or suggestive, or they may be disregarded altogether.

Another important component of study design is the determination of the sample size, or number of patients to include in the study. A sample size that is too small will yield a study in which the results are not strong enough (not statistically significant) to prove that the test treatment is effective. The sample size is based upon, among other things, the number of treatment and control groups in the study and an estimation of the expected differences between these groups.

The study design is contained within the study protocol, which is a detailed document that outlines every aspect of the study. The protocol is essentially a set of rules that the investigator(s) must follow. It covers such things as who may be entered into the study, how to collect and record data, and how to record and report adverse reactions. Violation of any of the rules set forth in the protocol can disqualify an investigator, a patient, or even the entire study.

Although the randomized, controlled, single and double blind studies are very common designs, there are other study designs which may be used. The sponsor may initially conduct dose-finding studies in order to find the optimal dose of a test drug to treat the target disease. In the cross-over design, patients receive both treatments being compared (or treatment and a placebo) which factors out inter-individual variability. Each patient would receive one treatment for a designated time period, their disease state would be evaluated, and then they would switch to the other treatment for a designated time period. Other, more complex study designs are also employed. However, with increasing complexity comes increasing difficulties in data analysis, interpretation, and validity.

ETHICAL CONSIDERATIONS

Federal regulations insure that the rights of subjects in a clinical trial are protected. Each clinical trial must be approved and monitored by a committee known as an Institutional Review Board, which has medical, scientific, and non-scientific members. Institutional Review Boards review and approve trial documents such as the protocol and informed consent form as well as the advertising materials needed to attract subjects. The purpose of the Institutional Review Board is to protect the rights, safety, and well-being of the study subjects.

The Food and Drug Administration requires that all participants in a clinical trial be informed of the details of the study. This process is called *informed consent*. Informed consent usually involves a lengthy document (informed consent form) that

describes key facts about the study including: the purpose of the research, what the goals are, what procedures will be done, what the possible risks are, what the possible benefits are, and what other treatments are available for the target disease. In addition, the informed consent form stresses that the subject can leave the study at any time. An important component of the informed consent process is that the subject has the opportunity to ask questions regarding the study and/or the consent form.

CONCLUSION

Clinical research plays an invaluable role in the ongoing process of finding effective and safe treatments for diseases. The information obtained by clinical trials provides physicians with the necessary information to make informed choices in the treatment of their patients. Clinical studies are key in identifying the optimal doses of a new drug and also in providing information regarding the occurrence and incidence of adverse reactions. However, clinical research is limited by sample size. Even studies comprised of thousands of subjects will fail to pick up extremely rare, possibly serious adverse reactions that materialize during clinical use.

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BELINDA ROWLAND

Developing Medications to Treat Substance Abuse and Dependence Dependence on drugs, ALCOHOL, or TOBACCO is difficult to treat, and practitioners have tried many approaches in their attempts to arrive at successful treatments. One approach is to develop medications, or pharmacological treatments. This approach is most effective when the medication is given along with behavioral treatments. These behavioral treatments help the individual cope with the underlying etiology of his or her drug use and the problems associated with drug use; they may also help ensure compliance in taking the medication that is prescribed.

PERPETUATION OF DRUG ABUSE: EUPHORIA AND WITHDRAWAL

Many people who are drug- or alcohol-dependent want to stop their habit, but often they have a difficult time doing so. There are at least two reasons for this difficulty. First, the drugs produce pleasant or euphoric feelings that the user wants to experience again and again. Second, unpleasant effects can occur when the drug use is stopped. The latter effect, commonly known as WITHDRAWAL, has been shown after prolonged use of many drugs, including alcohol, OPIATES (such as HEROIN), SEDATIVE HYPNOTICS, and anxiety-reducing drugs. Other drugs, such as COCAINE and even CAFFEINE (COFFEE and COLA drinks) and NICOTINE (cigarettes), are also believed to be associated with withdrawal effects after prolonged use. These unpleasant withdrawal effects are alleviated by further drug use. Thus drugs are used and abused because they produce immediate pleasant effects and because the drug reduces the discomfort of withdrawal.

The symptoms of withdrawal are fairly specific for each drug and include physiological effects and psychological effects. For example, alcohol withdrawal can be associated with shaking or headaches, and opiate withdrawal with anxiety, sweating, and increases in blood pressure, among other effects. Withdrawal from cocaine may cause depression or sadness, withdrawal from caffeine is associated with headaches, and withdrawal from nicotine often produces irritability. All drug withdrawals are also associated with a strong craving to use more drugs. Much work has been done to document the withdrawal effects from alcohol, opiates, BENZODIAZEPINES, and tobacco; however, documentation of withdrawal from cocaine or other stimulant drugs has only recently begun to be examined.

NEURAL CHANGES WITH CHRONIC DRUG USE

Both withdrawal and the pleasant or euphoric effects from drug use occur, in part, as a result of the drug's action on the brain. The immediate or

acute effects of most drugs of abuse affect areas of the brain that have been associated with "reward" or pleasure. These drugs stimulate areas normally aroused by natural pleasures such as eating or sexual activity. Long-term, or chronic, drug use alters these and other brain areas. Some brain areas will develop TOLERANCE to the drug effects, so that greater and greater amounts are needed to achieve the original effects of the drug. Some examples of drug effects that develop tolerance are the ANALGESIC or painkilling effect of opiates and the euphoria- or pleasure-producing effect of most drugs of abuse, which are probably related to their abuse potential.

Because some brain areas may also become sensitized, an original drug effect will either require a lesser amount of the drug to elicit the effect when the drug is used chronically or the effect becomes greater with chronic use. This phenomenon has been studied most extensively in cocaine use. Cocaine is associated with behavioral sensitization of motor activity in animals and paranoia (extreme delusional fear) in humans. There are physiological effects that develop tolerance or sensitization as well. For example, the chronic use of cocaine will sensitize some brain areas so that seizures are more easily induced. Other health risks of drug use will be addressed below.

In addition to these more direct acute and chronic drug effects, another phenomenon occurs with long-term drug use. This phenomenon is the *conditioned drug effect*, in which the environmental or internal (mood states) cues commonly presented with drug use become conditioned or psychologically associated with drug use. For example, when angry, a drug addict may buy or use drugs in a certain place with certain people. After frequently taking drugs under similar conditions, the individual can experience a strong craving or even withdrawal when in the environment in which he or she has taken drugs or feels angry. When the individual tries to stop using drugs, exposure to these conditioned cues can often lead to relapse because the craving and withdrawal effects are so powerful. Very little research has been done on the neural bases of these conditioned effects; thus it is not known whether these effects are mediated by similar or different neural mechanisms.

RESEARCH ON DRUG EFFECTS

Many of these acute and chronic effects of drugs on the brain have been investigated in animal research, which allows greater control over the research, including manipulations of drug exposure. A number of animal models are used to assess drug preferences, and, since most drugs that humans abuse are also preferred by animals, these models are useful for understanding human drug abuse. Moreover, animal research allows scientists to study directly the various areas of the brain that are involved in drug use. In addition, recent technological advancements on noninvasive IMAGING have allowed scientists to took at pictures of the brains of humans while they are being administered drugs or while they are withdrawing from drugs. This human work has also enhanced our knowledge of the drug effects on the brain as well as validated the information gained from animal research.

Another useful line of research in assessing the effects of drugs involves human laboratory studies. In one type of study, research volunteers who have had experience with the abused drugs are given a specific drug (e.g., morphine), and various psychological and physiological measurements are obtained. The psychological measurements can include reports from the subject on the effects of the drug as well as more sophisticated behavioral measures that tell the experimenter how much the drug is preferred. Another type of human laboratory study is to study the effects of drug withdrawal. For opiates, withdrawal can be precipitated by an opiate ANTAGONIST drug (NALTREXONE), and withdrawal signs and symptoms are measured. For other drugs (such as cocaine), withdrawal is more difficult to measure because little is known about their withdrawal syndromes.

Some of the information that scientists have learned from such studies includes delineating specific brain areas as well as the NEUROTRANSMITTERS (the chemicals released by the brain cells) involved in drug use and withdrawal. Thus, when specific neurotransmitters become identified as playing an important role in drug use or withdrawal, scientists can administer experimental drugs that act on these neurotransmitters to see if the animals will alter their drug preference or show less severe withdrawal signs. Researchers can also give these experimental drugs to the human research volunteers to see if the medication alters the subject's perception

of or behavior toward the abused drug or if it alleviates withdrawal symptoms. If the results from these animal and human laboratory studies are promising, then these agents can be tested on treatment-seeking, drug-dependent individuals in clinical trials. This latter type of research is more time-consuming and expensive than the laboratory studies, but it helps provide an answer to the ultimate question: Does this medication help an individual stop abusing drugs?

APPROACHES TO DEVELOPING MEDICATIONS FOR DRUG ABUSE

Researchers can use the knowledge gained from animal and human studies of the effects of drugs on the brain as they develop medications for alcohol and drug dependence. Most likely, one medication will be needed to help detoxify the drug-dependent individual and a second medication to help sustain abstinence from drug use. This two-phase medication regimen is used for opiate and alcohol treatment, and it may ultimately be the approach used for countering dependency on other drugs, such as cocaine, sedatives, and nicotine. In theory, a pharmacological treatment agent or medication would block or reduce either the acute, rewarding effect of the drug or the discomfort of withdrawal. In practice, few treatment drugs have been found to be very effective in sustaining abstinence from drugs or alcohol.

Any pharmacological agent should be able to be given orally, as this is much easier than other routes of administration, such as injections. The agent itself must be medically safe and not enhance any of the health risks associated with illicit drug use, since the individual may illicitly use drugs while being maintained on the treatment agent. Finally, the pharmacological treatment agent must be acceptable to the patient. That is, if the agent causes undesirable side effects, individuals will likely not take it.

Current research with alcohol and drug effects on the brain and with treatment outcome hold great promise for effective pharmacological agents. This search process will necessarily include the animal and human laboratory studies mentioned as well as medicinal chemistry research. Medicinal chemistry research is used to develop new compounds that have similar but slightly altered chemical structures to the abused drugs or to the neurotransmitters that mediate the drug or alcohol effects. These new compounds are then tested in animals to see if they produce therapeutic effects. These effects include having a low potential for being another drug of abuse and attenuating the effects of the abused drug under study, preferably in a way that would lead to decreased drug abuse.

EXAMPLES OF MEDICATIONS USED TO TREAT DRUG ABUSE

Several types of medications have been developed for countering various kinds of dependencies.

Opioid Dependence. Some of the best examples of pharmacotherapies for drug abuse were developed for opiate addicts. One of the first pharmacological agents used to treat opiate addicts is METHADONE. Methadone itself is an opiate drug and effectively reduces or blocks the withdrawal discomfort brought on by discontinuing use of heroin or other illegal opiate. Although methadone is itself addictive, it is delivered to the opiate-dependent patients in a facility with psychological and other medical and support treatments and services. Methadone is safer than opiates obtained illegally—in part because it is given orally. Because illegal opiates are often injected by addicts, they can lead to many diseases—including AIDS and hepatitis, if the needles are shared with an infected person. Illegal drug use is expensive, and many addicts steal to support their habit. Moreover, since drugs obtained illegally vary in their quality and purity, there is a greater chance of getting an overdose that produces severe medical problems and, perhaps, death. Thus methadone decreases the need to use illegal opiates, as a result of its ability to relieve withdrawal as well as to block the effects of other opiates by cross-tolerance. Moreover, it reduces the health risks and social problems associated with illegal opiate use.

Another treatment drug that was developed for opiate dependence and abuse is naltrexone. This agent blocks the ability of the opiate drug to act on the brain. Thus, if heroin addict maintained on naltrexone injects heroin, he or she will not feel the pleasant or other effects of the heroin. The principle behind this approach is based on research suggesting that drug use is continued, despite the dire consequences, because of the euphoria associated with its use. Once maintained on naltrexone, the addict may forget this association, because the drug

can no longer produce these effects. Unfortunately, although naltrexone works well for some, others will simply discontinue using the naltrexone in order to get high from drugs again.

Before opiate abusers can be maintained on the medication naltrexone, they must be detoxified from the opiate drugs in their systems. Although abstaining ("cold turkey") from heroin use for several days will accomplish detoxification, the withdrawal process is difficult because of the physical distress it causes. Thus, another DETOXIFICATION method was developed in which the withdrawal is precipitated, or triggered, with naltrexone, while the symptoms are treated with another medication, CLONIDINE. When withdrawal is precipitated, the symptoms are worse than that seen with natural withdrawal, but the symptom course is much briefer. Moreover, clonidine helps alleviate the symptoms, to make this shorter-term withdrawal process less severe.

Alcohol Dependence. An example of another type of medication is one used to treat alcoholism: DISULFIRAM. The basis for this agent's therapeutic effect is different from that of methadone or naltrexone. When someone is maintained on disulfiram, future alcohol ingestion will cause stomach distress and, possibly, vomiting, because the disulfiram prevents the breakdown of a noxious alcohol metabolite by the liver. Patients maintained on disulfiram should come to forget the pleasant effects of alcohol use, which is similar to the psychological basis of naltrexone maintenance. Moreover, they should begin to develop an aversion to alcohol use. Another similarity to the use of naltrexone is that disulfiram treatment of alcoholism has not been very successful, because the patient who wants to use alcohol again can simply stop using the disulfiram.

Some pharmacological agents have been tested to reduce craving for alcohol and thus help the alcoholic abstain from drinking. These drugs include naltrexone, which was developed for opiate addicts, and fluoxetine. The former medication is a potential treatment drug because most drugs of abuse are believed to be mediated, in part, through the brain's natural opiate system (ENDORPHINS, etc.). Based on research that implicates a specific neurotransmitter system (SEROTONIN) in alcohol craving, the latter medication and others of this type may be useful. However, as in the treatment of opiate abuse, alcoholics must be detoxified before any of these medications are used as maintenance agents.

Tobacco Dependence. One commonly used pharmacological treatment for tobacco dependence is NICOTINE GUM (Nicorette). The main reason to quit smoking is that it is linked to lung cancer, emphysema, and other serious illnesses. Yet the active ingredient in cigarettes, NICOTINE, is associated with pleasant effects and with withdrawal discomfort, thereby making it an extremely addicting drug. Providing smokers with nicotine replacement in the form of a gum will help them avoid the health risks associated with cigarettes. One problem with nicotine gum is that it is difficult to chew correctly; people need to be shown how to chew it in order to get the therapeutic effect. A patch is also available that is placed on the arm and automatically releases nicotine. This method shows good treatment potential. Detoxification from nicotine may also be facilitated with the medication clonidine, the same agent used to help alleviate opiate withdrawal symptoms.

Stimulant Dependence. Developing pharmacological treatment agents for stimulant (e.g., cocaine) dependence is a difficult task but has been the focus of a great deal of research. One of the difficulties for treating cocaine abuse is that cocaine affects many different neurotransmitter systems in various ways. Thus one approach may be to develop a treatment drug or regimen of drugs that affects a variety of neurotransmitter systems. However, the exact nature of the neural effects of cocaine are still not entirely understood.

Another difficulty is that it is not clear what approach to take in developing a treatment drug. One obvious technique in developing a medication for cocaine abuse is to use an agent that blocks the rewarding aspects of cocaine use. This type of drug would, presumably, decrease cocaine use because the rewarding effects are no longer experienced. However, this approach is similar to having opiate addicts use naltrexone, which has not been well accepted by heroin addicts. Clinical work with some treatment agents that were suggested to block the rewarding effects of cocaine did not prove to be useful in the treatment of abuse and dependence. Whether this lack of treatment effect resulted from a flaw in the method or from the limitations in our knowledge of cocaine's effects on neurotransmitter systems is not clear. One problem is that the poten-

tial blocking agents for cocaine may produce dysphoria, or an unpleasant feeling.

Another approach to treating cocaine abuse and dependence is based on a premise similar to that of methadone for opiate abuse. That is, a pharmacological agent similar in its effects to cocaine, but one that is not addicting, may be a useful anticraving agent. Just as methadone helps alleviate drug withdrawal, an agent of this type for cocaine abuse may alleviate the distress and craving associated with abstinence from cocaine. Several medications of this type have been tried, including bromocriptine and AMANTADINE. Thus far, these and other agents have shown some limited treatment promise.

Most of the approaches to developing pharmacological treatments for cocaine abuse have been based on research suggesting that one specific neurotransmitter (DOPAMINE) is important for cocaine's rewarding effects. Yet other neurotransmitters are activated during cocaine use and may be better targets for developing new treatment drugs. That is, although dopamine is critical for the rewarding aspects of cocaine use, other neurotransmitter systems may be more important in withdrawal distress. Although withdrawal distress from cocaine has been difficult to document, depression is thought to be one aspect of abstaining from chronic cocaine use. Antidepressant medications, such as desipramine and imipramine, have shown some, albeit limited, treatment potential.

Sedative Dependence. Current treatments for sedative dependence include detoxification agents, not anticraving agents. Detoxification is accomplished by tapering the dosage of BENZODIAZEPINES over two to three weeks. More recently, carbamazepine, an antiseizure analgesic medication, has been shown to relieve alcohol and sedative withdrawal symptoms, including seizures and delirium tremens. Future work with agents that block the actions of benzodiazepines may hold promise as a maintenance or anticraving agent to help the sedative abuser abstain from drug abuse.

CONCLUSION

One of the greatest lessons learned from the practice of giving medications to drug-abusing individuals is that these medications must be accompanied by psychological and social treatments and support. Medications do not work on their own. Moreover, medications that are developed based on theoretical principles of altering or blocking the drug's effects in the brain may not be useful in the practice of treating drug abuse and dependence, because the premises of how to develop a pharmacological treatment agent may not be accurate. Yet the largest research challenge is to understand the etiology and mechanisms of drug abuse. Thus more research in many fields is needed to identify potential medications in order to develop more effective treatments for the difficult problem of drug abuse and dependence.

(SEE ALSO: *Addiction: Concepts and Definitions; Imaging Techniques: Visualizing the Living Brain; Treatmeat/Treatment Types*)

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THERESE A. KOSTEN

Drugs as Discriminative Stimuli Human behavior is influenced by numerous stimuli in the environment. Those stimuli acquire behavioral control when certain behavioral consequences occur in their presence. As a result, a particular behavioral response becomes more or less likely to occur when those stimuli are present. For example, several laboratory experiments have demonstrated that it is possible to increase a particular response during a stimulus (such as a distinctively colored light) by arranging for reinforcement (such as a preferred food or drink) to be given following that

response when the stimulus is present; when that stimulus is absent, however, responses do not produce the reinforcer. Over a period of time, responding will then occur when the stimulus is present but not when it is absent. Stimuli that govern behavior in this manner are termed *discriminative stimuli* and have been widely used in behavioral and pharmacological research to better understand how behavior is controlled by various stimuli, and how those stimuli, in turn, might affect the activity of various drugs.

It is important to recognize that there are differences between discriminative stimuli that merely set the occasion for a response to be reinforced and other types of stimuli that directly *produce* or *elicit* responses. Discriminative stimuli do not coerce a response from the individual in the same way that a stimulus such as a sharp pierce evokes a reflexive withdrawal response. Instead, discriminative stimuli may be seen as providing guidance to behavior because of the unique history of reinforcement that has occurred in their presence.

DRUGS AS DISCRIMINATIVE STIMULI

Although the stimuli that typically govern behavior are external (i.e., located in the environment outside the skin), it is also possible for internal or subjective stimuli to influence behavior. One of the more popular methods to emerge in the field of behavioral pharmacology has been the use of drugs as discriminative stimuli. The procedure consists of establishing a drug as the stimulus, in the presence of which a particular response is reinforced. Typically, to establish a drug as a discriminative stimulus, a single dose of a drug is selected and, following its administration, one of two responses are reinforced; with rodents or nonhuman primates, this usually entails pressing one of two simultaneously available levers, with reinforcement being scheduled intermittently after a fixed number of correct responses. Alternatively, when saline or a placebo is administered, responses on the other device are reinforced. Over a number of experimental sessions, a discrimination develops between the administration of the drug and saline, with the interoceptive (subjective) stimuli produced by the drug seen as guiding or controlling behavior in much the same manner as any external stimulus, such as a visual or auditory stimulus. Once the discrimination has been established, as indicated by the selection of the appropriate response following either the training drug or the saline administration, it is possible to investigate aspects of the drug stimulus in the same way as one might investigate other physical stimuli. It is thus possible to determine gradients of intensity or dose-effect functions with the training drug as well as generalization functions aimed at determining how similar the training drug dose is to a different dose or to another drug substituted for the training stimulus.

BASIC EXPERIMENTAL RESULTS

One of the more striking aspects of the drug discrimination technique is the strong relationship that has been found between the stimulus-generalization profile and the receptor-binding characteristics of the training drug. For example, animals trained to discriminate between a BENZODIAZEPINE anxiolytic, such as CHLORDIAZEPOXIDE, and saline solution typically respond similarly to other drugs that also interact with the receptor sites for benzodiazepine ligands. Anxiolytic drugs that produce their effects through other brain mechanisms or receptors do not engender responses similar to those occasioned by benzodiazepines. This suggests that it is activity at a specific RECEPTOR that is established when this technique is used and not the action of the drug on a hypothetical psychological construct such as anxiety (Barrett & Gleeson, 1991).

Several studies have examined the effects of drugs of abuse by using the drug discrimination procedure, and they have established COCAINE and numerous other drugs—such as an OPIATE, PHENCYCLIDINE (PCP), or MARIJUANA—as a discriminative stimulus in an effort to help delineate the neuropharmacological or brain mechanisms that contribute to the subjective and abuse-liability effects of these drugs. As an example, Figure 1 shows the results obtained in pigeons trained to discriminate a 1.7 milligram per kilogram (mg/kg) dose of cocaine from saline. The dose-response function demonstrates that doses below the training dose of cocaine yielded a diminished percentage of responses on the key correlated with cocaine administration, which suggests that the lower doses of cocaine were less discernible than the training dose. In addition, other psychomotor stimulants such as AMPHETAMINE and METHAMPHETAMINE also produced cocaine-like responses, and this suggests

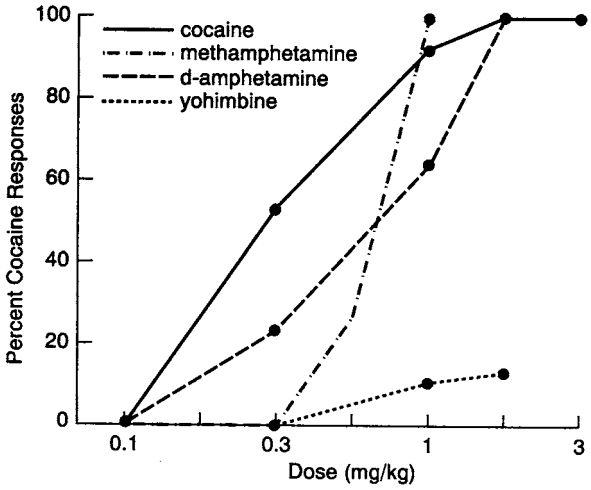

Figure 1
Discriminative Stimuli. Effects of establishing a dose of 1.7 mg/kg cocaine, administered intramuscularly, as a discriminative stimulus in pigeons. Following the administration of the training dose of cocaine, 30 consecutive pecks on one illuminated response key resulted in food reinforcement, whereas following the administration of saline, 30 consecutive pecks on a different key produced food. Once the discrimination was established, various doses of other drugs were substituted for cocaine. The discriminative stimulus effects of cocaine were dose-dependent, with doses from 0.1 to 1.7 producing increases in responding on the key correlated with the training dose of cocaine. Similarly, d-amphetamine and methamphetamine also resulted in responding on the cocaine key, thereby showing that these drugs have some of the same subjective stimulus properties and presumably neuropharmacological effects as cocaine. A drug that does not produce generalization, yohimbine, an α2-adrenoreceptor antagonist, resulted only in modest levels of responding on the cocaine-associated response key, which suggests that this is not a mechanism by which cocaine produces its subjective behavioral and pharmacological effects.
SOURCE: Adapted from Johnson & Barrett, 1993.

that these drugs share some of the neurochemical properties of cocaine. In contrast, other drugs, such as the α_2-adrenoreceptor antagonist yohimbine, along with several other drugs such as morphine, PCP, or marijuana (that are not illustrated) do not produce responding on the key correlated with cocaine administration—thereby suggesting that the mechanisms of action underlying those drugs, as well as their subjective effects, are not similar to those of cocaine and the other psychomotor stimulants in this figure.

IMPLICATIONS

The use of drugs as discriminative stimuli has provided a wealth of information on the way drugs are similar to more conventional environmental stimuli in their ability to control and modify behavior. The procedure has also increased our understanding of the neuropharmacological mechanisms that operate to produce the constellation of effects associated with those drugs. The technique has wide generality and has been studied in several species, including humans—in whom the effects are quite similar to those of nonhumans.

Because it is believed that the subjective effects of a drug are critical to its abuse potential, the study of drugs of abuse as discriminative stimuli takes on added significance. A better understanding of the effects of drugs of abuse as pharmacologically subjective stimuli provides a means by which to evaluate possible pharmacological as well as behavioral approaches to the treatment of drug abuse. For example, a drug that prevents or antagonizes the discriminative-stimulus effects (and presumably the neuropharmacological actions) of an abused drug might be an effective medication to permit individuals to diminish their intake of abused drugs, because the stimuli usually associated with its effects will no longer occur. Similarly, although little work has been performed on the manipulation of environmental stimuli correlated with the drug stimulus, it might be possible to design innovative treatment strategies in which other stimuli compete with the subjective discriminative-stimulus effects of the abused drug. Thus, a basic experimental procedure such as drug discrimination has provided a useful experimental tool for understanding the behavioral and neuropharmacological effects of abused drugs.

Further work may help design and implement novel treatment approaches to modifying the behavioral and environmental conditions surrounding the effects of abused drugs and thus result in diminished behavioral control by substances of abuse.

(SEE ALSO: *Abuse Liability of Drugs; Drug Types; Research, Animal Model*)

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Measuring Effects of Drugs on Behavior

People throughout world take drugs such as HEROIN, COCAINE, and ALCOHOL because these drugs alter behavior. For example, cocaine alters general activity levels; it increases wakefulness and decreases the amount of food an individual eats. Heroin produces drowsiness, relief from pain, and a general feeling of pleasure. Alcohol's effects include relaxation, increased social interactions, marked sedation, and impaired motor function. For the most part, the scientific investigations of the ways drugs alter behavior began in the 1950s, when chlorpromazine was introduced as a treatment for SCHIZOPHRENIA. As a result of this discovery, scientists became interested in the development of new medications to treat behavioral disorders as well as in the development of procedures for studying behavior in the laboratory.

HOW IS BEHAVIOR STUDIED?

The simplest way to study the effects of drugs on behavior is to pick a behavior, give a drug, and observe what happens. Although this approach sounds very easy, the study of a drug's effect on behavior is not so simple. Like any other scientific inquiry, research in this area requires careful description of the behaviors being examined. If the behavior is not carefully described, it is difficult to determine whether a change in behavior following drug administration is actually due to the drug.

Behavior is best defined by describing how it is measured. By specifying how to measure a behavior, an *operational definition* of that behavior is developed. For example, to study the way in which a drug alters food intake, the following procedure might be used: First, select several people and present each with a box of cereal, a bowl, a spoon, and some milk after they wake up in the morning. Then measure how much cereal and milk they each consume within the next thirty minutes. To make sure the measurements are correct, repeat the observations several times under the same conditions (i.e., at the same time of day, with the same foods available). From these observations, determine the average amount of milk and cereal consumed by each person. This is the baseline level. Once the baseline level is known, give a small amount of drug and measure changes in the amount of milk and cereal consumed. Repeat the experiment, using increasing amounts of the drug. This concept of baseline level and change from baseline level is common to many scientific investigations.

In addition to defining behavior by describing how it is measured, a good behavioral procedure is also (1) sensitive to the ways in which drugs alter behavior and (2) is reliable. Sensitivity refers to whether a particular behavior is easily changed as the result of drug administration. For example, food consumption may be altered by using cocaine, but other behaviors may not be. Reliability refers to whether a drug produces the same effect each time it is taken. In order to say that cocaine reliably alters the amount of food consumed, it should decrease food consumption each time it is given, provided that the experimental conditions surrounding its administration are the same.

WHAT FACTORS INFLUENCE A DRUG'S EFFECTS ON BEHAVIOR?

Although good behavioral procedures are necessary for understanding a drug's effects on behavior, pharmacological factors are also important determinants of a drug's effect. Pharmacological factors include the amount of drug given (the *dose*), how

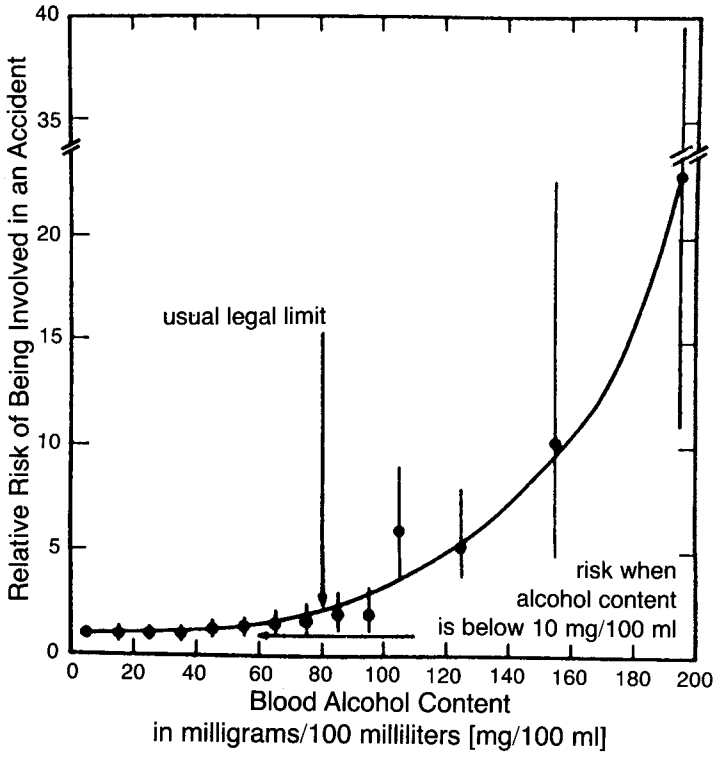

Figure 1
Risk of Being Involved in a Traffic Accident as a Function of the Amount of Alcohol in the Blood

quickly the drug produces its effects (its *onset*), the time it takes for its effects to disappear (its *duration*), and whether the drug's effects are reduced (*tolerance*) or increased (*sensitivity*) if it is taken several times. Although this point may seem obvious, it is often overlooked. It is impossible to describe the behavioral effects of a drug on the basis of just one dose of the drug, since drugs can have very different effects, depending on how much of the drug is taken. Moreover, the probability that a drug will produce an effect also depends on the amount taken. As an example, consider Figure 1, which shows the risk of being involved in a traffic accident as a function of the amount of alcohol in a person's blood.

The way in which a drug is taken is also important. Cocaine can be taken by injection into the veins, by smoking, or by sniffing through the nose. Each of these routes of administration can produce different effects. Environmental factors also influence a drug's effect. Cocaine might change the amount of cereal and milk consumed in the morning but it might not change the amount consumed at a different time of day or if other types of food are available. Finally, individual factors also influence the drug effect. These include such factors as how many times an individual has taken a particular drug; what happened the last time it was taken; or what one may have heard from friends about a drug's effects.

HOW IS BEHAVIOR STUDIED IN THE LABORATORY?

Human behavior is very complex, and it is often difficult to examine. Although scientists do conduct studies on people, many investigations of drug effects on behavior are carried out using animals. With animals, investigators have better control over the conditions in which the behavior occurs as well as better information about the organism's past experience with a particular drug. Although animal experiments provide a precise, controlled environment in which to investigate drug effects, they also have their limitations. Clearly, they cannot research all the factors that influence human behavior. Nevertheless, many of the effects that drugs produce on behavior in animals also occur in humans. Moreover, behavioral studies sometimes require a large number of subjects with the same genetic makeup or with no previous drug experi-

ence. It is easier to meet these requirements in animal studies than in studies with people.

Since animals are often used in research studies, it is important to remember that behavioral scientists are very concerned about the general welfare of their animals. The U.S. Animal Welfare Act set standards for handling, housing, transporting, feeding, and veterinary care of a wide variety of animals. In addition, all animal research in the United States is now reviewed by a committee that includes a veterinarian experienced in laboratory-animal care. This committee inspects animal-research areas and reviews the design of experiments to ensure that the animals are treated well.

WHAT APPROACHES ARE USED TO EXAMINE DRUG EFFECTS?

In general, there are two ways to examine drug effects on behavior in the laboratory. One approach relies on observation of behavior in an animal's home cage or in an open area in which the animal (or person) can move about freely. When observational approaches are used, special precautions are necessary. First of all, the observer's presence should not disrupt the experiment. Television-monitoring systems and videotaping make it possible for the observer to be completely removed from the experimental situation. Second, the observer should not be biased. The best way to insure that the observer is not biased is to make the observer "blind" to the experimental conditions; that is, the observer does not know what drug is given or which subject received the drug. If the study is done in human subjects, then they also should be blind to the experimental conditions. An additional way to make sure observations are reliable is to use more than one observer and compare observations. If these precautions are taken, observational approaches can produce interesting and reliable data. Indeed, much of what is known about drug effects on motor behavior, food or water intake, and some social behaviors comes from careful observational studies.

Another approach uses the procedures of classical and operant conditioning. This involves training animals to make specific responses under special conditions. For example, in a typical experiment of this sort, a rat is placed in an experimental chamber and trained to press a lever to receive food. The number and pattern of lever presses are measured with an automatic device, and changes in responding are examined following drug administration. These procedures have several advantages. First, they produce a very consistent measure of behavior. Second, they can be used with human subjects as well as with several different animal species. Third, the technology for recording behavior eliminates the need for a trained observer.

WHAT BEHAVIORS DO DRUGS ALTER?

Some of the behaviors that drugs alter are motor behavior, sensory behavior, food and water intake, social behavior, and behavior established with classical and operant conditioning procedures. By combining investigations of these behaviors, scientists classify drugs according to their prominent behavioral effects. For example, drugs such as AMPHETAMINE and cocaine are classified as PSYCHOMOTOR STIMULANTS because they increase alertness and general activity in a variety of different behavioral procedures. Drugs such as MORPHINE are classified as analgesics because they alter the perception of pain, without altering other sensations such as vision or audition (hearing).

Motor Behavior. Most behaviorally active drugs alter motor behavior in some way. Morphine usually decreases motor activity, whereas with cocaine certain behaviors occur over and over again (that is, repetitively). Other drugs, such as alcohol, may alter the motor skills used in DRIVING a car or operating various types of machinery. Finally, some drugs alter exploratory behavior, as measured by a decrease in motor activity in an unfamiliar environment. Examination of the many ways in which drugs alter motor behavior requires different types of procedures. Some of these procedures examine fine motor control or repetitive behavior; others simply measure spontaneous motor activity.

Although changes in motor behavior can be observed directly, most studies of motor behavior use some sort of automatic device that does not depend on human observers. One of these devices is the running wheel. The type of running wheel used in scientific investigations is similar to the running wheel in pet cages. This includes a cylinder of some sort that moves around an axle when an animal walks or runs in it. The only difference between a running wheel in a pet cage and a running wheel in

TABLE 1
Blood Alcohol Level and Behavioral Effect

Percent Blood Alcohol	*Behavioral Effect*
0.05	alertness reduced
0.10	reaction time prolonged
0.20	motor function impaired
0.30	severe motor impairment
0.40	consciousness lost

the laboratory is its size and the addition of a counter that records the number of times the wheel turns. Another device for measuring motor behavior uses an apparatus that is surrounded by photocells. If the animal moves past one of the photocells, a beam of light is broken and a count is produced. Yet another way to measure motor behavior is with video tracking systems. An animal is placed in an open area and a tracking system determines when movement stops and starts as well as its speed and location. This system provides a way to look at unique movement patterns such as repetitive behaviors. For example, small amounts of amphetamine increase forward locomotion, whereas larger amounts produce repetitive behaviors such as head bobbing, licking, and rearing. Until recently, this type of repetitive behavior was measured by direct observation and description.

Although technology for measuring motor behavior is very advanced, it is important to remember that how much drug is given, where it is given, and the type of subject to whom it is given will also influence a drug's effect on motor behavior. Whether a drug's effects are measured at night or during the day is an important factor. The age, sex, species, and strain of the animal is also important. Whether food and water are available is another consideration as well as the animal's previous experience with the drug or test situation. As an example, see Table 1, which shows how the effects of alcohol on motor behavior differ depending on the amount of alcohol in a person's blood.

Sensory Behavior. The integration and execution of every behavior an organism engages in involves one or more of the primary senses, including hearing, vision, taste, smell, and touch. Obviously, a drug can affect sensory behavior and thereby alter a number of different behaviors. For example, drugs such as LYSERGIC ACID DIETHYLAMIDE (LSD) produce visual abnormalities and HALLUCINATIONS. PHENCYCLIDINE (PCP) produces a numbness in the hands and feet. Morphine alters sensitivity to painful stimuli.

It is difficult to investigate drug effects on sensory behavior, since changes in sensory behavior cannot be observed directly. In order to determine whether someone hears a sound, one must report having heard it. In animal studies, rats or monkeys are trained to press a lever when they hear or see a given stimulus. Then a drug is given and alterations in responding are observed. If the drug alters responding, it is possible that the drug did so by altering sensory behavior; however, care must be taken in coming to this conclusion since a drug might simply alter the motor response used to measure sensory behavior without changing sensory behavior at all.

One area of sensory behavior that has received considerable attention is pain perception. In most procedures for measuring pain perception, a potentially painful stimulus is presented to an organism and the time it takes the organism to respond to that stimulus is observed. Once baseline levels of responding are determined and considered reliable, a drug is given. If the time it takes the organism to respond to the stimulus is longer following drug administration and if this change is not because the animal is too sedated to make a response, then the drug probably has altered pain perception.

Among the most common procedures used to measure pain perception is the tail-flick procedure in which the time it takes an animal to remove its tail from a heat source is measured prior to and after administration of a drug such as morphine. Another commonly used procedure measures the time it takes an animal to lick its paws when placed on a warm plate or to remove its tail from a container of warm water. Thus, an alteration in pain perception is operationally defined as a change in responding in the presence of a painful stimulus. It is also important to note that the animal, not the experimenter, determines when to respond or remove its tail. Also, these procedures do not produce long-term damage or discomfort that extends beyond the brief experimental session.

Food and Water Intake. The simplest way to measure food and water intake is to determine how much an organism eats or drinks within a given period of time. A more thorough analysis might also include counting the number of times an organism eats or drinks in a single day, or measuring

the time between periods of eating and periods of drinking. Several factors are important in accurately measuring food and water intake. For example, how much food or water is available to the organism and when is it available? Is it a food the organism likes? When did the last meal occur?

In animals, food intake is often measured by placing several pieces of pelleted food of a known weight in their cages. The food that remains after a period of time is weighed and subtracted from the original amount to get an estimate of how much was actually eaten. Water intake is usually measured with calibrated drinking tubes clipped to the front of the animal's cage or with a device called a drinkometer, which counts the number of times an animal licks a drinking tube. An accurate measure of fluid intake also requires a careful description of the surrounding conditions. For example, was fluid intake measured during the day or during the night? Was food also available? What kind of fluid was available? Was there more than one kind of fluid available? These procedures are also used to examine drug intake. If rats are presented with two different drinking tubes, one with alcohol, another with water, they will generally drink more alcohol than water; however, the amount they drink is generally not sufficient to produce intoxication or physical dependence. Rats will drink a large amount of alcohol as well as other drugs of abuse such as morphine and cocaine when these drugs are the only liquid available. Indeed, most animals will consume sufficient quantities to become physically dependent on alcohol or morphine.

Social Behavior. Behaviors such as aggression, social interaction, and sexual behavior are usually measured by direct experimenter observation. Aggressive behavior can be measured by observing the number of times an animal engages in attack behavior when another animal is placed into its cage. In some cases, isolation is used to produce aggressive behavior. Sexual behavior is also measured by direct observation. In the male rat or cat, the frequency of behaviors such as mounting, intromission, and ejaculation are observed. Another interesting procedure for measuring social behavior is the social interaction test. In this procedure, two rats are placed together and the time they spend in active social interaction (sniffing, following, grooming each other) is measured under different conditions. In one condition, the rats are placed in a familiar environment; in another condition, the environment is unfamiliar. Rats interact more when they are in a familiar environment than when they are in an unfamiliar environment. Moreover, antianxiety drugs increase social interaction in the unfamiliar area. These observational techniques can produce interesting data, provided that they are carried out under well-controlled conditions, the behavior is well-defined, and care is taken to make sure the observer neither disrupts the ongoing behavior nor is biased.

Classical Conditioning. Classical conditioning was made famous by the work of the Russian scientist Ivan Pavlov in the 1920s. In those experiments, Pavlov used the following procedure. First, dogs were prepared with a tube to measure saliva, as shown in Figure 2. Then Pavlov measured the amount of saliva that was produced when food was given. The amount of saliva not only increased when food was presented but also when the caretaker arrived with the food. From these careful observations, Pavlov concluded that salivation in response to the food represented an inborn, innate response that did not require any learning. Because no learning was required, he called this an unlearned (unconditioned) response and the food itself an unlearned (unconditioned) stimulus. The dogs did not automatically salivate, however, when the caretaker entered the room; but after the caretaker and the food occurred together several times, the presence of the caretaker was paired with (or conditioned to) the food. Pavlov called the caretaker the *conditioned stimulus* and he called the salivation that occurred in the presence of the caretaker a *conditioned response*.

Events in the environment that are paired with or conditioned to drug delivery can also produce effects similar to the drug itself, much in the same way that Pavlov's caretaker was conditioned to food delivery. For example, when heroin-dependent individuals stop taking heroin, they experience a number of unpleasant effects, such as restlessness, irritability, tremors, nausea, and vomiting. These are called withdrawal or abstinence symptoms. If an individual experiences withdrawal several times in the same environment, then events or stimuli in that location became paired with (or conditioned to) the withdrawal syndrome. With time, the environmental events themselves can produce withdrawal-like responses, just as the caretaker produced salivation in Pavlov's dogs.

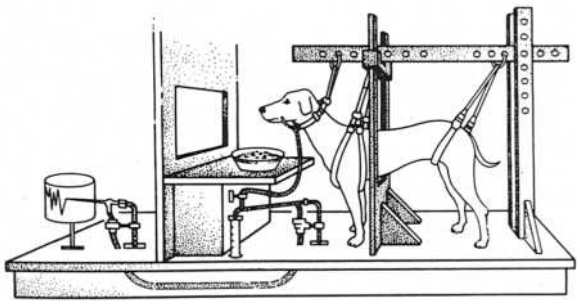

Figure 2
Diagram of Pavlov's Classical Conditioning Experiment. A tube is attached to the dog's salivary duct, and saliva drops into a device that records the number of drops

Operant Conditioning. About a decade after Pavlov's discovery of classical conditioning, another psychologist, B. F. Skinner, was developing his own theory of learning. Skinner observed that certain behaviors occur again and again. He also observed that behaviors with a high probability of occurrence were behaviors that produced effects on the environment. According to Skinner, behavior "operates" on the environment to produce an effect. Skinner called this process *operant conditioning*. For example, people work at their jobs because working produces a paycheck. In this situation, working is the response and a paycheck is the effect. In other situations, a person does something to avoid a certain effect. For example, by driving a car within the appropriate speed limit, traffic tickets are avoided and the probability of having a traffic accident is reduced. In this case, the response is driving at a given speed and the effect is avoiding a ticket or an accident.

If the effect that follows a given behavior increases the likelihood that the behavior will occur again, then that event is called a *reinforcer*. Food, water, and heat are common reinforcers. Drug administration is also a reinforcer. It is well known that animals will respond on a lever to receive intravenous injections of morphine, cocaine, and amphetamine, as well as a number of other drugs. Not all drugs are self-administered, however. For example, animals will respond to avoid the presentation of certain nonabused drugs such as the ANTIPSYCHOTICS (medications used in the treatment of schizophrenia). Because there is a good correlation between drugs that are self-administered by animals and those that are abused by people, the self-administration procedure is often used to examine drug-taking behavior.

In most operant conditioning experiments, animals perform a simple response such as a lever press or a key peck to receive food. Usually the organism has to make a fixed number of responses or to space responses according to some temporal pattern. The various ways of delivering a reinforcer are called *schedules of reinforcement*. Schedules of reinforcement produce very consistent and reliable patterns of responding. Moreover, they maintain behavior for long periods of time, are easily adapted for a number of different animals, and provide a very accurate measure of behavior. Thus, they provide a well-defined, *operational measure* of behavior, which is used to examine the behavioral effects of drugs.

Motivation, Learning, Memory, and Emotion. One of the biggest challenges for behavioral scientists is to develop procedures for measuring drug effects on processes such as motivation, emotion, learning, or memory since these behaviors are very difficult to observe directly. Drugs certainly alter processes such as these. For example, many drugs relieve anxiety. Other drugs produce feelings of pleasure and well-being; still others interfere with memory processes. Given the complexity of devised procedures, they are not described in detail here; however, it is important to emphasize that the approach for examining the effects of drugs on these complex behaviors is the same as it is for any behavior: First, carefully define the behavior and describe the conditions under which it occurs. Second, give a drug and observe changes in the behavior. Third, take special care to consider pharmacological factors, such as how much drug is given, when the drug is given, or the number of times the drug is given. Fourth, consider behavioral factors, such as the nature of the behavior examined, the conditions under which the behavior is examined, as well as the individual's past experience with the behavior.

SUMMARY

To find out how drugs alter behavior, several factors are considered. These include the PHARMACOLOGY of the drug itself as well as an understanding of the behavior being examined. Indeed, the behavioral state of an organism, as well as

the organism's past behavior and experience with a drug contribute as much to the final drug effect as do factors such as the dose of the drug and how long it lasts. Thus, the examination of drug effects on behavior requires a careful description of behavior with special attention to the way in which the behavior is measured. Behavioral studies also require a number of experimental controls, which assure that changes in behavior following drug administration are actually due to the drug itself and not the result of behavioral variability.

(SEE ALSO: *Addiction: Concepts and Definitions; Aggression and Drugs; Causes of Drug Abuse; Pharmacodynamics; Psychomotor Effects of Alcohol and Drugs; Reinforcement; Research, Animal Model; Sensation and Perception and Effects of Drugs; Tolerance and Physical Dependence*)

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LINDA A. DYKSTRA

Measuring Effects of Drugs on Mood

Subjective effects are feelings, perceptions, and moods that are the personal experiences of an individual. They are not accessible to other observers for public validation and, thus, can only be obtained through reports from the individual. Subjective-effect measures are used to determine whether the drug is perceived and to determine the quantitative and qualitative characterization of what is experienced. Although subjective effects can be collected in the form of narrative descriptions, standardized questionnaires have greater experimental utility. For example, they may be used to collect the reports of individuals in a fashion that is meaningful to outside observers, can be combined across subjects, and can provide data that are reliable and replicable. The measurement of subjective effects through the use of questionnaires is scientifically useful for determining the pharmacologic properties of drugs—including time course, potency, abuse liability, side effects, and therapeutic utility. Many of the current methods used to measure subjective effects resulted from research aimed at reducing drug abuse.

HISTORY

Drug abuse and drug addiction are problems that are not new to contemporary society; they have a long-recorded history, dating back to ancient times. For centuries, various drugs including ALCOHOL, TOBACCO, MARIJUANA, HALLUCINOGENS, OPIUM, and COCAINE, have been available and used widely across many cultures. Throughout these times, humans have been interested in describing and communicating the subjective experiences that arise from drug administration. Although scientists have been interested in the study of PHARMACOLOGY for many centuries, reliable procedures were not developed to measure the subjective effects of drugs until recently.

Throughout the twentieth century, the U.S. GOVERNMENT has become increasingly concerned with the growing problem of drug abuse. To decrease the availability of drugs with significant ABUSE LIABILITY, the government has passed increasingly restrictive laws concerning the possession and sale of existing drugs and the development and marketing of new drugs. The pressing need to regulate drugs that have potential for misuse prompted the government to sponsor research for the development of scientific methodologies that would be useful in assessing the abuse liability of drugs.

Two laboratories that made major contributions to the development of subjective-effect measures were Henry Beecher and his colleagues at Harvard

University and the government-operated Addiction Research Center (ARC) in Lexington, Kentucky. Beecher and his colleagues at Harvard conducted a lengthy series of well-designed studies that compared the subjective effects of various drugs—opiates, sedatives, and stimulants—in a variety of subject populations that included patients, substance abusers, and normal volunteers and highlighted the importance of studying the appropriate patient population. Additionally, this group laid the foundation for conducting studies with solid experimental designs, which include double-blind and placebo controls, randomized dosing, and characterization of dose-response relationships. Investigators at the ARC conducted fundamental studies of both the acute (immediate) and chronic (long-term) effects of drugs, as well as physical dependence and withdrawal symptoms (e.g., Himmelsbach's opiate withdrawal scale). A number of questionnaires and procedures now in use to study the subjective effects of drugs were developed, including the Addiction Research Center Inventory and the Single Dose Questionnaire. Although many of the tools and methods developed at the ARC are still in use, other laboratories have since modified and expanded subjective-effect measures and their applications.

MEASURES

Question Format. Subjective-effects measures are usually presented in the form of groups of questions (questionnaires). These questions can be presented in a number of formats, the most frequently used of which are ordinal scales and visual analog scales. The ordinal scale is a scale of ranked values in which the ranks are assigned based upon the amount of the measured effect that is experienced by each individual. Subjects are usually asked to rate their response to a question on a 4- or 5-point scale (e.g., to rate the strength of the drug effect from 0 to 4, with 0 = not at all; 1 = a little; 2 = moderately; 3 = quite a bit; and 4 = extremely). A visual-analog scale is a continuous scale presented as a line without tick marks or sometimes with tick marks to give some indication of gradations. A subject indicates the response by placing a mark on that line, according to a particular reference point; for example, lines are usually anchored at the ends with labels such as "not at all" and "extremely." Visual-analog scales can be unipolar (example: "tired," rated from no effect to extremely), or they may be bipolar (example: "tired/alert," with "extremely tired" at one end, "extremely alert" at the other, and "no effect" in the center). Another frequently used format is the binomial scale, usually in the form of yes/no or true/false responses, such as the Addiction Research Center Inventory. A fourth format utilizes a nominal scale, in which the response choices are categorical in nature and mutually exclusive of each other (e.g., drug class questionnaire).

Questionnaires. Frequently used subjective-effect measures include investigator-generated scales, such as adjective-rating scales, and standardized questionnaires, such as the Profile of Mood States and the Addiction Research Center Inventory. A description of a number of questionnaires follows; however, this list is illustrative only and is not meant to be exhaustive.

Adjective Rating Scales. These are questionnaires on which subjects rate a list of symptoms, describing how they feel or effects associated with drug ingestion. The questionaires can be presented to subjects with either visual-analog or ordinal scales. Items can be used singly or grouped into scales. Some adjective-type scales are designed to measure global effects, such as the strength of drug effects or the subject's liking of a drug, while other adjective rating scales are designed to measure specific drug-induced symptoms. In the latter use, the adjectives used may depend on the class of drugs being studied and their expected effects. For example, studies of amphetamine include items such as "stimulated" and "anxious," while studies of opioids include symptoms such as "itching" and "talkative." To study physical dependence, symptoms associated with drug withdrawal are used; for example, in studies of opioid withdrawal, subjects might rate "watery eyes," "chills," and "gooseflesh." Most adjective-rating scales have not been formally validated; investigators rely on external validity.

Profile of Mood States (POMS). This questionnaire was developed to measure mood effects in psychiatric populations and for use in testing treatments for psychiatric conditions such as depression and anxiety. It is a form of an adjective-rating scale. This scale was developed by Douglas McNair, Ph.D., and has been modified several times. It exists in two forms—one consisting of sixty-five and another of seventy-two adjectives describing mood states that are rated on a five-point scale from "not at all" (0)

to "extremely" (4). The item scores are weighted and grouped by factor analysis into a number of subscales, including tension-anxiety, depression-dejection, anger-hostility, vigor, fatigue, confusion-bewilderment, friendliness, and elation. This questionnaire has been used to measure acute drug effects, usually by comparing measures collected before and after drug administration. Its use in drug studies has not been formally validated; however, it has been validated by replication studies in normal and psychiatric populations and in treatment studies.

Single Dose Questionnaire. This was developed in the 1960s at the ARC to quantify the subjective effects of opioids. It has been used extensively and has been modified over time. This questionnaire consists of four parts; (1) a question in which subjects are asked whether they feel a drug effect (a binomial yes/no scale); (2) a question in which subjects are asked to indicate which among a list of drugs or drug classes is most similar to the test drug (a nominal scale); (3) a list of symptoms (checked yes or no); and (4) a question asking subjects to rate how much they like the drug (presented as an ordinal scale). The list of drugs used in the questionnaire includes placebo, opiate, stimulant, marijuana, sedative, and other. Examples of symptoms listed are turning of stomach, skin itchy, relaxed, sleepy, and drunken. While this questionnaire has not been formally validated, it has been used widely to study opioids, and the results have been remarkably consistent over three decades.

Addiction Research Center Inventory (ARCI). This is a true/false questionnaire containing more than 550 items. The ARCI was developed by researchers at the ARC to measure a broad range of physical, emotive, and subjective drug effects from diverse pharmacological classes. Subscales within the ARCI were developed to be sensitive to the acute effects of specific drugs or pharmacological classes (e.g., morphine, amphetamine, barbiturates, marijuana); feeling states (e.g., tired, excitement, drunk); the effects of chronic drug administration (Chronic Opiate Scale); and drug withdrawal (e.g., the Weak Opiate Withdrawal and Alcohol Withdrawal Scale). The ARCI subscales most frequently used in acute drug-effect studies are the Morphine-Benzedrine Group (MBG) to measure euphoria; the Pentobarbital-Chlorpromazine-Alcohol Group (PCAG) to measure apathetic sedation; and the Lysergic Acid Diethylamide Group (LSDG) to measure dysphoria or somatic discomfort. The use of the MBG, PCAG, and LSDG scales has remained standard in most studies of abuse liability. Subscales on this questionnaire were developed empirically, followed by extensive validation studies.

Observer-rated Measures. These may frequently accompany the collection of subjective effects and are often based on the subjective-effect questionnaires. Ratings are made by an observer who is present with the subject during the study, and items are limited to those drug effects that are observable. Observer-rated measures may include drug-induced behaviors (e.g., talking, scratching, activity levels, and impairment of motor function), as well as other drug signs such as redness of the eyes, flushing, and sweating. Observer-rated measures can be designed using any of the formats used in subject-rated measures. Examples of observer-rated questionnaires that have been used extensively are the Single Dose Questionnaire, which exists in an observer-rated version, and the Opiate Withdrawal Scale developed by Himmelsbach and his colleagues at the ARC.

USES OF SUBJECTIVE-EFFECT MEASURES

The methodology for assessing the subjective effects of drugs was developed, in large part, to characterize the abuse liability, the pharmacological properties, and the therapeutic utility of drugs. *Abuse liability* is the term for the likelihood that a drug will be used illicitly for nonmedical purposes. The assessment of the abuse-liability profile of a new drug has historically been studied by comparing it with a known drug, whose effects have been previously characterized. Drugs that produce euphoria are considered more likely to be abused than drugs that do not produce euphoria.

Subjective-effects measures may also be used to characterize the time course of a drug's action (such as time to drug onset, time to maximal or peak effect, and the duration of the drug effect). These procedures can provide information about the pharmacological properties of a particular drug, such as its drug class, whether it has AGONIST or ANTAGONIST effects, and its similarity to prototypic drugs within a given drug class. Subjective-response reports are also useful in assessing the efficacy (the ability of a drug to produce its desired

TABLE 1
Typical Response Profiles for Sedatives, Stimulants, and Opiates on Selected Subjective-Effect Measures

	Global Effects	*ARCI*	*POMS*	*Adjectives*
Sedatives	Drug Effect Liking High	PCAG	Fatigue (increase) Vigor (decrease)	Tired Sleepy Relaxed Drunk
Stimulants	Drug Effect Liking High	MBG	Vigor (increase) Fatigue (decrease)	Stimulated Nervous Thirsty Jittery
Opiates	Drug Effect Liking High	MBG PCAG		Nauseous Itchy Nodding Energetic

effects), potency (amount or dose of a drug needed to produce that effect), and therapeutic utility of a new drug. Subjective reports provide information regarding the potency and efficacy of a new drug in comparison to available treatment agents. Subjective-effect measures may be useful in determining whether a drug produces side effects that are dangerous or intolerable to the patient. Drugs that produce unpleasant or dysphoric mood-altering effects may have limited therapeutic usefulness.

DESCRIPTION OF MAJOR FINDINGS OBTAINED WITH DIFFERENT DRUG CLASSES

Drugs of different pharmacological classes generally produce profiles of subjective effects that are unique to that class of drugs and that are recognizable to individuals. The subjective effects of major pharmacological classes have been characterized using the questionnaires described above. Table 1 lists some major pharmacological classes and their typical effects on various instruments. While global measures provide quantitative information regarding drug effects, they tend not to differentiate among different types of drugs. Nevertheless, the more specific subjective-effect measures, such as the ARCI and the Adjective Rating Scales, yield qualitative information that can differentiate among drug classes.

CONCLUSION

Measures of the subjective effects of drugs have been extremely useful in the study of pharmacology. Questionnaires have been developed that are sensitive to both the global effects and the specific effects of drugs; however, research is still underway to develop even more sensitive subjective-effect measures and new applications for their use.

(SEE ALSO: *Abuse Liability of Drugs; Addiction: Concepts and Definitions; Causes of Substance Abuse; Drug Types*)

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Motivation Motivation is a theoretical construct that refers to the neurobiological processes responsible for the initiation and selection of such goal-directed patterns of behavior as are appropriate to the physiological needs or psychological desires of the individual. *Effort* or *vigor* are terms used to describe the intensity of a specific pattern of motivated behavior. Physiological "drive" states, caused by imbalances in the body's homeostatic regulatory systems, are postulated to be major determinants of different motivational states. Deprivation produced by withholding food or water is used routinely in studies with experimental animals to establish prerequisite conditions in which nutrients or fluids can serve as positive reinforcers in both operant and classical conditioning procedures. In more natural conditions, the processes by which animals seek, find, and ingest food or fluids are divided into appetitive and consummatory phases. Appetitive behavior refers to the various patterns of behavior that are used to locate and bring the individual into direct contact with a biologically relevant stimulus such as water. Consummatory behavior describes the termination of approach behavior leading subsequently to ingestion of food, drinking of fluid, or copulation with a mate.

Incentive motivation is the term applied to the most influential psychological theory that explains how the stimulus properties of biologically relevant stimuli, and the environmental stimuli associated with them, control specific patterns of appetitive behavior (Bolles, 1972). According to this theory, the initiation and selection of specific behaviors are triggered by external (incentive) stimuli that also guide the individual toward a primary natural incentive, such as food, fluid, or a mate. Drugs of abuse and electrical brain-stimulation reward can serve as artificial incentives. In a further refinement of this theory, Berridge and Valenstein (1991) defined incentive motivation as the final stage in a three-part process. The first phase involves the activation of neural substrates for pleasure, which in the second phase are associated with the object giving rise to these positive sensations and the environmental stimuli identified with the object. The critical third stage involves processes by which salience is attributed to subsequent perceptions of the natural incentive stimulus and the associated environmental cues. It is postulated that this attribution of "incentive salience" depends upon activation of the mesotelencephalic dopamine systems. The sensation of pleasure and the classical associative learning processes that mediate stages one and two respectively are subserved by different neural substrates.

In the context of drive states as the physiological substrates of motivation, the level of motivation is manipulated by deprivation schedules in which the subject is denied access mainly to food or water for fixed periods of time (e.g., twenty-two hours of food deprivation). An animal's increased motivation can be inferred from measures such as its running speed in a runway to obtain food reward. Under these conditions, speed is correlated with level of deprivation. Another measure of the motivational state of an animal is the amount of work expended for a given unit of food, water, or drug. Work here is defined as the number of lever presses per reinforcer. If one systematically obtains an increase in the number of presses, one can identify a specific ratio of responses per reward beyond which the animal is unwilling to work. This final ratio is called the break point. In the context of drug reinforcement, the break point in responding for COCAINE can be increased or decreased in a dose-dependent manner by dopamine agonists and antagonists respectively.

Appetitive behavior also can be measured directly in animal behavior studies either by an animal's latency (the time it takes) in approaching a source of food or water during presentation of a conditioned stimulus predictive of food, or simply by measuring the animal's latency approaching a food dispenser when given access to it. The fact that these appetitive behaviors are disrupted by dopamine antagonists has been interpreted as evidence of the role of mesotelencephalic dopamine pathways in incentive motivation.

In extending these ideas to the neural bases of drug addiction, Robinson and Berridge (1993) emphasized the role of sensitization, or enhanced behavioral responses to fixed doses of addictive drugs, that occurs after repeated intermittent drug treat-

ment. Neurobiological evidence indicates that sensitization is directly related to neuroadaptations in the mesotelencephalic dopamine systems. As a result of these neural changes, a given dose of amphetamine, for example, causes enhanced levels of extracellular dopamine and an increase in the behavioral effects of the drug. Given the role proposed for the mesotelencephalic DOPAMINE systems in incentive salience, it is further conjectured that craving, or exaggerated desire for a specific object or its mental representation, is a direct result of drug-induced sensitization. In this manner, repeated self-administration of drugs of abuse, such as AMPHETAMINE, produce neural effects that set the stage for subsequent craving for repeated access to the drug.

(SEE ALSO: *Brain Structures and Drugs; Causes of Substance Abuse; Research, Animal Model*)

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RESEARCH, ANIMAL MODEL The articles in this section describe studies of the effects of drugs on animals in the laboratory. These studies are important because many of our current beliefs about the nature of drug dependence involve concepts of learning and reinforcement, and many recently developed treatments are founded on these beliefs. The section contains *An Overview of Drug Abuse* research using animal models and detailed articles on various research concepts beings explored in this way: *Conditioned Place Preference; Conditioned Withdrawal; Drug Discrimination Studies; Drug Self-Administration; Environmental Influences on Drug Effects; Learning, Conditioning, and Drug Effects—An Overview; Learning Modifies Drug Effects; Learning Modifier Drug Effects; Operant Learning Is Affected by Drugs*.

See also *Aggression and Drugs: Research Issues; Motivation and Incentives*; and the articles in the section entitled *Research*.

An Overview of Drug Abuse A great deal of biomedical research is based on the belief that only through careful scientific analysis will we achieve a sound understanding of the problem of drug abuse and how to control it. Animal models of a human condition are an integral part of that analysis. Animal models were developed to help us understand the factors that control drug abuse. Under laboratory conditions it is possible to control many factors, such as the environment, genetics, drug history, and behavioral history, that cannot easily be controlled outside the laboratory. When these factors can be controlled, their influence on drug abuse can be precisely determined. As always, the use of animals involves the assumption that the behavior of animals is a valid model of a human disorder. The drug abuse research that has been conducted to this point makes it clear that this is a valid assumption.

There are three major animal models of aspects of drug abuse to consider: PHYSICAL DEPENDENCE, drug self-administration, and drug discrimination. Each of these has provided basic information about the fundamental processes that control drug abuse. In addition, each has provided practical information about the abuse potential of new drugs. Information on both of these topics represents an important contribution of animal research to solving the problems of drug abuse.

PHYSICAL DEPENDENCE

Often when a drug is administered repeatedly, TOLERANCE develops to its effects. That is, the dose of drug that is taken must be increased to achieve the same effect. With prolonged exposure to high doses, physical (or physiological) dependence may develop. That is, the person is dependent on the drug for normal physiological functioning. The existence of physical dependence is revealed when drug administration is stopped. When the drug is no longer administered, various physical changes begin to appear. Depending on the specific drug, these could include autonomic signs (e.g., diarrhea

and vomiting), somatomotor signs (e.g., exaggerated reflexes, convulsions), and behavioral signs (e.g., decreases in food and water intake). These effects have been called withdrawal but in the literature are also known as abstinence syndrome.

Historically, it was believed that physical dependence was the cause of drug addiction. That is, it was felt that one had to become physically dependent on a drug before abuse would occur and that the drug dependence or addiction was motivated by the need to relieve the abstinence syndrome. One of the major contributions of modern drug abuse research has been to make it clear that this is not true. In fact, much drug abuse occurs in people who are not physically dependent. Nevertheless, since the need to avoid the abstinence syndrome can increase the likelihood that a person will continue to abuse a drug, it is important that we understand physical dependence. Also, it would be desirable for new drugs that are developed not to produce physical dependence.

The development of physical dependence is most common with the OPIOIDS (morphine and morphine-like drugs) and central nervous system (CNS) depressants (e.g., BARBITURATES and ALCOHOL). Since opioids are very valuable painkillers but produce physical dependence when used repeatedly, there has been great interest in the development of drugs that can kill pain but do not produce physical dependence. Standard approaches to testing new opioids in animals for their potential for inducing physical dependence have been developed. In the early stages of testing, a new drug that has been found to be an effective ANALGESIC is given to an animal that is physically dependent on morphine (mice, rats, dogs, and monkeys have been used). After giving the drug, a trained observer scores the occurrence, intensity, and duration of abstinence signs such as shivering, restlessness, irritability, abdominal cramps, vomiting, diarrhea, and decreased eating and drinking. The drug may not affect the abstinence syndrome; it may relieve it or it may make the syndrome worse. A drug that relieves morphine abstinence probably will produce morphine-like physical dependence and may not be considered for further development on this basis. On the other hand, a drug that has no effect on abstinence, or even makes it worse, probably will not produce morphine-like physical dependence and may be worth pursuing. Often such a drug will be evaluated for its ability to produce physical dependence when it is administered repeatedly. A drug that produces no physical dependence of its own is clearly a candidate for further development.

Literally hundreds of new opioid drugs have been evaluated in animals for their capacity to produce physical dependence, and, in the process, we have learned much about physical dependence. It is clear that the higher the drug dose and the more frequent the exposure, the more intensive the physical dependence that develops. But recent research with human subjects has strongly suggested that even a single dose of an opioid may produce some level of physical dependence. Research has also shown that drugs that suppress the signs of morphine abstinence in a dependent animal generally have morphine-like effects themselves. That is, they suppress respiration and cough, kill pain, and have the potential to be abused and produce physical dependence. These drugs are known as opioid agonists. Other drugs, known as opioid ANTAGONISTS, may cause abstinence signs and symptoms to appear. Opioid antagonists do not have morphine-like effects themselves but are capable of blocking or reversing the effects of morphine and morphine-like drugs. Still other drugs, called mixed AGONIST-ANTAGONISTS, can have either type of effect, depending on dose and whether the animal is physically dependent. This group of drugs has proven particularly interesting in terms of its contribution to our understanding of how opioids work. In addition, many of them are effective analgesics with apparently low potential to produce physical dependence.

Other classes of drugs besides opioids produce physical dependence in animals as well. Many of the basic findings about physical dependence on CNS depressants (e.g., dose and frequency) are similar to what has been found with opioids. However, the abstinence syndrome can be even more severe than that seen with opioids. HALLUCINATIONS and even life-threatening convulsions can develop when long-term abuse of a barbiturate or alcohol is stopped. Abstinence syndromes have also been found after long-term exposure to TETRAHYDROCANNABINOL (THC), the active ingredient in MARIJUANA, and PHENCYCLIDINE (PCP). On the other hand, the abstinence syndrome that follows long-term exposure to such CNS stimulants as AMPHETAMINE or COCAINE is, by comparison, mild.

DRUG SELF-ADMINISTRATION

The distinguishing characteristic of drug abuse is the behavior of drug self-administration. When that behavior becomes excessive and has adverse consequences for the individual or society, the individual is considered to be a drug abuser. Therefore, the development of animal models for studying drug self-administration was the essential first step toward identifying factors that control the behavior. Humans consume drugs by several different routes of administration, including oral (e.g., alcohol), intravenous (e.g., cocaine and heroin), and inhalation (e.g., nicotine and crack cocaine). Although some of the factors that control drug abuse may be independent of the route of administration, others may not. Therefore, it has been important to develop models in which animals self-administer drugs by each of these routes.

Early attempts to study drug self-administration in animals involved oral self-administration. Oral self-administration of drugs has proven difficult to establish in laboratory animals, probably because most drug solutions have a bitter taste. Also, when consumed orally, the onset of the drug effect is relatively slow, making it difficult for the animal to make the association between drinking and drug effect. For these reasons, when first given a choice between water and a drug solution, most animals choose the water. However, conditions can be arranged so that the animal drinks large amounts of the drug solution in relatively short periods, by making the drug solution available when food is available, either as a meal or delivered repeatedly as small pellets of food. After a period of drug consumption in association with food, food can be removed from the experiment and the animal will continue to consume the drug orally. When given a choice between the drug solution and water, the animal will prefer the drug solution. This approach has been particularly important for research with alcohol, since humans abuse this drug orally.

To study intravenous self-administration, an animal is surgically implanted with a chronic intravenous catheter through which a drug can be administered. The animal wears a backpack and tether that protect the catheter and attach to a wall of the cage. The cage usually has levers that the animal can press to receive a drug injection and lights that can be turned on to signal that a drug injection is available. At that time, a lever press turns on an electric pump and injects a drug solution through the catheter into the vein. In this way, the animal model mimics intravenous drug injection by humans using a syringe. Since taste is not a factor and onset of drug action is rapid, conditioning animals to inject drugs by the intravenous route has proven relatively straightforward.

Reliable methods for administering drugs to animals by inhalation are important for studying the abuse of drugs that are inhaled, such as TOBACCO, SOLVENTS, or CRACK. Methods for studying solvent inhalation have been available for several years. Usually an animal is given the opportunity to press a lever to deliver a brief bolus of solvent vapor to the area around its nose. Methods for studying crack cocaine smoking in monkeys have only recently been developed. Monkeys are first trained to suck on a drinking tube; then the apparatus is arranged so that sucking on the tube delivers crack smoke to the monkey. Although the technique is new, it shows promise for the study of smoking in laboratory animals.

Research using these animal models has shown that, with few exceptions, animals self-administer the same drugs that humans abuse and show similar patterns of intake. For example, when given unlimited access to stimulants like amphetamine, both humans and animals alternate periods of high drug intake with periods of no drug intake. In the case of heroin, both animals and humans gradually increase drug intake to levels that are then stable for months and even years. In addition, animals do not self-administer drugs that humans do not abuse (e.g., aspirin) and even avoid those that humans report to be unpleasant (e.g., ANTIPSYCHOTIC DRUGS). These basic findings validate this as an excellent animal model of drug abuse by humans. The exceptions are the hallucinogens and marijuana, which animals do not readily self-administer.

Research using the self-administration model has increased our understanding of drug abuse in several different areas. It has become clear that drug self-administration is controlled by events that are initiated inside (e.g., a drug-induced change in brain chemistry) or outside (e.g., stress) the organism. With regard to events initiated inside the organism, we have begun to learn about the NEUROTRANSMITTER systems in the brain that are activated when drugs are self-administered. These changes are probably responsible for producing the

drug effect that people find desirable and that maintains their self-administration (the reinforcing effect). A substantial amount of recent research has focused on the neurotransmitter changes that are involved in the reinforcing effect of cocaine. It has been known for some time that cocaine increases the concentration of certain neurotransmitters in synapses. Research indicates that it is this effect on certain synapses in the CNS that use the neurotransmitter DOPAMINE in the brain that almost certainly plays the primary role in cocaine's reinforcing effect. Similar research suggests that the neurotransmitter SEROTONIN may play a primary role in the effects of alcohol.

Even though neurotransmitter changes occur when an individual self-administers a drug, they are not always sufficient to maintain drug self-administration or to make it excessive. Events initiated outside the organism—that is, environmental events—can increase or decrease drug self-administration. In the case of alcohol, for example, consumption can be increased in animals simply by presenting other things of value (e.g., food pellets) every few minutes. Although it is not known exactly why this occurs, analogous conditions may increase the consumption of alcohol and other drugs by some humans. Drug self-administration can also be decreased by environmental conditions. For example, increasing the cost of a drug or the effort required to obtain it decreases consumption. Drug self-administration can also be decreased by imposing punishment or by making valuable alternatives to drug self-administration available.

Animal research has also made it clear that certain individuals may, because of their genetic makeup, be more susceptible to the effects of alcohol or other drugs. For example, genetically different strains of rats can differ in their sensitivity to the effects of codeine, morphine, or alcohol. Also, animals can be selectively bred to be more or less sensitive to the effects of a drug. These findings clearly demonstrate a genetic component to drug sensitivity. Research suggests that these animals differ in the amounts of these drugs that they will self-administer. How broadly this conclusion cuts across drugs of abuse is unknown but is an active area of research.

In short, drug abuse research with animals has made it clear that whether drug self-administration occurs depends on an interaction between a drug, an organism, and an environment. A susceptible individual in an environment in which a drug is available and in which conditions encourage drug self-administration is more likely to be a drug abuser than one in which environmental conditions discourage drug abuse.

DRUG DISCRIMINATION

When a person takes a drug of abuse, it has effects that the person feels and can describe. These effects are called *subjective effects* (versus *objective effects* that can be seen by an observer), and they play an important role in drug abuse. A person is more likely to abuse a drug that has effects that the person describes as pleasant than one that the person describes as unpleasant.

The subjective effects of drugs of abuse have been studied in humans for many years and in several different ways. Early research involved administering drugs, usually morphine-like drugs, to former heroin addicts who then answered questionnaires that were designed to detect and classify the subjective effects of the drug. The single-dose opiate questionnaire asks the subject whether he or she can feel the drug, to identify the drug, to describe the symptoms, and to rate how much he or she likes it. The Addiction Research Center Inventory consists of a series of true-false statements that describe internal states that might be produced by drugs. The Profile of Mood States is a list of adjectives that can be used to describe mood. Responses to these questionnaires depend on variables such as type of drug and drug dose. Recent research has examined the subjective effects of a wider variety of drugs (including stimulants and depressants) not only in experienced but also inexperienced subjects. The purpose of this research is to understand the factors that can influence a person's subjective response to drugs of abuse.

Since subjective effects require a verbal description of an internal state, they can be directly studied only in humans. Over the past twenty to thirty years, however, it has become clear that animals can be trained to respond in a way that suggests they can detect the internal state produced by a drug. The behavioral paradigm is called DRUG DISCRIMINATION, and the drug effect is called a *discriminative stimulus effect*. Although a number of drug-discrimination paradigms have been developed, the most common is a two-lever paradigm in which the animal is trained to press one lever after

it has received a drug injection and the second lever after an injection of the drug vehicle or, in some cases, another drug. Responding on the lever that is appropriate to the injection is reinforced, usually by presenting a food pellet, while responding on the incorrect lever is not. If this is done repeatedly over a period of several weeks, the animal learns to respond almost exclusively on the lever associated with the injection. Although it is impossible to know what an animal feels, it seems as if the animal is reporting whether it feels the drug by the lever it presses. The animal can then be asked to "tell" us whether a new drug "feels" like the training drug. It will respond on the drug lever if the new drug is similar to the training drug and on the vehicle lever if it is not. It can also be "asked" whether a drug blocks the effects of the training drug. If the test drug blocks the effect of the training drug, it will respond on the vehicle lever when given both drugs.

There is a strong correspondence between the classification of drugs by humans based on their subjective effects and those by animals based on their discriminative stimulus effects. Research using the drug-discrimination model has increased our understanding of control of behavior by drugs in several different ways. First, this research has made it clear that behavior that is learned under the influence of a drug is more likely to occur again when the drug or a similar drug is taken again. This is a fundamental mechanism by which drugs control behavior. As with drug self-administration, a substantial amount of recent research has focused on the neurotransmitter changes that are involved in the discriminative stimulus effects of cocaine and alcohol. Again, dopamine seems to play a prominent role in this effect of cocaine, while serotonin may mediate the effects of alcohol. Environmental events, by contrast, do not seem to alter the discriminative stimulus effects of drugs substantially. However, little research has been done in this area.

ABUSE LIABILITY TESTING AND TREATMENT RESEARCH

One important application of animal models of drug abuse is the prediction of the likelihood that a new drug will be abused if it is made available to people. Clearly, the prevalence of abuse of a drug can be reduced by restricting its availability, and drugs with high potential for abuse should be the least available. All the models discussed here are used for predicting some aspect of the abuse liability of new drugs. However, the task is not simply a matter of detecting abuse liability and making the drug unavailable. ABUSE LIABILITY must be considered in the context of any potential therapeutic use of the drug, and a cost-benefit analysis that weighs liability for abuse against therapeutic benefits should be made.

Opioids are an excellent example of these considerations. Morphine is often the only appropriate analgesic for intense PAIN. However, it produces physical dependence and has a high potential for abuse. A drug that produces analgesia equivalent to or greater than that of morphine but does not produce physical dependence would be a highly desirable compound. Techniques for establishing this have been described in related articles. A new drug can be tested for its ability to suppress abstinence syndrome in monkeys that are dependent on morphine and for its ability to produce physical dependence of its own type in naive animals. A similar approach is taken with the drug in drug self-administration experiments. We may ask whether the drug maintains self-administration in experienced monkeys or whether naive monkeys will initiate self-administration. In addition, we can evaluate whether the drug is likely to be preferred to morphine by allowing an animal to choose between morphine and the new drug or determining how hard the animal will work to receive an injection of the drug relative to how hard it will work for morphine. Finally, we can ask whether the drug has discriminative stimulus effects that are similar to those of morphine or of any other drug of abuse. A drug that supports physical dependence, is self-administered, and has morphine-like discriminative stimulus effects is likely to have high potential for abuse in humans and unlikely to be a viable substitute for morphine. On the other hand, a drug that lacks one or more (preferably all) of these effects may be worth pursuing.

Animal models of drug abuse have been used for the development of drugs that may be useful in the treatment of drug abuse. In some ways it seems unusual to suggest treating a drug abuse problem with another drug. However, in the case of opioids, METHADONE, a morphine-like agonist, has proven to be quite useful in the treatment of opioid dependence. Although the drug is still self-administered and physical dependence is maintained, treatment with methadone allows the person to lead a rela-

tively normal life that does not require the high-cost behaviors (e.g., crime, intravenous injection) associated with abuse of illicit opioids.

The animal models described here, particularly drug self-administration and drug discrimination, are now being applied to the development of drugs that may be useful in treatment. These approaches are based on the reasonable but as yet unvalidated assumption that blocking or mimicking the reinforcing and subjective effects of drugs will decrease drug abuse. In the case of cocaine, dopamine antagonists and, surprisingly, opioids have shown some promise in animal models as potential treatment compounds. It is not yet clear whether these compounds will be effective in humans. Nevertheless, this is an area of active research that shows promise for helping with treatment of drug abuse for development as treatment compounds.

(SEE ALSO: *Abuse Liability of Drugs; Reinforcement; Research*)

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WILLIAM WOOLVERTON

Conditioned Place Preference A procedure called conditioned place preference has been used to study the "rewarding" effects of drugs. The procedure is designed to ask the question "When given a choice, will an animal prefer an environment in which it has experienced a drug to one in which it has not?" To answer this question, an animal is placed in an experimental chamber that is divided into two compartments that are different in some way. For example, they may have different floors and/or distinctive odors. Initially, the animal is placed in the chamber for several preconditioning trials and the time spent in each compartment is measured. Usually, a rat exhibits some preference for one or the other side in these trials. At this point, the experimenter can do one of two things—(1) modify the compartments in some way, perhaps by changing the lighting, so that equal time is spent in the two chambers before proceeding (balanced procedure), or (2) go ahead with the experiment with unequal preferences (unbalanced procedure). With either procedure, conditioning trials are conducted next.

To run conditioning trials, a barrier is placed in the middle of the chamber that does not allow the animal to switch sides. The drug of interest is then administered to the animal and it is confined to one compartment for usually fifteen to thirty minutes. If the unbalanced procedure is used, the animal is usually placed in the compartment that was initially avoided. A second group may be given a placebo (a substance that has no effect) under these same conditions or a placebo may be given to these same animals before placing them in the second compartment in alternating sessions. In this way, the effect of the drug is associated with a particular environment. After several—three to ten—conditioning sessions, the animal is placed in the chamber without being given the drug, and the door is removed so that the animal can spend time in either compartment. The length of time spent in each chamber is recorded and used as a measure of preference for that chamber.

The hypothesis underlying this sort of experiment is that the length of time spent in an environment should increase if that environment is associated with the effects of a drug of abuse. In fact, many studies have shown that this does happen with drugs such as HEROIN, COCAINE, and AMPHETAMINES. In the balanced procedure, animals spend more time in the drug-associated side than in the other side. In the unbalanced procedure, the animals spend more time in the drug-associated side than they did previously, but only rarely demonstrate an actual preference for it. As would be expected, preference is greater with higher doses of the drug and does not occur with placebo injections. In addition, it does not occur with drugs that are not typically abused, such as antipsychotic drugs, antidepressant drugs, and opioid antagonists. Thus, it seems likely that the technique measures a drug effect that is related to drug abuse.

Like other models for studying drug abuse, conditioned place preference has strengths and weak-

nesses. Among its strengths is that animals are tested in a drug-free state. Therefore, the measure of preference is not influenced by the direct effects of drugs. The procedure can be done with drug injections given by routes other than intravenous, therefore surgical preparation is not involved. Moreover, the procedure is rapid, with maximum effect usually evident within three conditioning sessions.

The major weakness relates to the drug effects that it is measuring. Since drug administration is not due to the behavior of the animal (i.e., self-administration), it is by definition not a reinforcing effect. Although many of the same drugs that are self-administered induce place preferences, it is not clear whether the drug effect studied in conditioned place preference is the same as that studied in procedures that directly measure reinforcing effects. Another weakness is that is it not known whether it is meaningful to compare drugs in terms of their ability to engender place preferences. That is, if drug X induces a greater place preference than drug Y, does it have more abuse potential? Finally, because the procedure involves the simple behavioral response of moving from one chamber to another, it is not known whether it can be used to study some of the complex behavioral variables that are known to be determinants of drug self-administration. Despite these ambiguities, however, the simplicity of the procedure makes it likely that it will continue to be useful for studying drug abuse.

(SEE ALSO: *Abuse Liability of Drugs; Reinforcement*)

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Conditioned Withdrawal Upon cessation from drug taking, many individuals experience unpleasant effects (i.e., WITHDRAWAL), which can include both physiological and psychological symptoms. For example, for OPIATE drugs such as MORPHINE and HEROIN, withdrawal symptoms can include restlessness, anorexia, gooseflesh, irritability, nausea, and vomiting. Withdrawal symptoms are most pronounced following a long history of exposure to ALCOHOL and opiates, but a variety of withdrawal symptoms can occur after exposure to most psychoactive drugs.

As with most other drug effects, researchers have shown that these withdrawal symptoms can be conditioned or linked by learning to environmental cues. This research on *conditioned withdrawal* has included both human case reports and laboratory animal research. For example, Vaillant (1969) reported that individuals who had been abstinent from opiates for months would experience "acute craving and withdrawal symptoms" upon reexposure to situations previously associated with opiate use. Further, Goldberg and Schuster (1967) showed that withdrawal symptoms also can be conditioned in laboratory animals. In their experiment, rhesus monkeys were addicted to morphine by giving them the drug repeatedly. The monkeys were then given an occasional injection of nalorphine, an opiate antagonist, which immediately led to the monkeys exhibiting signs characteristic of withdrawal. The injection of nalorphine was always given in the presence of a specific environmental stimulus, in this case a tone. Following several exposures to the tone paired with nalorphine, Goldberg and Schuster found that presentation of the tone itself was sufficient to produce the withdrawal signs.

The behavioral mechanism most likely to account for the phenomenon of conditioned withdrawal is *classical conditioning* (also known as *Pavlovian*). In Pavlov's classic experiments on this type of conditioning, a neutral stimulus such as a bell, is repeatedly paired with a nonneutral stimulus such as food. Eventually the bell itself elicited salivation, which was initially observed only to the food. In conditioned withdrawal, a neutral stimulus (e.g., a bell, a needle, a room, a friend, a street corner, or certain smells) is paired with the nonneutral stimulus of withdrawal until eventually those neutral stimuli will also elicit withdrawal symptoms.

The phenomenon of conditioned withdrawal can have important implications for drug-abuse treatment. The experience of drug withdrawal is often an important factor in the long-term maintenance of drug abuse. That is, as individuals experience withdrawal, they are likely to seek out a new drug supply to relieve withdrawal symptoms. An important aspect of drug-abuse treatment is relieving the symptoms of withdrawal during the period immediately following the cessation of drug use. Conditioned effects, however, are often long-lasting and do not depend on the continued presentation of the initial nonneutral stimulus (in this case withdrawal). Even after a patient has been withdrawn from a drug, stimuli that have been conditioned to elicit withdrawal symptoms may still be effective. Therefore, upon reexposure to those stimuli a patient may be much more likely to relapse to drug abuse. Thus, to be successful, any treatment regimen for drug abuse must deal with conditioned withdrawal.

(SEE ALSO: *Causes of Substance Abuse; Wekler's Pharmacologic Theory of Drug Addiction*)

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CHARLES SCHINDLER
STEVEN GOLDBERG

Drug Discrimination Studies When a person takes a drug of abuse, it has effects that a person feels and can describe. These are termed *subjective effects* and they play an important role in drug abuse. People are more likely to abuse a drug that has effects they describe as pleasant than one they describe as unpleasant.

The subjective effects of drugs of abuse have been studied in humans for many years and in several different ways. Early research involved administering drugs, usually morphine-like drugs, to former HEROIN addicts—who then answered questionnaires that were designed to detect and classify the subjective effects of the drug. The single-dose OPIATE questionnaire asks subjects whether they can feel the drug, to identify the drug, to describe the symptoms, and to rate how much they like it. The Addiction Research Center Inventory consists of a series of true/false statements that describe internal states that might be produced by drugs. The Profile of Mood States is a list of adjectives that can be used to describe mood. Responses to these questionnaires depend on variables such as type of drug and drug dose. Recent research has examined the subjective effects of a wider variety of drugs (including STIMULANTS and DEPRESSANTS) in both experienced and inexperienced subjects. The purpose of this research is to understand the factors that can influence a person's subjective response to drugs of abuse.

Since subjective effects require a verbal description of an internal state, they can only be studied directly in humans. Since the 1960s, however, it has become clear that animals can be trained to respond in a way that suggests they can detect the internal state produced by a drug. The behavioral paradigm is called DRUG DISCRIMINATION, and the drug effect is called a *discriminative stimulus effect*. Although a number of drug-discrimination paradigms have been developed, the most common is a two-lever paradigm. Here the animal is trained to press one lever after it has received a drug injection and the second lever after an injection of the drug vehicle or, in some cases, another drug. Responding on the lever that is appropriate to the injection is reinforced, usually, by a food pellet; responding on the incorrect lever is not reinforced. If this is done repeatedly over a period of several weeks, the animal learns to respond almost exclusively on the lever associated with the injection.

Although it is difficult to know what an animal feels, it seems as if the animal is telling us whether it feels the drug or not by the lever it presses. The animal can then be asked to "tell" us whether a new drug "feels" like the training drug. It will respond on the drug lever if it does and on the vehicle lever if it does not. It can also be "asked" whether a drug blocks the effects of the training drug. If the test drug does block the effect of the training drug, the animal will respond on the vehicle lever when given both drugs.

CONCLUSIONS

What makes this area of research so exciting are the striking similarities between the classification of drugs by humans, based on their subjective effects, to those by animals, based on their discriminative stimulus effects. Therefore, this animal model can be used to investigate the influence of factors such as genetics, drug history, and behavioral history—factors that cannot be easily controlled in human subjects—on the subjective effects of drugs. It also allows us to predict whether a new drug is likely to have subjective effects, like a known drug of abuse, or is likely to block the subjective effects of the drug of abuse, without giving the drug to humans. If an animal is trained to discriminate a drug of abuse and presses the drug lever when given the new drug, then it is highly likely that the new drug will have subjective effects in humans similar to those of the drug of abuse. Its availability might then be restricted. If the animal responds on the vehicle lever when given the combination of the new drug and the drug of abuse, the new drug may block the subjective effects of the drug of abuse. Such a drug might then be useful for treating drug abuse.

(SEE ALSO: *Abuse Liability of Drugs; Drug Types; Sensation and Perception*)

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WILLIAM WOOLVERTON

Drug Self-Administration One factor that distinguishes a drug of abuse from a drug that is not abused is that taking the drug of abuse increases the likelihood that it will be taken again. In such a case, we say that this drug has reinforced the drug self-administration response and that it has reinforcing effects. Factors that influence reinforcing effects, therefore, profoundly influence drug self-administration and drug abuse. Knowing the reinforcing effects of drugs is essential to understanding drug abuse.

Techniques developed on laboratory animals allow us to study the reinforcing effects of drugs, using the intravenous and oral routes as well as smoking. To study intravenous self-administration, the researcher surgically implants a chronic intravenous line (a catheter) through which a drug can be administered. Laboratory animals (rats, mice, monkeys, and so on) live in cages in which they can operate some device, usually a lever press, that turns on an electric pump to send some drug solution through the catheter. Oral self-administration is harder to establish, since drugs are usually bitter; however, by arranging conditions so that large amounts of drug solution are ingested in relatively short periods—usually by adding the drug to water when food is available—researchers can condition animals to self-administer drugs orally. Research on the smoking of TOBACCO or CRACK-COCAINE is important and this too needs conditioning for reliable study.

Animals used in research studies have shown that, with few exceptions, they abuse the same drugs that humans abuse and show similar patterns of intake. (Exceptions include MARIJUANA and HALLUCINOGENS, such as LSD, which animals do not seem to find reinforcing.) Drug self-administration studies have been used to predict whether a new drug is likely to be abused by humans if it becomes easily available. More important, such research has allowed us to understand some factors that can increase or decrease the reinforcing effects of drugs that contribute to human drug abuse. Some of these factors relate to the drug itself; others to the environment. For example, drugs that increase the concentration of the NEUROTRANSMITTER DOPAMINE in the synapses of the brain (e.g., cocaine) are more likely to have abuse potential than those that do not.

Research has made it clear that even the most preferred drug—cocaine—will be self-administered differently depending on environmental conditions. If more lever presses are required to obtain it (it "costs" more), less is consumed. Drug self-administration can also be decreased by punishment or by making valuable alternatives available. In short, drug self-administration research has shown that whether a drug will be abused is determined by a complex interaction between the drug, the environment, and the organism. Current research is aimed at understanding the dynamics of that interaction in a quantitative way.

(SEE ALSO: *Abuse Liability of Drugs; Adjunctive Drug Testing*)

WILLIAM WOOLVERTON

Environmental Influences on Drug Effects More than any other discipline, the field of behavioral PHARMACOLOGY has attempted to understand the influence of nonpharmacological, or environmental, factors on the effects of abused drugs. Since the classic demonstration by Dews (1955, 1958) showing that the effects of pentobarbital and METHAMPHETAMINE depend on the manner in which behavior is controlled by the schedule of REINFORCEMENT, researchers have been interested in various environmental influences on the effects of drugs. Some of these effects are described elsewhere in this encyclopedia (and see Barrett, 1987, for a more detailed review). This article reviews additional influences to illustrate the overwhelming conclusion that the effects of a drug depend on complex environmental variables that may override the typical pharmacological effects of a compound. Indeed, the evidence for environmental influences on drug action is so compelling that when the effects of abused drugs are characterized, "susceptible to environmental modulation" should be a salient distinguishing description along with physiological features.

BEHAVIORAL CONSEQUENCES

The specific manner in which behavior is controlled by its consequences may often represent a strong influence on drug action. In research situations, this is apparent in the effects of AMPHETAMINE or COCAINE on punished and nonpunished responses maintained by the presentation of food. Low rates of nonpunished responses are typically increased by these drugs (PSYCHOMOTOR STIMULANTS), whereas comparable low rates on punished responses are not affected by these drugs or are only decreased further. In the Dews studies (1955, 1958), the effects of the drugs differed depending on whether behavior was maintained at relatively high response rates under a fixed-ratio schedule that provided food following every *n*th response or whether responses occurred at lower rates under a fixed-interval schedule that provided food for the first response after *t* minutes. Explanations of the differential effects of the drugs could not be based on different levels of motivation, since these schedule conditions alternated sequentially within the same experimental session. Although these and similar results were obtained under carefully controlled experimental conditions, such findings document the essential point that environmental conditions surrounding and/or supporting behavior play a very important role in determining the effects of drugs.

BEHAVIORAL CONTEXT

The environmental modulation of drug effects has been shown repeatedly, by using schedule-controlled responses and various types of events. These findings represent two areas of research demonstrating how drug effects are modified directly by existing environmental conditions:

(1) More remote influences can also influence drug action. In behavioral history, for example, consequences that have occurred in the distant past can significantly alter the effects of abused drugs even though no traces of that experience are apparent in current behavior.

(2) In other studies in which environmental influences helped determine the effects of an abused drug, behavioral consequences occurring under one experimental condition alter the action of drugs occurring under different conditions. In this case, the conditions that interact are relatively close in time. For example, in an experiment with monkeys, exposure to a procedure in which responses avoided the delivery of a mild electric shock completely reversed the effects of amphetamine on punished responses that had occurred in a different and adjacent context (i.e., under different stimulus conditions from the avoidance schedule and separated by only a few minutes).

Comparable results, although with different species, different schedule conditions, and different environmental events, have also been arrived at with ALCOHOL, cocaine, and CHLORDIAZEPOXIDE (Barrett, 1987). The findings show the generality of this phenomenon—that the environment is an important variable contributing to the effects of drugs on behavior. The actions of a drug at its receptor site and the transduction

mechanisms that ensue can be affected by events occurring in the environment.

SUMMARY

The studies described here indicate the powerful influences that exist in the environment that can alter the course of the effects of abused drugs. Such findings illustrate the need to examine those influences and the manner in which they occur, although it is often tempting to attribute all changes in behavior to the abused drug. Consequences that are immediate, as in the existing environment, or remote, such as in the individual's past experience, help determine the acute effects of drugs and may also contribute to long-term abuse and persistent drug use.

(SEE ALSO: *Adjunctive Drug Taking; Causes of Substance Abuse; Reward Pathways and Drugs; Tolerance and Physical Dependence*)

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JAMES E. BARRETT

Intracranial Self-Stimulation (ICSS)

The intracranial self-stimulation (ICSS) procedure is used to study the effects of drugs on reward processes, or regions involved in pleasurable feelings, in the brain. In humans undergoing brain surgery, researchers were able to induce limb movements or produce sensations by electrically stimulating various regions of the cortex. Similarly, electrical stimulation of certain brain regions in the rat was reinforcing, or pleasurable, thus creating a new area for brain research. An electrode capable of delivering varying intensities and durations of electrical impulses was implanted in the brain of a rat. These animals could be trained to press a lever that would activate the implanted electrode, sending a small impulse to a specific brain region. In addition, animals could also be trained to press a lever that would "shut off" brain impulses in other regions. These animals will give up food and water, and even sexual activities, in order to perform tasks that lead to brain stimulation in certain regions. Based on these results, this procedure was recognized as a method by which mechanisms underlying drug addiction could be studied.

Early work in brain stimulation involved mapping out which brain areas would support self-stimulation in animals, primarily rats. Animals were trained using operant procedures in which a press of the lever would deliver an electrical stimulus to the brain. Researchers found two systems of reward in the rat brain using ICSS: a dorsal (closer to the back of the animal) system projecting from the caudate/septal area through the dorsal thalamus to the tectum, and a ventral system (closer to the abdomen of the animal), the medial forebrain bundle. The "punishment" system seemed to be located in the diencephalon and the tegmentum. Rats will readily self-stimulate when electrodes are implanted into the ventral tegmental area (VTA) and substantia nigra, brain regions associated with reward. Researchers hypothesized that, by stimulating these brain regions, the rats were activating their own dopamine neurons electrically, thus producing the effects of reward. Dopamine is a neurotransmitter found in the brain of rodents and primates. This neurotransmitter is thought to be involved in the rewarding or pleasurable effects of drugs of abuse.

Drugs can interact with the established pattern of self-stimulation in an animal. Interactions between drugs and ICSS suggest that these treatments act through the same mechanisms. The rate at which the animal presses the lever is correlated with the intensity of the current being delivered to the brain. However, the rate at which the animal presses the lever is not necessarily related to the amount of pleasure the animal is experiencing. The influences of various drugs on self-stimulation behavior can be due to a variety of effects, such as increases or decreases in general activity, changes in motivation or memory, etc. To state that a drug has an effect on self-stimulation, these possibilities must be ruled out. To do this, one can compare

data describing the effects of the test drug in other behavioral paradigms (e.g., locomotor activity, self-administration) to the effects observed in ICSS.

Despite these limitations, researchers have collected interesting data, examining the effects of various drugs of abuse on rate of self-stimulation. Animals were trained to press a lever that would result in electrical stimulation of the brain. Then, the intensity of the stimulation was lowered so that the animals would not press the lever very often. When the animals were given the psychomotor stimulant amphetamine, the animals began to press the lever at a very high rate that gradually declined to the rate observed at low stimulation intensities. To rule out that animals might be pressing the lever more often due to the motor-activating effects of amphetamine, these researchers looked at the effects of amphetamine on lever-pressing in rats that were not receiving any brain stimulation. They saw no changes in lever-pressing before or after the rats were given amphetamine. Thus, they concluded that amphetamine enhances the reward produced by the subthreshold stimulation by activating reward pathways in the brain.

Another approach in using ICSS to measure the rewarding effects of drugs is to train animals to regulate the intensity of the stimulation that they receive in the brain. Animals are given access to two levers in the test chamber. When the animal pressed one of these levers for the first time, a relatively high level of brain stimulation was delivered. However, subsequent presses of the lever deliver decreasing levels of stimulation. The animal can "reset" the stimulation to the original high level by pressing the second lever. Under these conditions, the animals reliably reset their stimulation level once it drops below a certain point. From this measurement, researchers are able to determine each animal's reward threshold in a very reliable way. Regardless of the initial level of stimulation, these animals would press the reset lever at the same intensity of stimulation. Drugs such as amphetamine and morphine have "threshold-lowering" effects, such that the animals would press the reset lever at a lower intensity after receiving these drugs. This suggests that these drugs are themselves reinforcing, or pleasurable.

ICSS has been used to study the effects of the chronic administration of cocaine. Depending on the frequency of administration and amount of cocaine given, difference changes in ICSS responses have been observed. When low doses of cocaine were given once or several times a day, no changes in the ICSS threshold were observed. However, when higher doses of cocaine were administered for seven days, the reward threshold was increased in these animals, indicating that tolerance to the rewarding effect of cocaine had developed and/or that the effects of cocaine had become less pleasurable. In addition, animals that self-administered cocaine also exhibited this increase in the ICSS reward threshold. These experimental results are comparable with those observed in human drug users who take increasingly greater amounts of drug to achieve the same pleasurable effect over a long period of time.

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Heather L. Kimmel

Learning, Conditioning, and Drug Effects—An Overview The effects of abused drugs can be examined at many levels, ranging from the molecular to the cellular to the behavioral. Each of these research areas contributes significant information to understanding the mechanisms by which drugs of abuse and alcohol produce their diverse effects. The most tangible sign of both immediate and long-term actions of abused drugs is their effects on behavior. Often it is incorrectly assumed that behavior is a passive reflection of more significant events occurring at a different and (usually) more molecular level. Understanding those cellular events is occasionally viewed as the key to understanding drug abuse and to intervention strategies. In fact, however, behavior itself and the variables that control it play a prominent and often profound role in directly determining drug action and, most likely, those cellular and molecular events that participate in behavior and in the ef-

fects of drugs. The variables that guide and influence behavior also affect molecular substructures—therefore, behavioral and neurobiological processes are interdependent.

EXPERIMENTAL ANALYSIS OF BEHAVIOR AND DRUGS OF ABUSE

The progression of behavioral approaches in the study of the effects of abused drugs is characteristic of the cumulative and evolutionary nature of scientific progress. A number of techniques are now available that permit the development and maintenance of a variety of behaviors that are remarkably stable over time, sensitive to a number of interventions, and reproducible within and across species. These procedures have evolved over the past several years and reflect the combined efforts of individuals in different disciplines ranging from psychology, pharmacology, physiology, and ethology. For the most part, research studying the effects of abused drugs on behavior has been conducted by two basic procedures. One procedure uses *unconditioned behavior*, such as locomotor activity that is more spontaneous in its occurrence (but still influenced by environmental conditions) and requires no specific training before it can be studied. Many PSYCHOMOTOR STIMULANTS such as COCAINE and AMPHETAMINE, for example, produce large and consistent increases in locomotor activity in laboratory animals. Frequently, however, unconditioned behavior is produced or elicited by the presentation of specific stimuli, and it is then brought under experimental control by arranging for the production of a response to a stimulus other than that originally responsible for its occurrence. The Russian physiologist Ivan Pavlov, for example, performed extensive studies in 1927, in which he used the unconditioned salivary response to food and to conditioned stimuli paired with food to study processes of classical or *respondent* conditioning. Although this approach has been used somewhat less often than other techniques, respondent conditioning procedures still serves as a very useful method for studying drug action (Barrett & Vanover, 1993).

The second procedure, which is designated as *operant conditioning*, uses the methods and techniques developed by the pioneering American psychologist B. F. Skinner (1938) to investigate behavior controlled by its consequences. The body of experimental research using operant conditioning techniques to study the effects of abused drugs is extensive (see Iversen & Lattal, 1991, for general reviews of the techniques and applications).

Unconditioned and Conditioned Respondent Behavior. Respondent behavior is elicited by specific stimuli and usually involves no specific training or conditioning, since the responses studied are typically part of the behavioral repertoire of the species and are expressed under suitable environmental conditions. Although the factors responsible for the occurrence of these behaviors presumably lie in the organism's distant evolutionary past, certain unconditioned responses can be brought under more direct and immediate experimental control through the use of procedures first discovered and systematically explored by Pavlov. These procedures consist of expanding the range of stimuli capable of producing an elicited response. In respondent conditioning, previously noneffective stimuli acquire the ability to produce or elicit a response by virtue of their temporal association with an unconditional stimulus, such as food, which is capable of eliciting a response without prior conditioning. Thus, when a distinctive noise, such as a tone, is repeatedly presented at the same time that or shortly before food is given, the tone acquires the ability to elicit many of the same responses originally limited to food.

Respondent behaviors depend primarily on *antecedent* events that elicit specific responses. Typically, these responses do not undergo progressive differentiation, that is, the responses to either a conditioned or an unconditioned stimulus are generally quite similar. These procedures also do not establish new responses but simply expand the range of stimuli to which that response occurs.

Operant Behavior. In contrast to respondent behavior, operant behavior is controlled by *consequent* events, that is, it is established, maintained, and further modified by its consequences. Operant behavior occurs for reasons that are not always known. The responses may have some initially low probability of occurrence or they may never have occurred previously. Novel or new responses are typically established by the technique of "shaping," in which a behavior resembling or approximating some final desired form or characteristic is selected, increased in frequency and then further differentiated by the provision of a suitable consequence, such as food presentation to a food-de-

prived organism. This technique embodies the principle of reinforcement and has been widely used to develop operant responses such as lever pressing by rodents, humans, and nonhuman primates. Behavior that has evolved under such contingencies may bear little or no resemblance to its original form and can perhaps only be understood by careful examination of the organism's history. Although some behaviors often appear unique or novel, it is likely that the final product emerged as a continuous process directly and sequentially related to earlier conditions. The manner in which operant responses have been developed and maintained, as well as further modified, has been the subject of extensive study in the behavioral pharmacology of abused drugs and has had a tremendous impact on this field. Many of the potent variables that influence behavior, such as reinforcement, punishment, and precise schedules under which these events occur, also are of critical import in determining how a drug will affect behavior.

Respondent Versus Operant Behavior. Although it is possible to tell operant behavior from respondent behavior in a number of ways, these processes occur concurrently and blend almost indistinguishably. For example, the administration of a drug may elicit certain behavioral and physiological responses such as increased heart rate and changes in perception that are respondent in nature; stimuli associated with the administration of that drug may also acquire some of the same ability to elicit those responses. If the administration of the drug followed a response and if the subsequent frequency of that response increased, then the drug also could be designated a reinforcer of the operant response. Thus, these important behavioral processes frequently occur simultaneously and must be considered carefully in experimental research, and also in attempting to understand the control of behavior by abused drugs. The primary distinctions between operant and respondent behavior now appear to be the way these behaviors are produced and the possible differential susceptibility to modification by consequent events. Respondent behavior is produced by the presentation of eliciting stimuli; characteristic features of these behaviors are rather easily changed by altering the features of the eliciting stimulus such as its intensity, duration, or frequency of presentation. Under all of these conditions, however, the response remains essentially the same.

In contrast, operant behavior depends to a large extent on its consequences, and with this process, complex behavior can develop from quite simple relationships. One has only to view current behavior as an instance of the organism's previous history acting together with more immediate environmental consequences to gain some appreciation for the continuity and modification of behavior in time. Current behavior is often exceedingly difficult to understand because of the many prior influences or consequences that no longer operate but which may leave residual effects. The effects of a particular consequence or intervention can be quite different depending on the behavior that exists at the time the event occurs. An individual's prior history, then, is important not only because it has shaped present behavior but also because it will undoubtedly determine the specific ways in which the individual responds to the current environment. Accordingly, prior behavioral experience can have a marked effect in determining how a drug will change behavior.

BEHAVIORAL METHODOLOGY AND THE EVALUATION OF ABUSED DRUGS

Experiments with drugs and behavior were initiated in Pavlov's laboratory in Russia during the time that Pavlov was studying the development of conditioned respondent procedures (see Laties, 1979, for a review of this early work). Early experiments with the effects of drugs on operant behavior were initiated shortly after Skinner began his pioneering work (Skinner & Heron, 1937). More intensive studies using drugs and operant-conditioning techniques were not conducted, however, until effective drugs for the treatment of various psychiatric disorders such as SCHIZOPHRENIA were introduced in the 1950s. These discoveries prompted the development and extension of behavioral techniques to study these drugs, and many of the procedures were subsequently used in the study of abused drugs. From these combined efforts, several key principles evolved that have served as the foundation for understanding and evaluating the effects of abused drugs.

Environmental Events. As already discussed, behavior can be controlled by a wide range of environmental events. One question that arose

early in the study of the behavioral effects of drugs was whether the type of environmental event that controlled behavior contributed to the effects of a drug—that is, whether a behavior controlled by a positive event, such as food presentation, would be affected in the same manner as a behavior controlled by a more negative event, such as escape from an unpleasant noise or bright light. Although seemingly straightforward, the issue is not easily addressed because other known factors contribute to the actions of drugs, such as the rate at which a behavior controlled by the event occurs. If rates are not similar, any comparison between drug effects on behavior controlled by those different events might be spurious. Indeed, when such comparisons have been conducted in nonhuman primates under carefully controlled conditions, it has been shown that the type of environmental event controlling behavior can play an important role in determining the qualitative effects of a drug on behavior (Barrett & Witkin, 1986; Nader, Tatham, & Barrett, 1992). For example, when the effects of certain drugs such as ALCOHOL or MORPHINE were studied by using behavioral responses of monkeys who were similarly maintained by a food stimulus or a mild electric-shock stimulus, the drugs produced different effects depending on the maintaining event (Barrett & Katz, 1981). These findings suggest that the manner in which behavior is controlled by its environmental consequences—that is, the characteristics of the environment—can be of considerable importance in determining how an individual will be affected by a particular drug. This was one of the experiments that supported the view that a drug is not a static molecule with uniform effects, but rather that the way the substance interacts with its receptor and initiates the cascade of biochemical processes depends very much on the dynamic interaction of behavior within its environment. When the issue is viewed in this light, it is clear that environmental events and the way they impinge on behavior contribute substantially to the specific effects of a drug and its impact on the individual organism.

Examples of similar types of environmental control over pharmacological effects of drugs also come from studies that employed respondent conditioning procedures to demonstrate that stimuli paired with morphine or heroin injections can influence the development and manifestation of fundamental pharmacological processes such as tolerance and lethality (Siegel, 1983). These studies add to the rather convincing body of evidence that environmental conditions accompanying the administration and effects of the drug can be of considerable importance in determining the effects of that drug when it is administered, as well as when it is subsequently administered.

Behavioral and Pharmacological History. In addition to pointing to the contribution of the immediate environment in determining the effects of abused drugs, a number of studies demonstrated that the consequences of *past* behavior could also contribute significantly to the effects of drugs, often by resulting in an action that is completely opposite to that shown in organisms without that history. These findings convey the complexity involved in understanding the effects of drugs of abuse, and the difficulties in attempting to understand their actions in humans with more complex life histories than those of experimental animals. In addition, related studies showed that prior experience with one drug could also directly affect the manner in which behavior is influenced by other drugs.

Early studies using different training conditions to develop a visual discrimination in pigeons demonstrated that an antipsychotic drug, Thorazine (chlorpromazine), and an antidepressant drug, imipramine, had different effects on that discriminative behavior, depending on how the training occurred (Terrace, 1963). Similarly, studies that used exploratory behavior of rats in mazes demonstrated that the effects of a mixture of amphetamine (STIMULANT) and a BARBITURATE drug (DEPRESSANT) depended on whether the rats had been previously exposed to the maze (Steinberg, Rushton, & Tinson, 1961). More recently, studies with squirrel monkeys showed that prior behavioral experience can influence the effects of a wide range of drugs, including morphine, cocaine, and amphetamine, as well as alcohol, under a variety of experimental conditions (summarized by Barrett, Glowa, & Nader, 1989; Nader et al., 1992). In one study, for example, the effects of amphetamine were studied on behavior reinforced by food that was also suppressed by punishment. Under these conditions, amphetamine produced a further decrease in punished responding. If those same monkeys, however, were then exposed to a procedure in which responding postponed or avoided punishing shock and were then returned to the punishment condition, amphetamine no longer decreased re-

sponding; instead, it produced large *increases* in suppressed responding. Thus, the effects of amphetamine in this study depended on the prior behavioral experience of the animal.

These findings raise a number of issues surrounding the etiology of drug abuse as well as issues pertaining to an individual's risk for or vulnerability to abusing particular drugs. If, as seems likely, certain drugs are abused because of their effects on behavior, and those behavioral effects are related to past history, then the historical variables become exceptionally important in eventually understanding and treating, as well as preventing, drug abuse. Perhaps previous behavioral experience generates conditions under which a drug may have quite powerful actions on behavior and on the subjective effects that drug produces; by virtue of their previous history, the susceptible individuals may be predisposed to drug abuse. If these arguments are valid, it should be possible, after achieving a better understanding of the factors, to develop behavioral strategies for "inoculating" or "immunizing" individuals against particular drug effects. Although such possibilities may seem remote at this time, it is very clear that behavioral variables can direct the effects of abused drugs in striking and significant ways.

SUMMARY

Although drugs of abuse have a reliable and predictable spectrum of effects under a broad range of conditions, the implications from studies are that many of the more characteristic effects of abused drugs can be altered by the organism's history and by the environmental conditions under which the drug is and has been administered. As Folk (1983) said so eloquently, "Pharmacological structure does not imply motivational destiny"; the reasons for the effects of an abused drug depend on more than the static molecular properties of that drug. Both past and present environmental factors can play an overwhelming role in determining the behavioral effects of abused drugs, and they may indeed be a major source of the momentum behind the continued use and abuse of those substances.

(SEE ALSO: *Abuse Liability of Drugs; Addiction: Concepts and Definitions; Adjunctive Drug Taking; Causes of Substance Abuse; Reinforcement; Vulnerability as Cause of Substance Abuse*)

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Learning Modifies Drug Effects A general framework within which to understand the basic processes and principles of respondent conditioning (as first discovered in the 1920s by Russian physiologist Ivan Pavlov [1849–1936] and subsequently elaborated in many laboratories over the next six decades) is described elsewhere. Here, specific examples of the role of conditioned drug effects are provided in an effort to more fully develop the point that conditioned or learned responses come about as a reaction to stimuli that have been associated with drug injections. These stimuli can play a powerful role in governing subsequent behavior in the absence of the drug.

CONDITIONED EFFECTS OF DRUGS

In addition to studies described previously showing that tolerance to the effects of a drug, as well as lethality, can depend on respondent-conditioning phenomena, a number of additional studies have demonstrated the conditioning of WITHDRAWAL and other responses that are typically associated only with the presentation or removal of the drug. For example, by pairing a tone stimulus with the administration of nalorphine, an OPIOID ANTAGONIST that precipitates withdrawal signs or the abstinence syndrome (agitation, excessive salivation, and emesis) in morphine-dependent subjects, it was possible to show in rhesus monkeys that the tone acquired the ability to elicit withdrawal responses when presented in the absence of natorphine (Goldberg & Schuster, 1967; 1970). Striking illustrations of similar conditioned withdrawal responses in HEROIN addicts, as well as CRAVING, in which environmental stimuli trigger the disposition to self-administer the drug, also have been described. These behavioral responses to stimuli that have been previously associated with drug withdrawal or administration often occur after a prolonged period of time spent without drugs (O'Brien, 1976).

In some cases, drugs also acquire stimulus control over behavior in a procedure known as *state-dependent learning*. This procedure is different in some ways from that used to study drugs as discriminative stimuli. State-dependent learning refers to the finding that subjects exposed to a particular procedure when injected with a drug often are impaired upon reexposure to that condition if the drug is not present. Thus, the drug can be viewed as part of the original context in which a response was learned. One concern that stems from the finding that behavior learned during a drug-related condition is impaired in the absence of the drug is that of the potentially enduring problems related to frequent abuse of drugs during adolescence—a period often associated with major developmental and cognitive growth.

REINFORCING EFFECTS OF DRUG-PAIRED STIMULI

Thus far, the focus has been on the effects of environmental stimuli paired with the administration of a drug rather than on stimuli paired with a drug as a reinforcer. As has been frequently demonstrated, and as is true of many stimuli, drugs can have multiple functions. These include *discriminative* effects, which set the occasion for certain responses to occur, and they also include *reinforcing* effects, whereby a response is increased in probability when a reinforcing drug follows the occurrence of that response. Drug self-administration techniques have been very informative and useful in the study of the effects of abused drugs.

One additional experimental procedure that has been used in this field of research is that of repeatedly pairing a rather brief visual or auditory stimulus (e.g., a light or a tone, respectively) with the reinforcing administration of the drug and then using that stimulus also as a reinforcer to maintain behavior without drug administration. Perhaps the most compelling work in this area stems from a procedure in which a stimulus was presented according to a schedule to follow a particular response. On certain occasions, that stimulus also was associated with the administration of a drug—that is, the stimulus occurred at various times without the drug and then also just preceding the drug. Known technically as a "second-order schedule," this technique exerts powerful control over the occurrence and patterning of behavior, and it results

in sustained responding for extended time periods in the absence of anything but the stimuli that have been paired with the administration of the drug itself (Katz & Goldberg, 1991). In other words, conditioned stimuli that have been paired with a drug can exert considerable control over behavior.

SUMMARY

To summarize, conditioned drug effects play an important role in the behavior stemming from drug abuse. Stimuli correlated with the administration of a drug, as well as behavior in the presence of that drug, frequently result in those stimuli gaining considerable control over the discriminative effects or reinforcing effects of that drug (or both). Perhaps this is one of the main reasons that drug effects are so compelling and problematic: Not only does the drug itself have powerful effects, but stimuli correlated with the drug also acquire the ability to produce similar effects.

(SEE ALSO: *Addiction: Concepts and Definitions; Causes of Substance Abuse; Memory and Drugs: State Dependent Learning; Research*)

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JAMES E. BARRETT

Operant Learning Is Affected by Drugs

According to psychologist B. F. Skinner, behavior that is rewarded or *reinforced* is more likely to occur again. The family dog soon learns that hanging around the kitchen table brings food. In this example, the food is a reinforcer because it increases the likelihood that the dog will spend time near the kitchen table. Thus, the dog's behavior "operates" on the environment to produce an effect. This process is called *operant conditioning*. The techniques of operant conditioning are used widely to establish new behaviors both in humans as well as in animals. Because behavior that is operantly conditioned is very sensitive and reliable, it is often used to examine drug effects.

A TYPICAL OPERANT CONDITIONING EXPERIMENT

In most operant conditioning experiments, an animal is placed in a special chamber which is called a Skinner box after the man that developed operant conditioning. A typical operant chamber, which is shown in Figure 1, has a response key or lever and a place for delivering food. The animal's responses are counted by a computer and also recorded on a roll of paper that shows the distribution of responses over time. Although the experimental chamber in Figure 1 is designed for animals, operant conditioning procedures are also used to examine drug effects in humans. In these studies, the person may sit in a chair and respond by moving a joystick or perhaps sit at a keyboard and respond to stimuli on a computer screen.

SCHEDULES OF FOOD DELIVERY

In most operant conditioning experiments in animals, responses on a lever or key produce food according to some schedule. Behavior maintained by a schedule of reinforcement is called *schedule-controlled behavior*. For example, the pigeon or rat may have to make a specific number of responses in order to receive food. When this occurs, the organism is responding under a *fixed ratio schedule*. A similar schedule is the *variable ratio schedule* in which reinforcement occurs after an unpredictable number of responses. With both the fixed ratio and the variable ratio schedules, animals respond very quickly, in fact, under a fixed ratio schedule that requires thirty responses for food delivery, pigeons

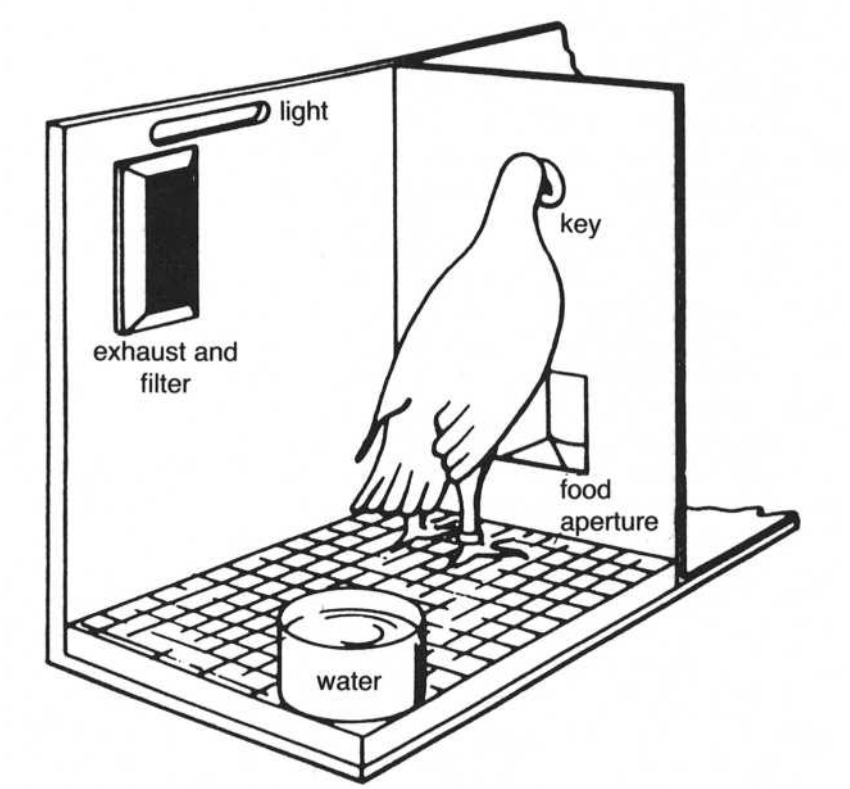

Figure 1
Diagram of an Operant Conditioning Chamber. When the pigeon presses the key, food is delivered. A separate device counts the number of times the pigeon pecks the key.
SOURCE: L. S. Seiden, & L. A. Dykstra (1977).

may respond as fast as five times a second. Another operant schedule is the *fixed interval schedule* in which the first response that occurs after a specified period of time produces food. With this schedule, rates of responding increase as the time for food delivery approaches. For example, in a fixed interval five-minute schedule. responding is very low during the first two minutes of the interval; responding picks up speed during the third and fourth minutes of the interval and becomes very rapid during the last minute, just before the food is delivered.

By comparing drug effects on different schedules of REINFORCEMENT, scientists have shown that the way in which a drug alters responding depends on the rate of responding produced by a given schedule of reinforcement as well as the amount (or dose) of drug given. Thus, a drug's effects are *rate-dependent* as well as *dose-dependent*. The rate-dependency theory of drug action was first proposed by Peter Dews in the early 1960s and is best exemplified by the effects of amphetamine. Dews noted that amphetamine alters responding differently under a schedule of reinforcement that produces low rates of responding than under a schedule of reinforcement that produces high rates of responding. Specifically, a small amount of AMPHETAMINE increases very low rates of responding, whereas the same amount of amphetamine either decreases or does not change high rates of responding. Other drugs in the amphetamine class such as COCAINE and METHYLPHENIDATE (Ritalin) also alter responding in a rate-dependent manner.

One of the most interesting aspects of the rate-dependency theory of drug action is that it emphasizes the importance of behavioral as well as pharmacological factors in determining a drug's effect on behavior. Thus, the rate at which an animal responds is an important determinant of the way in which a drug alters behavior. It also helps to explain why drugs such as amphetamine and methylphenidate, which generally increase motor activity, might be useful in treating hyperactivity. Since hyperactive children respond at a very high rate, amphetamine would be expected to decrease these high rates of responding.

In contrast to the rate-dependent effects observed for amphetamine-like drugs. the most notable effect of drugs such as MORPHINE is that they decrease rates of responding under several different schedules of reinforcement. The extent to which morphine decreases rate of responding depends on how much morphine is given. Thus, its effects are dose-dependent. Moreover, like all schedule-controlled behavior, these decreases in rate of responding are very consistent and reliable. If a rat is trained to respond under a fixed ratio schedule of food presentation and then given morphine, morphine will decrease rates of responding by about the same amount each time it is given; however, if morphine is given daily for a week or more, its rate-decreasing effects diminish. In other words, TOLERANCE develops. Interestingly, the development of tolerance depends on the behavior examined as well as how much drug is given.

Morphine's effects on responding under schedules of reinforcement are also used as a baseline to investigate the biochemical and physiological events that occur when morphine is given. Opioid antagonists, which block the binding of morphine to opioid receptors, are able to reverse morphine's effects on schedule-controlled behavior. Since morphine's effects on responding are blocked when opioid receptors are blocked, these data suggest that morphine produces its behavioral effects by interacting with opioid receptors. Responding under schedules of reinforcement is also used to examine the biochemical and physiological effects of other drugs. For example, amphetamine's effects on schedules of reinforcement are altered by drugs that interfere with the neurotransmitter dopamine, suggesting that the dopamine system is involved in amphetamine's behavioral effects.

SCHEDULES OF PUNISHMENT

Although schedule-controlled behavior generally is maintained by the delivery of food, in some situations, responding is punished by the presentation of an unpleasant event. In a typical punishment procedure, responding is first maintained by a schedule of food delivery. Brief periods are then added during which responding is both reinforced by food and also punished by an unpleasant event. As a result, responding occurs at a lower rate during periods in which responding is punished than during unpunished periods. Figure 2 shows the design of a typical punishment procedure. First, note in the first panel that responding maintained by food alone occurs at a high rate. In the second panel, responding is punished by the addition of an unpleasant event and, as a result, rate of responding is decreased during the punishment period. The third panel shows that a drug such as alcohol selectively affects responding during the punishment period by restoring rates of responding to their baseline levels. Because these increases in punished responding occur following alcohol as well as a number of other antianxiety agents, but not following drugs such as morphine or amphetamine, increases in punished responding may reflect the antianxiety properties of these drugs. Indeed, the punishment procedure is used by a number of pharmaceutical companies to predict whether a drug might be useful in treating anxiety.

SCHEDULES OF REINFORCEMENT AS A WAY TO MEASURE LEARNING

Schedules of reinforcement are also used to examine the rate at which new behaviors are learned. Clearly, it takes some time to train an animal to respond under a schedule of reinforcement. This period of training is called the acquisition period and provides a measure of learning. One way to design a learning experiment is to measure how long it takes a group of rats to learn to respond under a schedule of reinforcement when a drug is given and compare that to how long it takes another group of rats to learn the same task without a drug. In experiments such as these, animals are usually trained to respond under very complicated schedules of reinforcement. Sometimes the animal has to complete the requirements of several different schedules in order to obtain food; in other procedures, the animal responds differently in the presence of different kinds of stimuli. In another procedure, the time it takes an animal to learn a pattern of responses is determined when a drug is given and compared to the time it takes the same animal to learn a different pattern of responses without a drug. ETHANOL, the BARBITURATES, and several antianxiety drugs all increase the number of errors animals make in learning new response sequences. Studies using a similar procedure in humans show that ethanol and certain antianxiety drugs also increase the number of errors people make when they learn new response sequences.

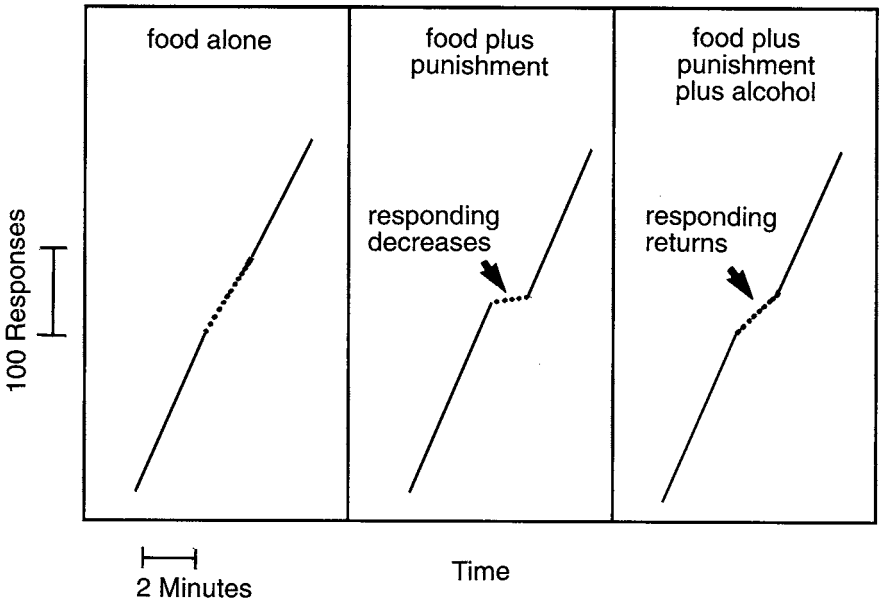

Figure 2
Diagram of a Typical Punishment Procedure. In the first panel, responding is maintained by food alone. The second panel shows responding maintained by food as well as responding during a period in which responding is punished. During this period, responding is decreased. The third panel shows the effects of ethanol on punished responding.

SUMMARY

Schedules of reinforcement offer several advantages for studying the behavioral effects of drugs. First, schedule-controlled responding is very consistent and remains unchanged for long periods of time. This consistency makes it easy to examine changes in behavior after a drug is given. Second, schedule-controlled behavior can be used with human subjects as well as with several different animal species, including mice, rats, pigeons, and monkeys. Finally, schedule-controlled behavior is recorded with automatic devices so that the experimenter is completely removed from the experiment and the nature of the behavior is easy to measure. From these studies, several important concepts have emerged. Scientists have shown that the behavioral effects of drugs depend not only on the amount of drug given, but they also depend on the nature of the behavior being examined. Both the rate of occurrence of a behavior as well as the presence of punishing stimuli are very important determinants of how drugs alter behavior.

The PSYCHOMOTOR STIMULANTS increase responding under schedules of reinforcement when responding occurs at a low rate; when responding occurs at higher rates, the psychomotor stimulants decrease rates of responding. The most notable effect of morphine is that it decreases overall rates of responding. Alcohol and the antianxiety agents are unique in that they increase responding that is suppressed by the presentation of a punishing stimulus. Finally, several drugs interfere with the learning of complex patterns of responding.

(SEE ALSO: *Adjunctive Drug Taking; Behavioral Tolerance; Memory and Drugs: State Dependent Learning; Memory, Effects of Drugs on; Reinforcement; Tolerance and Physical Dependence*)

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RESEARCH AND THE U.S. GOVERNMENT *See* U.S. Government: Agencies Supporting Substance Abuse Research

REWARD PATHWAYS AND DRUGS The observation that animals would work in order to receive electrical stimulation to discrete brain areas was first described by Olds and Milner (1954). In this paper, they stated, "It is clear that electrical stimulation in certain parts of the brain, particularly the septal area, produces acquisition and extinction curves which compare favorably with those produced by conventional primary reward." This phenomenon is usually referred to as *brain-stimulation reward* (BSR), *intracranial self-stimulation* (ICSS), or *intracranial stimulation* (ICS).

Most abused substances increase the rate of response (lever pressing) for rewarding ICS, and this has been interpreted as an increase in the reward value of the ICS. Because changes in rate of response could also be a function of the effects of the drug on motor performance, a number of methods have been developed that control for the confounding nonspecific effects of the drugs under study, at least in part. The three most commonly used procedures are phase shifts (Wise et al., 1992), two-level titration (Gardner et al., 1988), and the psychophysical discrete-trial procedure (Kornetsky & Porrino, 1992). Using these threshold methods for determining the sensitivity of an animal to BSR, there is general agreement that most of the commonly abused substances do in fact increase the sensitivity of animals to the rewarding action of the electrical stimulation and this action is independent of any motor effects of the substances.

PHASE SHIFTS

In this method, rates of response are determined at various intensities of stimulation. Data are usually presented as rate-intensity (rate-frequency) functions. If a drug shifts the rate-intensity function to the left, it is interpreted as an increase in sensitivity of the animal to the rewarding stimulation. A shift to the right is interpreted as a decrease in sensitivity. Threshold (sometimes called *locus of rise*) is defined as the intensity that yields half the maximum rate of response for the animal. If the maximum rate becomes asymptotic at approximately the same stimulus intensity as observed after saline, it is assumed that any phase shift is a direct effect of the drug on the reward value of the stimulation, not the result of a nonspecific motor effect of the drug.

TWO-LEVER TITRATION

In this procedure, rats are placed in a chamber with two levers; pressing one of the levers results in rewarding stimulation, but at the same time the response attenuates the intensity of stimulation by a fixed amount. A response on the second lever resets the intensity to the original level. The threshold is defined as a mean intensity at which the reset response is made.

PSYCHOPHYSICAL DISCRETE TRIAL METHOD

A wheel manipulandum is usually used, although the method has been employed using a response lever. In this method, discrete trials are used, each demanding only a single response by the rat in order to receive the rewarding stimulation. A trial consists of an experimenter-delivered (noncontingent) stimulation. If the animal responds by turning the manipulandum within 7.5 seconds, it receives a second stimulation at the identical stimulation intensity as the first stimulus. Current intensities are varied in a stepwise fashion or descending and ascending order. This yields a response-intensity function, with the threshold defined as the intensity at which the animal responds to 50 percent of the trials. Of the methods currently used, this is the only one that does not make use of the response rate as an integral part of the procedure for the determination of the reward threshold—thus it is independent of the rate of response and the possible confounding motor effects of the drug.

DOPAMINE AND ICS

Although most abused drugs lower the threshold for ICS for some drugs, the findings have not always been consistent, particularly with HALLUCINOGENS and the SEDATIVE-HYPNOTICS, including ALCOHOL (ethanol). For the most part, the threshold-lowering effects caused by the abused substances are compatible with the hypothesis that facilitation of DOPAMINE is involved in their rewarding effects. Drugs that increase dopamine availability at the synapse facilitate ICS, and those that block dopamine transmission decrease ICS (i.e., they raise the threshold—or the amount of current—needed to produce rewarding effects).

DOPAMINE

Because abused substances clearly enhance the rewarding value of the intracranial stimulation and not simply cause a general increase in motor behavior, the brain-stimulation-reward model directly allows for the study of the neuronal mechanisms involved in the rewarding effects of abused substances. Although this is not as homologous a model of drug-taking behavior as is the self-administration model, it predicts as well as the self-administration model the ABUSE LIABILITY of compounds, and it readily lends itself to analysis of the mechanisms involved in the rewarding effects of abused substances.

(SEE ALSO: *Research, Animal Model*)

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CONAN KORNETSKY

RITALIN *See* Methylphenidate

ROCKEFELLER DRUG LAWS The Rockefeller drug laws are a set of New York MANDATORY SENTENCING statutes for drug crimes. They were proposed by New York's Governor Nelson A. Rockefeller in reaction to a HEROIN epidemic in his state. These laws, which took effect on September 1, 1973, require that judges impose lengthy prison sentences on drug traffickers, with a large category of drug offenders receiving life imprisonment. The goal was to deter people from both drug use and trafficking by imposing tough and certain punishments. Although the law was immediately challenged as violating the Cruel and Unusual Punishment clause of the U.S. and New York constitutions, the New York Court of Appeals unanimously upheld the law

Within a few years, however, the state's prison population began to swell, as increasing numbers of defendants were subjected to the provisions of the Rockefeller laws. From 1969 to 1979, the prison population doubled, from 12,000 to 24,000. In the same time period, the percentage of incarcerated nonviolent drug offenders increased from 10 percent to over 30 percent. In spite of these laws, the crime rate continued to grow. A major evaluation concluded that neither drug use nor drug trafficking was reduced after the law was passed. The likelihood that a defendant, once arrested, would be incarcerated did not increase—although the likelihood that a defendant, once convicted, would be imprisoned did increase (Joint Committee on New York Drug Law Evaluation, 1977).

The processing of cases became much more expensive for New York. For every crime affected by the law, the percentage of defendants pleading guilty fell, and the proportion of trials increased. The evaluators concluded that it "took between ten and fifteen times as much court time to dispose of a case by trial as by plea." The average time to handle a drug prosecution in New York City, for example, doubled, rising from 172 days in 1973 to 351 days in 1976.

Although the legislature realized the ineffectiveness of the stated purposes of the laws, neither it nor a succession of governors has proposed repealing the laws. Instead, the legislature has sought to amend the laws in ways that reduce their scope. In 1977, the legislature removed marijuana from the definition of crimes dealing with controlled substances and created a new sentencing law for marijuana sale and possession. The possibility of life imprisonment for marijuana offenses was eliminated.

The legislature tinkered with the laws again in 1979. This time it increased the amount of weight of the drug necessary to trigger higher-level felonies. It also reduced the minimum sentence range for certain drug convictions and eliminated a classification from the statute. The 1979 amendments also gave the courts the ability to retroactively resentence defendants who had been convicted based on the original weight and classification schemes.

Despite these changes, they have done little to reduce the harshness of the sentencing practices or reduce the prison population. In 1998, the state prisons held 70,000 inmates, three times the number incarcerated in 1979. Most significantly, 30 percent of the prison population is comprised of nonviolent drug offenders.

By the late 1990s, many in the legal community argued for repeal of the Rockefeller laws, believing that they imposed disproportionate punishment on nonviolent drug offenders and ignored drug treatment options. However, Governor George Pataki responded in 1999 with only a minor change in the laws. Pataki proposed legislation that slightly alters the laws by offering first-time drug couriers a chance to cut their sentences by five years. Under this proposal, the appellate courts would be allowed to review and reduce sentences by five years for first-time felony offenders under the harshest provision of the laws, which now calls for a maximum of fifteen years to life. This proposal was similar to one proposed by Chief Judge Judith S. Kaye, who also called for allowing trial judges to defer the prosecution of nonviolent drug offenders for up to two years and to divert them to drug treatment programs. However, the legislature did not act on these reform efforts, leaving the status quo in place.

(SEE ALSO: *Drug Laws, Prosecution of; Opioids and Opioid Control: History*)

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ROHYPNOL Known by a variety of street names such as roofies, roach, R-2, trip and fall, and rope or "the date-rape drug," rohypnol is the trade name for the benzodiazepine FLUNITRAZEPAM, a sedative-hypnotic drug used medically in a number of countries. Rohypnol has recently become a widely abused drug in Sweden, Mexico, Italy, the United Kingdom, the United States, and South Africa, a trend made more troubling by the fact that many users regard it as relatively safe. Rohypnol, in fact, has many dangerous and undesirable effects for the illicit user. It has been associated with an increased risk of violence and accidents as well as stupor, coma, memory loss, and death. Its ability to induce unconsciousness and amnesia has led to its use in sexual assaults in the United States (hence, its reputation as a date-rape drug) as well as robberies.

Although never approved for use in the United States (where it is illegal) rohypnol is a commonly prescribed BENZODIAZEPINE in Europe and elsewhere. Like other benzodiazepines, such as VALIUM (DIAZEPAM) or Xanax (Alprazolam), it is useful in the medical treatment of sleep disorders and anxiety, though only under supervision by a doctor. Benzodiazepines act at brain receptors for the inhibitory neurotransmitter GABA, which is also the site of action for another, older class of sedative-hypnotic drugs and barbiturates. Although generally safer than barbiturates, benzodiazepines like rohypnol share some of the same dangers especially when mixed with ETHANOL, a common practice among illicit drug users. These dangerous effects range from incontinence, behavioral disinhibition, violence, delirium, and black-outs to stupor, respiratory depression, and death. These effects all stem from the ability of rohypnol to depress brain function.

At lower doses, benzodiazepines can reduce anxiety and cause relaxation and a loosening of inhibitions somewhat similar to the effects of ALCOHOL, another drug that acts as a depressant on the central nervous system. As with many abused drugs, the continued use of rohypnol results in increased tolerance, requiring larger doses to produce the same effects. Larger doses mean narrower margins of safety and the increased incidence of side effects, especially memory loss and deficits in learning. Drinking alcohol in combination with rohypnol makes serious consequences all the more likely. Of still greater concern for the illicit user is that chronic use of sedative-hypnotic drugs like rohypnol can produce a level of physiologic dependence greater than that resulting from OPIATE drugs like HEROIN or MORPHINE. Abrupt WITHDRAWAL from regular use can produce complications ranging from the relatively mild, such as restlessness and anxiety, to more severe effects like tremor, hallucinations and convulsions similar to those experienced during severe alcohol withdrawal. These complications can be best avoided through a medically supervised withdrawal.

Rohypnol has received much media attention in the United States for its apparent involvement in a number of sexual assaults or rapes. Because it can quickly render an unsuspecting victim unconscious, rohypnol lends itself to this kind of crime. As rohypnol is odorless and tasteless and easily dissolved in drinks, it can be offered to a victim without arousing suspicion. Although media attention has focused on particular drugs like rohypnol and GHB, it should be noted that a variety of drugs can and are being used in this manner, including barbiturates, opiates, other benzodiazepines and ethanol. Ethanol remains several times more likely to be associated with sexual assault than any other drug, including rohypnol, even though rohypnol and drugs like it are more effective in rapidly producing the stupor and memory loss desired by this type of criminal.

RICHARD G. HUNTER

ROLLESTON REPORT OF 1926 (U.K.)

The Rolleston Report of 1926 helped to establish British policy toward OPIATES, COCAINE, and other drugs. It institutionalized a drug policy in which medical expertise and public-health considerations were given importance along with punishment and criminal penalties. The British policies were, in this sense, different from U.S. policies toward drugs that emerged during the same period and in response to similar international agreements. The historical background leading to the formation of an elite committee of British physicians, chaired by Sir Humphrey Rolleston, had four major phases.

ENDING THE COMMERCIAL OPIUM TRADE

During the nineteenth century, the British established commercial opium trading by fighting and winning two Opium Wars with China: Opium grown and sold by monopoly in British-dominated India provided a quarter of the revenue for the British government in India. Prepared opium (for smoking) was exported to Chinese ports by the East India Company, where British authorities collected tax revenues on it for the Chinese government. Missionaries in China and their anti-opium allies in Britain, the United States, and Canada lobbied strongly against profiting from the British-sponsored vice. They also educated the public about opium smoking and commercial opium trading.

The U.S. government stimulated the convening of several international conferences from 1909 to 1914. These conferences reached agreements that all signatory governments would enact legislation ending commercial opium trading and restricting opium and cocaine to "legitimate medical practice." The Indo-Chinese opium trade ended in 1914. These international conventions were included in the Versailles Treaty that ended World War I. "Legitimate medical practice" and appropriate controls and/or penalties were not specified in the international treaties.

OPIUM CONTROLS AND GROWTH OF THE MEDICAL PROFESSION

During the nineteenth century, opiates were the only effective way to relieve the symptoms of many physical ailments (most medicines used today, including aspirin, became available only in the twentieth century). OPIUM and its derivative MORPHINE (Britain was the world's leading manufacturer) were available in patent medicines, in alcoholic solutions, and in other commercial products. The emerging professions of pharmacist and medical

physician with advanced training and specialized knowledge were anxious to differentiate themselves from a motley group of healers—chemists, herbalists, barber-dentists, patent-medicine sellers, and others. In the 1850s, such persons could provide opiates to patients since they were not then illegal, and preparations containing opiates provided substantial revenues. Opium eating and LAUDANUM (an alcoholic solution of opiates) consumption were then widespread in Britain.

British pharmacists became eager to restrict sales of opiates to qualified sellers—but only in such a way that "professional" trade would not be harmed and could be expanded. The 1868 Poisons Act restricted opiate sales to pharmacists. This act mandated the labeling of opiates and required pharmacists to keep records of purchasers. (Similar restrictions on opiate sales in the United States did not occur until the 1906 Food and Drug Act.) Pharmacists, however, could continue to sell opiates directly to customers without a prescription from a physician, and physicians could prescribe or sell opiates to patients. In the early 1880s physicians and researchers in Europe, England, and the United States almost simultaneously began to write about the opium habit and morbid cravings for opiate drugs. In 1884 physicians in England founded the Society for the Study of Inebriety, which promoted a disease model of addiction and the need for treatment.

By 1900, physicians emerged as an elite group who defined all aspects of health care and medical practice in British society; pharmacists "policed" the Poisons Act and effectively retained control of dispensing opiates and other drugs. Thus, by 1914, British pharmacists and physicians had almost a half century of experience, professional collaboration, an ongoing professional association concerned with the dispensing of opiates, and attempts to contain opiate consumption and habitual use.

PRESSURE TOWARD CRIMINAL PENALTIES

In 1914, when the international opium convention (Hague Convention) was to go into effect, several British agencies could not decide which one should take responsibility for implementing legislation and regulation of drugs. Then World War I began in August 1914 and Sir Malcolm Delevingne, an undersecretary at the Home Office, took primary responsibility. He suggested using the War Powers Act to stop sales of cocaine and opiates to soldiers unless they were based on a prescription by a doctor that was "not to be repeated" (refilled without further prescription). Violators, however, could be fined only five pounds. Two or three cases were publicized and introduced the British public to "dope fiend" fears, but they continued to be rare.

After World War I, Delevingne argued that drug control was a police responsibility for the Home Office (where it has remained ever since). The 1920 Dangerous Drug Act was vague about two critical issues—whether doctors/pharmacists could prescribe for themselves, and whether doctors could "maintain" addicts. In 1921 and 1924, the Home Office proposed regulations that ignored the rights of professionals and imposed many complex procedures. It also sought powers of search and seizure, higher fines, and longer sentences for convictions. Thus, the Home Office was making regulations that would subject doctors to criminal sanctions and circumscribe their prescribing practices—as was already happening in the United States.

APPOINTMENT OF THE ROLLESTON COMMITTEE

The Home Office needed the cooperation of the medical profession to determine the appropriateness of maintenance dosages for addicts, and it sought to determine whether gradual reduction was the appropriate treatment for addiction. The Home Office and the medical profession each recognized the legitimacy of the other's position. Both realized that a partnership was needed. Thus, these two elite groups began a collaboration to define and resolve problems and appropriate practices regarding narcotics control. All persons appointed to the committee were medical personnel representing government agencies or nongovernment physician-interest groups. The chairman, Sir Humphrey Rolleston, was president of the Royal College of Physicians and a noted exponent of the disease view of ALCOHOLISM. Another member had written the authoritative article on narcotic addiction in 1906. Police and law enforcement officials without medical training were not represented.

Committee Deliberations and Recommendations. The committee was to consider and advise as to the circumstances, if any, in which the supply of morphine and heroin (including prepara-

tions containing morphine and heroin) to persons suffering from addiction to these drugs, may be regarded as medically advisable and as to the precautions which it is desirable that medical practitioners administering or prescribing morphine or heroin should adopt for the avoidance of abuse, and to suggest any administrative measures that seem expedient for securing observance of these precautions.

During a year and a half of deliberations and twenty-three meetings, the committee heard evidence from thirty-four witnesses. The Home Office submitted a memorandum that structured the questions and inquiry. Witnesses represented a wide diversity of opinion, particularly regarding appropriate treatment for addicts. Prison doctors favored harsher treatment, especially abrupt withdrawal of opiates (going cold turkey). Even consultants specializing in treatment rarely agreed on points of procedure and treatment. Most witnesses and commission members accepted the disease nature of addiction.

There was wide agreement, however, that addiction to HEROIN or morphine (both opiates) was a rare phenomenon and a minor problem in BRITAIN. Most addicts were middle class and many were members of the medical profession. Relatively few criminal or lower-class addicts were then known, so criminal sanctions appeared unneeded and inappropriate. The committee report concluded that "the condition must be regarded as a manifestation of disease and not as a mere form of vicious indulgence."

From this conclusion, many recommendations followed. The most important was that some addicts might need continued administration of morphine (or other opiates) "for relief of morbid conditions intimately associated with the addiction." Thus, the committee effectively supported maintenance of an addict for long periods of time, possibly for life.

The committee also made several recommendations for administrative procedures to lessen the severity of the drug problem. Practitioners were mandated to notify the Home Office when they determined someone was addicted; but physicians could continue to provide treatment and prescribe opiates to addicts. Gradual reduction rather than abrupt withdrawal was the recommended treatment, in part to keep addicts in treatment rather than to drive them to illicit suppliers. A medical tribunal was established to promote the profession's own policing of members who became addicted. The committee also opposed banning heroin (which was a useful medication and a very small problem in Britain at the time).

LEGACY OF THE REPORT

Shortly after the Rolleston Report was completed, its recommendations were included in amendments to the Dangerous Drug Act (1926). Although this act has been amended numerous times since then, the provisions adopted from the Rolleston Report remain in effect in the 1990s. Although cocaine was included as a narcotic in this report, separate recommendations for treatment were not made. *Cannabis* (MARIJUANA) was not included in this report. The Rolleston Report did not address the issue of illegal sales or transfers of opiates; no criminal or penal sanctions were recommended.

The *British Medical Journal* was content: The medical view of addiction as a disease needing treatment, and not a vice necessitating punishment and penal sanction, had been formally accepted as government policy. Medical professionals, rather than criminal-justice personnel, would be responsible for individual decisions about whether patients were addicts, and prescribe appropriate quantities of opiates, including on a maintenance basis. Any questions about appropriate prescribing practices and physician addiction would be handled by a committee specializing in addiction. As a result, almost no British physician has been arrested and/or tried for opiate-related violations.

The foundations of what is sometimes called the British system of drug policy had been established. From 1926 to 1960, this system worked well. Names of fewer than 1,000 addicts were forwarded to the Home Office each year, most of them medical personnel. Local practitioners could and did prescribe heroin and other opiates to their patients, including registered addicts. Some addicted patients were maintained on heroin, occasionally for years. They received their drugs from a local pharmacy. Addicts were also provided with clean needles and syringes. Drug treatment consisted almost entirely of individual physicians counseling addicted patients and providing drugs. Almost no illicit sales of opiates or cocaine occurred during these years. One staff member at the Home Office

was responsible for all registrations and personally knew most of the addicts in Britain; he frequently helped addicts find doctors and/or assistance. The Home Office also covened meetings with addiction specialists to address any policy issues that arose. Thus, the British established what might be described as a system of drug control that gave due weight to medical values and public-health considerations. Most observers now agree, however, that the "system" worked because the problem was limited in size rather than that the problem was small because of the system. It worked well for half a century until the numbers of addicts increased substantially, because of drug dealing on an international scale, the widespread use of drugs during the 1960s–1980s countercultural revolution, and the increased immigration to Britain of former colonial citizens of the crumbling empire. By the 1960s, the upsurge in heroin use and the abuse of cocaine, marijuana, and other drugs left Britain with a drug problem of both licit and illicit substances that outstripped even the British system's handling capabilities.

(SEE ALSO: *Britain, Drug Use in; British System of Drug-Addiction Treatment; Heroin: The British System; International Drug Supply Systems; Opioids and Opioid Control: History; Policy Alternatives: Prohibition of Drugs Pro and Con; Sweden, Drug Use in*)

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ROOFIES *See* Rohypnol; Slang and Jargon

RUBBING ALCOHOL Rubbing alcohol is known as isopropyl alcohol (C_3H_8O); it is one of the more useful of the commercial alcohols, included in hand lotions and many cosmetic items as well as in antifreeze or deicer products. A 70 percent solution has more germicidal properties than does ETHANOL (drinking alcohol), so it is used in many health-care situations, both in households and in medical facilities. It is also used for massages and by athletic trainers to treat skin and muscle groups, hence the term *rubbing*. It has a drying effect on the skin and causes blood vessels to dilate; its distinctive odor is associated with doctor's offices, since it is used to clean the skin being prepared for an injection.

When rubbing alcohol is ingested either pure or added to beverages, the result is toxic—with symptoms lasting longer than those seen after drinking ethanol (alcoholic beverages), because isopropyl alcohol is slowly metabolized to acetone, another toxic substance.

(SEE ALSO: *Inhalants*)

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S. E. LUKAS

RUSH *See* Slang and Jargon

RUTGERS CENTER OF ALCOHOL STUDIES For all the years of its existence, the Rutgers Center of Alcohol Studies (initially founded in 1940 as the Yale Center in New Haven, Connecticut) has been centrally involved in generating significant research findings on alcohol, alcoholics, and alcoholism. Through those same years, the center's mission has also included education, service, and information dissemination to the university community of which it was a part, the nation, and the world.

The Center of Alcohol Studies was founded at Yale University by Professor E. M. JELLINEK; it was developed from the well-known Yale Laboratory of Applied Physiology, directed by Professor Howard W. Haggard, which first began to study the physiology of alcohol (ethanol) in the 1930s. In recognition of the paucity of scientific journals publishing work on alcohol and alcoholism then, faculty at the center founded *The Quarterly Journal of Studies on Alcohol* in 1940. The journal's first issue was edited by Professor Haggard; shortly thereafter, Mark Keller, a longtime editor of the *Quarterly Journal*, became the journal's managing editor. Keller

served as editor of what is now the *Journal of Studies on Alcohol* for more than thirty years as a faculty member of the Center of Alcohol Studies at both Yale and Rutgers, and also became a very substantial figure in the alcohol field by virtue both of his position and his many carefully wrought, penetrating, insightful talks and articles on a wide range of alcohol-related subjects.

Recognizing the absence at the time of methods and agencies for the dissemination of the practical results of research on and experience with alcohol problems, the faculty of the center founded the Summer School of Alcohol Studies (SSAS) in 1943. It was then and continues today to be oriented toward meeting the needs of persons who work directly with the problems of alcohol use and alcoholism. The SSAS attracts students from around the world for its one- and three-week residential summer programs.

The pressing informational needs of the infant field of alcohol studies led to the development of the library of the Center of Alcohol Studies, which now possesses the most complete special collection on alcohol and alcoholism in the world, along with a complete collection of journals and books on alcohol and related subjects. The research library now maintains a collection of more than 100,000 materials. The Classified Abstract Archive of the Alcohol Literature contains about 20,000 abstracts of scientific work from a wide range of disciplines cross-indexed in depth up to 1976; the McCarthy Collection of original scientific papers; the Ralph G. Connor Collection of Alcohol-Related Research Instruments; and several extensive, continuously updated bibliographic series. The library's users include students, educators, and health service professionals.

Faculty at the Yale Center of Alcohol Studies initiated the first research program on treatment, as well as the Yale Plan on Alcoholism for Industry—a forerunner of modern EMPLOYEE ASSISTANCE PROGRAMS (EAPs). Center faculty also founded the first State Commission on Alcoholism. The research faculty at the center has continued to grow. By the mid-1990s, it comprised a substantial number of biochemists and physiologists, sociologists and psychologists, epidemiologists and preventionists—all engaged in studying an array of topics from etiology and physiology to prevention and treatment, with relevance to alcohol, alcoholics, and alcoholism.

In December 1994, Rutgers University approved a proposal by the Center to create the Rutgers Center of Alcohol Studies Faculty Practice Plan. This program provides assessment, intervention, and referral services to alcohol abusers who need help. The Center also offers the Drinkers Risk Reduction Program (DRRP), which was created for individuals concerned with their own drinking or the drinking of a loved one. DRRP employs both an assessment and intervention program, including a comprehensive interview, self-change program, self-control training, and a referral service.

The Center for Alcohol Studies' Basic Sciences Division conducts research on a number of projects, from alcohol and stress to the study of the effects of acute intoxication on people. The Clinical Research Division explores addiction assessment and research. The Education and Training Division conducts numerous one-day seminars throughout the academic year. Seminar topics not only include all aspects of alcohol and alcohol abuse, but also touch upon such subjects as gambling, HIV and AIDS, and tobacco.

In 1962, the Center of Alcohol Studies moved to Rutgers University, New Brunswick, New Jersey, into a building funded in part by a generous gift from R. Brinkley Smithers. From that time until he retired from Rutgers in 1975, Professor Seldon Bacon headed the Center of Alcohol Studies. A distinguished sociologist who had joined the center's faculty shortly after it was founded at Yale, Bacon played a key role for several decades in many of the most important developments in alcoholism nationally. At the Center of Alcohol Studies, he was instrumental in expanding the Yale Plan, developing the Summer School of Alcohol Studies, and nurturing the social-science research base that continues to be one of the center's major contributions.

In 1985, Smithers gave the center another extremely generous gift, permitting it to add to its building as well as to establish a prevention center and an annual prevention symposium.

(SEE ALSO: *Addiction Research Foundation of Ontario (Canada); U.S. Government Agencies*)

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S

SADD *See* Students Against Destructive Decisions

SAFE USE OF DRUGS *See* Prohibition of Alcohol

SAMHSA *See* U.S. Government Agencies

SAODAP *See* U.S. Government Agencies

SCHIZOPHRENIA Schizophrenia is a psychiatric illness that can be profoundly disabling and is usually chronic in nature. The cause is not known, but there appears to be a genetic predisposition. The etiology has been conceptualized in a stress/diathesis (vulnerability) model: Biological and environmental factors (e.g., drug abuse, psychosocial stresses) interact with a genetic vulnerability to precipitate the illness. Several theories have been proposed to explain the observed biological abnormalities of the disorder, including overactivity of the dopamine neurotransmitter systems in the central nervous system, changes in brain structure (e.g., enlargement of the lateral cerebral ventricles) and brain function (e.g., decreased frontal lobe function [hypofrontality], as evidenced by diminished blood flow, and deficits in attention and sensory filtering). Psychological and social factors are considered important in the expression and course of the disorder. It is likely that schizophrenia constitutes a group of disorders rather than a single entity; these disorders present with similar clinical signs and symptoms, but the etiologies, treatment responsiveness, and course of illness in each vary.

Detailed descriptions of the illness date back to the nineteenth century. Emil Kraepelin (1856–1926) used the term *dementia praecox* to describe psychiatric states with an early onset and deteriorating course. Eugen Bleuler (1857–1939) coined the term *schizophrenia* for a "splitting of the mind," in his belief that the illness was a result of the disharmony of psychological functions. The diagnosis of schizophrenia requires observation and clinical interviewing. No sign or symptom is specific for the illness, nor do any laboratory tests exist to establish the diagnosis. The DIAGNOSTIC AND STATISTICAL MANUAL *for Mental Disorders-3rd edition* contains the diagnostic guidelines of the American Psychiatric Association for schizophrenia. These include: the presence of characteristic psychotic symptoms (delusions, HALLUCINATIONS, a thought disorder, inappropriate emotion); impaired work, social functioning, and selfcare; and continuous signs of the illness for at least six months. The symptoms of an affected individual can change with time, therefore longitudinal follow-up is important. It should be noted that certain of these symptoms can be indicative of other conditions (including drug abuse [cocaine, crack, PCB, amphetamines], head injury, brain tumors, as well as other psychiatric disorders). Furthermore, it is

important to take into account the educational level, intellectual ability, and cultural affiliation of the individual when making a diagnosis. The onset of illness is usually in late adolescence or early adulthood and is generally insidious. The typical course of schizophrenia is characterized by exacerbations and remissions. A gradual deterioration in functioning generally occurs that eventually reaches a plateau. However, a small proportion of persons may recover. It is estimated that 20 percent to 30 percent of affected individuals can lead somewhat normal lives whereas another 20 to 30 percent continue to experience moderate symptoms.

The prevalence rates of schizophrenia vary to a limited degree worldwide, but in the United States the lifetime prevalence is estimated to be 1 percent (about one in one-hundred people). In industrialized countries, there is a disproportionate number of schizophrenic patients in the lower socioeconomic classes. Some experts feel this is due to the schizophrenic's loss of education and social opportunity, while others feel this is more a direct result of the stresses of poverty.

The management of affected individuals involves hospitalization when there is an exacerbation of the illness, plus the use of medication. The mainstay of pharmacologic treatment is the class of drugs known as ANTIPSYCHOTICS. Many antipsychotics are available and they act to control the psychotic symptoms; most of them do so by blocking the actions of the neurotransmitter, dopamine. About 75 percent of patients respond to these drugs; however, there are side effects, including muscle stiffness, tremors, and weight gain. The drugs may also cause tardive dyskinesia (TD), a disorder that causes involuntary, repetitive movements of the body, mouth, and tongue.

Some of the more commonly prescribed antipsychotics include: chlorpromazine, fluphenazine, haloperidol, olanzapine, and risperidone. The atypical antipsychotic, clozapine, has been identified as the best choice for managing resistant schizophrenia; however, up to 73 percent of patients treated with clozapine report clinically relevant side effects. These can be quite severe, and include potentially fatal neuroleptic malignant syndrome (NMS), myocarditis, cardiomyopathy, and dangerous lowering of white blood cell count (for the latter, regular and frequent blood testing is required during the treatment period). In a study following 8,000 patients in Australia who started clozapine treatment between January 1993 and March 1999, fifteen developed myocarditis, and eight developed cardiomyopathy; a total of six patients died within the six years.

After a person has recovered from an acute episode of schizophrenia, the emphasis is on practical aspects of management: living arrangements, self-care, employment, and social relationships. Education of and support made available to family members are important and can have an impact on relapse rates in the patient. Many schizophrenic patients have to remain on antipsychotic medication for prolonged periods, since the rate of relapse is high after drug discontinuation. Side effects, primarily of a neurologic nature (e.g., TD), are a source of concern, but in most cases the benefits of symptom control outweigh the risks of pharmacotherapy. Making sure that the patient complies with medication use is often a problem.

(SEE ALSO: *Amphetamine; Cannabis sativa; Complications: Mental Disorders*)

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SCHOOLS AND DRUGS *See* Education and Prevention

SCID *See* Structured Clinical Interview for DSM-IV

SCOPOLAMINE AND ATROPINE Scopolamine (*d*-hyoscine) and attopine (*dl*-hyosycamine) is a tropane alkaloid found in the leaves and seeds of several plant species of the family Solanaceae, including deadly nightshade (*Atropa bella-donna*) and henbane (*Hyoscyamus niger*). Atropine, a major alkaloid in deadly nightshade, is also found in JIMSONWEED (*Datura stramonium*). In Europe, in centuries past, henbane was a component of socalled witches' brews or was applied as an ointment to mucous membranes. According to some folktales, the idea that witches fly on broomsticks was derived from the sensation of a flying experience after the use of such ointments.

Scopolamine and atropine have very similar actions. They act as competitive antagonists at both peripheral and central muscarinic cholinergic receptors. Scopolamine is still sometimes used clinically for the treatment of motion sickness. The compound also causes central nervous system depression, leading to drowsiness, amnesia, and fatigue. It also has some euphoric effects and abuse liability, but these are not considered to be of such magnitude to require control of the drug under the Controlled Substances Act. Attopine has fewer actions on the central nervous system than scopolamine. It is used to reduce actions at peripheral cholinergic structures—it produces decreased gastric and intestinal secretions as well as spasms and also results in pupillary dilation. It blocks the action of the vagus nerve that results in slowing of the heart. It is often used before operations to prevent unwanted reflex slowing of the heart beat.

High doses of either of these tropane alkaloids can cause confusion and delirium accompanied by decreased sweating, dry mouth, and dilated pupils.

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SECOBARBITAL Secobarbital, prescribed and sold as Seconal, is a short-acting BARBITURATE used principally as a SEDATIVE-HYPNOTIC drug but occasionally as a preanesthetic agent. It is a nonspecific central nervous system (CNS) depressant and greatly impairs the mental and/or physical abilities necessary for the safe operation of automobiles and complex machinery.

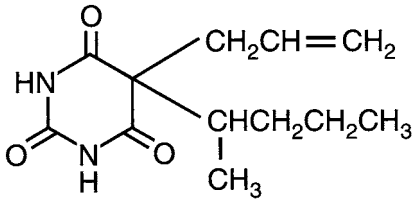

Figure 1
Secobarbital

Before the introduction of the BENZODIAZEPINES, it was the drug most commonly used to treat insomnia. Prolonged or inappropriate use of secobarbital can produce TOLERANCE AND PHYSICAL DEPENDENCE. If high doses have been used, abrupt cessation can result in severe WITHDRAWAL symptoms that include convulsions. Secobarbital is more likely to be abused than benzodiazepines and appears to produce greater euphoria in certain individuals than would a comparable sedative dose of a benzodiazepine. Consequently, it is classified as a Schedule II class drug in the CONTROLLED SUBSTANCES ACT, which indicates that although it is acceptable for clinical use, it is considered to have a high abuse potential. As with other barbiturates, it should never be combined with another CNS depressant because respiratory depression can occur.

(SEE ALSO: *Abuse Liability of Drugs: Testing in Humans; Drug Interaction and the Brain; Drug Interactions and Alcohol*)

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SCOTT E. LUKAS

SECONDHAND SMOKE *See* Tobacco: Medical Complications

SECONOAL *See* Secobarbital

SECULAR ORGANIZATIONS FOR SOBRIETY (SOS) Secular Organizations for Sobriety (SOS National Clearinghouse, P.O. Box 5, Buffalo, New York 14215) is a self-help organization for alcohol and drug users, founded as an alternative to ALCOHOLICS ANONYMOUS (AA) and other groups based on AA. It was intended to offer help to people who are uncomfortable with the emphasis on spirituality that is a central tenet of the AA Twelve-Step Programs. Founded by James Christopher, SOS began with a 1985 article. "Sobriety without Superstition," describing Christopher's own path to sobriety. SOS claimed in 1991 to have an international membership of 20,000, making it the largest of the alternative groups. In 1987, it was recognized by the State of California as an alternative to AA in sentencing offenders to mandatory participation in drug rehabilitation. Members of SOS are not necessarily nonreligious; however, many do not believe in an intervening higher power who takes responsibility for their individual problems.

Unlike AA—which emphasizes that the individual is powerless over alcoholism and must look to a "higher power" for help in achieving and maintaining sobriety—SOS and other alternative organizations assert the capacity of individuals to control their own behavior. SOS stresses total abstinence, personal responsibility, and self-reliance as the means to achieve and maintain sobriety (recovery), but the organization recognizes the importance of participating in a mutually supportive group as an adjunct to recovery. Members learn that open and honest communication aids in making the appropriate life choices that are essential to recovery. SOS shares with other self-help groups the importance of anonymity and the abstention from all drugs and alcohol.

SOS consists of a nonprofit network of autonomous nonprofessional local groups dedicated solely to helping individuals with alcohol and other drug addictions. It encourages and is supportive of continued scientific inquiry into the understanding of alcoholism and drug addiction.

Among other self-help organizations that see themselves as alternatives to AA are RATIONAL RECOVERY (RR) and Women for Sobriety (WFS).

(SEE ALSO: *Coerced Treatment for Substance Offenders; Disease Concept of Alcoholism and Drug Abuse; Treatment Types*)

JEROME H. JAFFE

SEDATIVE Sedative is a general term used to describe a number of drugs that decrease activity, moderate excitement, and have a calming effect. The primary use for these drugs is to reduce ANXIETY, but higher doses will usually cause sleep (a drug used primarily to cause sleep is called a *hypnotic*). Although the term *sedative* is still used, the drugs usually prescribed to produce this calming effect are BENZODIAZEPINES, which are more commonly known as antianxiety agents, or minor tranquilizers.

(SEE ALSO: *Barbiturates; Drug Types; Sedative-Hypnotic*)

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SEDATIVE-HYPNOTIC Sedative-hypnotic drugs are used to reduce motor activity and promote relaxation, drowsiness, and sleep. The term is hyphenated because, by adjusting the does, the same group of drugs can be used to produce mild sedation (calming, relaxation) or sleepiness. Thus, the distinction between a sedative and a

hypnotic (sleeping pill) is often a matter of dose—lower doses act as sedatives and higher doses promote sleep.

In some people, sedative-hypnotics can produce a paradoxical state of excitement and confusion. This tends to occur more frequently in the very young and older populations. Some of these drugs have the potential to be abused. Very high doses of most sedative-hypnotic drugs will produce general anesthesia and can depress respiration so much that breathing must be maintained artificially or death will occur. The benzodiazepines are an exception to this in that higher doses typically produce sleep and are far less likely to severely depress respiration.

One of the first agents to be added to the list of the classic sedatives (alcohol and opiates) was bromide, introduced in 1857 as a treatment of epilepsy. Chloral hydrate was introduced in 1869, and paraldehyde was first used in 1882. The barbiturates were introduced in the early 1900's and remained the dominant drugs for inducing sleep and sedation until the bezodiazepines were developed in the late 1950's and early 1060's. A number of miscellaneous non-barbiturate sedatives (ethchlorvynol, glutethimide, carbromal, methylparafynol, methprylon, methaqualone) were introduced in the 1940's and 1950's, and for a brief period rivaled the barbiturates in popularity, but their used declined rapidly along with the use of barbiturates. The bromides were recognized to have toxic properties, but they were still in use until the mid-twentieth century; chloral hydrate and paraldehyde were used well into the late 1970's and are still used in some places. Some drugs with other medical uses are prescribed as hypnotics, but the effectiveness of these substances remains to be proven in well-controlled clinical trials.

An advance in the development of sedative-hypnotics occurred with the discovery of non-benzodiazepine drugs that also act on the bensodiazepine receptor. Zolpidem and zaleplon are short acting hypnotics that demonstrate fewer side-effects and less tendency for rebound insomnia when they are discontinued, a common problem with the benzodiazepines. These drugs also demonstrate less abuse potential than many of the other sedative-hypnotics and little respiratory depression.

(SEE ALSO: *Abuse Liability of Drugs; Drug Interactions and Alcohol; Drug Types; Suicide and Substance Abuse*)

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SEDATIVES: ADVERSE CONSEQUENCES OF CHRONIC USE Sedative drugs are also called hypnotics or SEDATIVE-HYPNOTICS. They are sometimes referred to as "minor tranquilizers" or "anxiolytics" (antianxiety medications). Technically, a *sedative* decreases activity and calms, while a *hypnotic* produces drowsiness, allowing for the onset and maintenance of a state of Sleep similar to natural sleep and from which the sleeper may be easily awakened. The same drug used for sedation, pharmacologically induced sleep, and general systemic anesthesia may be seen to induce a continuum of central nervous system (CNS) depression. Such drugs are usually referred to, therefore, as sedative-hypnotics, and they are widely prescribed in the treatment of insomnia (sleep problems). Although some people take these drugs only occasionally and for specific sleep problems (grief, time-limited stress, long-distance flights), many more take them over prolonged periods (months and even years) as a presumed aid to nightly sleep. They do this despite medical advice to restrict such drugs to about two weeks of use.

All the sedatives are available in tablets or capsules for oral dosage, and some are also available for intravenous or intramuscular administration. Almost all sedatives have the same behavioral effects as alcohol (ethanol). Many persons who abuse sedatives, are, or have been problem drinkers. According to guidelines published by the American Psychiatric Association (1990), patients with a history of alcoholism or other drug abuse problems should not be treated with benzodiazepine seda-

tives on a chronic basis because they are at high risk of developing benzodiazepine abuse.

USE OF HYPNOTICS

Sleep problems in adults are of three main types (1) problems of falling asleep (sleep initiation), (2) problems staying asleep (sleep maintenance), and (3) early-morning wakening. Sleep-onset problems vary little with age; early-morning wakening is often secondary to depression; and sleep-maintenance problems show a clear and marked increase with aging. Whereas approximately 10 percent of young adults complain of serious sleep problems, this increases to 30 to 50 percent of those aged seventy or older (Morgan, 1990).

This age-related pattern for complaints of insomnia is reflected in the pattern of use of sedative-hypnotic drugs. For example, in the United States 2.6 percent and in Britain 4 percent of adults take a benzodiazepine as a sleep inducer during any given year (Mellinger, Balter, & Uhlenhuth, 1985; Dunbar et al., 1989). In the elderly, this increases to 16 percent use in a year, with 73 percent of those taking the drug regularly for a year or more. Indeed, 4 percent of people older than 65 had used the drug continuously for more than a decade (Morgan et al., 1988). Across all age groups, roughly twice as many women as men take sedative-hypnotic drugs.

The most commonly prescribed hypnotics include several benzodiazepines: flurazepam (Dalmane), quazepam (Doral), temazepam (Restoril), and triazolam (Halcion). Other hypnotics not related to the benzodiazepines are chloral hydrate (Noctec), a chloral derivative, and hydroxyzine (Vistaril), an antihistamine.

BENZODIAZEPINES

BENZODIAZEPINES remain by far the most frequently used sedative-hypnotic drugs (although there are some new compounds with differing modes of action). The key concerns in the hypnotic use of the benzodiazepines are (1) adverse effects experienced while the patient is taking the drug; (2) possible physical and psychological dependence; and (3) rebound insomnia and WITHDRAWAL symptoms when the patient stops taking the drug.

Classification. Benzodiazepines can be classified on pharmacokinetic grounds into long-acting (e.g., flurazepam, diazepam [Valium], chlordiazepoxide [Librium]); medium-acting (temazepam) and short-acting (triazolam, oxazepam [Serax], lorazepam [Ativan]) sedative-hypnotics. Their efficacy, at least in short-term use, has been well documented. The pattern of improvement in sleep corresponds fairly closely with the pharmacokinetic properties of each drug, providing that factors of absorption and elimination are taken into account. For example, temazepam is absorbed relatively slowly and has little effect on sleep-initiation time, whereas triazolam is absorbed relatively rapidly, which brings sleep on more quickly.

Each sedative-hypnotic has a minimally effective dose, but the dose that is usually effective may be twice as high as the minimum. Further increases may, however, cause side effects and rebound insomnia without substantially improving sleep. In sleep-laboratory studies, many benzodiazepines are found to lose their efficacy after about two weeks of nightly use. Subjectively, however, patients often feel that their sleep is improved for longer periods than this.

Adverse effects. Benzodiazepine sedatives have three major adverse effects: cumulative effects with repeated dosage, particularly if the patient has not yet metabolized the previous dose; additive effects when given with other classes of sedatives or with alcohol; and residual effects after the medication is discontinued. Patients taking benzodiazepines may feel drowsy, have reduced psychomotor speed, and impaired concentration. These in turn can adversely affect their ability to function; patients should be cautioned about driving and operating machinery while taking these drugs. The longer-acting the drug, the more pronounced are these effects. Tolerance to these sedative effects builds up to some extent over repeated use of the drug. Age-related changes in the way that drugs are metabolized and excreted mean that benzodiazepines accumulate more in older patients and, therefore, adverse effects are more pronounced in the elderly.

All benzodiazepines can impair the users ability to learn and remember new information. This memory impairment is most pronounced a few hours after taking the drug, so when taken as a sleep aid, such effects may be much reduced by the

time the person wakes the next morning. Again, the elderly are particularly prone to such effects. As with other adverse effects, higher doses cause greater problems. Rarer adverse effects include disinhibition and aggressive behavior. These effects have been reported for some benzodiazepines (e.g., triazolam, flunitrazepam) more than others.

Rebound insomnia refers to the heightened insomnia that may occur when the patient stops taking the drug, such that the sleep pattern is actually worse than it was before the medication. Studies have established that rebound insomnia is generally at its worst following the shorter-acting benzodiazepines and its least following the longer-acting benzodiazepines (Roehrs et al., 1986). Rebound is clearly dose-related, so the lowest effective dose should be prescribed, with rebound effects described to warn the patient about overdosing for "faster" or "better" drug-induced sleep.

Abuse, dependence and withdrawal. Some argue that rebound insomnia is itself a sign of physiological dependence on benzodiazepine hypnotics (e.g., Morgan, 1990). Others insist that dependence is shown only when withdrawal from a drug leads to symptoms other than a rebound of the original problems. In general, psychological dependence on benzodiazepines can develop rather rapidly. After only a few weeks, patients who attempt to discontinue the medication may experience restlessness, disturbing dreams, paranoid ideas and delusions, and feelings of tension or anxiety in the early morning. Withdrawal following moderate-dose usage may include dizziness, increased sensitivity to light and sound, and muscle cramps. Withdrawal following high-dose usage may result in seizures and delirium.

The syndrome of withdrawal from benzodiazepines may be slow in onset because these drugs remain in the body for relatively long periods. Withdrawal appears to be most severe in patients who used benzodiazepines that are absorbed rapidly and have a rapid decline in blood serum levels (alprazolam, lorazepam, and triazolam). In patients who abused both benzodiazepines and alcohol, a delayed benzodiazepine withdrawal syndrome may complicate withdrawal from alcohol. Patients who are high-dose abusers of benzodiazepines usually require inpatient detoxification.

Abuse. Animal studies indicate that benzodiazepines, like cocaine and opioids, activate a brain reward pathway in the brains of most mammals. In humans, the benzodiazepines have reinforcing effects that appear to be more pronounced in frequent users of other recreational drugs. For example, alcoholics and HEROIN addicts will at times use benzodiazepines to eke out their supply of first-preference drug, since ALCOHOL and heroin are also depressants.

Abuse of benzodiazepines by themselves is relatively unusual, but sometimes occurs among users who seek a "high" from massive amounts of these drugs. Street drug dealers sell benzodiazepines at a relatively low cost in most major cities. Some abusers combine benzodiazepines with other drugs to enhance the effects; for example, some believe that taking diazepam half an hour after an oral dose of methadone will produce a "high" that is more intense than can be obtained from taking either drug by itself.

Overdose. Overdosing on benzodiazepines is a medical emergency. It is marked by respiratory depression, low blood pressure, shock, coma, and eventual death. Flumanezil (Romazicon) is a benzodiazepine antagonist that can be given intravenously to reverse the sedative effects of an overdose.

OTHER SEDATIVE/HYPNOTIC DRUGS

Barbiturates. Barbiturates were used until the 1950s as sleeping pills but were superseded by the benzodiazepines. With the exception of phenobarbital (Luminal), which is still used as a sedative and as an anticonvulsant, the barbiturates are rarely prescribed.

Chloral Derivatives. These compounds, which include chloral hydrate, are sometimes used with elderly patients since they are less likely to cause restlessness in confused or demented patients. They are also relatively safe to give to children for sedation before or after surgery. Chloral derivatives can, however, cause gastric irritation and rashes.

Antihistamines. Diphenhydramine (Benadryl, Nytol, Sominex) and hydroxyzine (Atarax, Vistaril) are often prescribed for patients who need only a mild sedative. They are safe and do not produce dependency. They should not, however, be used together with alcohol. The most common side effect of these medications is dry mouth.

Newer Medications. Newer compounds include such nonbenzodiazepine hypnotics as zopiclone and zolpidem (Ambien), which act either

atypically or selectively on benzodiazepine receptors. They are chemically distinct from benzodiazepines and from each other. Both are short-acting drugs and at normal clinical doses cause little residual (hangover) sedation. The risk of rebound insomnia or dependence with these compounds is thought to be low but not absent (Lader, 1992).

Buspirone (BuSpar) is the only antianxiety medication that is not a sedative. Because it does not produce depressant effects or dependence, it is being used increasingly in the treatment of depression as well as anxiety. Unlike the sedatives, buspirone does not affect the patient's alertness or motor skills, it does not intensify the effects of alcohol, and it does not produce a withdrawal syndrome.

(SEE ALSO: *Accidents and Injuries from Drugs; Addiction: Concepts and Definitions; Aging, Drugs, and Alcohol; Barbiturates: Complications; Benzodiazepines: Complications; Drug Interaction and the Brain; Drug Interactions and Alcohol; Memory, Effects of Drugs on; Prescription Drug Abuse*)

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SEIZURES, BRAIN *See* Complications, Neurological

SEIZURES OF DRUGS The seizure of drugs is a salient consequence of a variety of U.S. enforcement programs, but particularly of interdiction. It provides evidence that the U.S. criminal-justice system is imposing costs on drug distribution. A large seizure offers the most vivid evidence that senior members of the drug trades are subject to serious risks.

Seizures from smugglers have often been used as a measure of the effectiveness of interdiction efforts. One argument suggests that the larger the quantity of drugs seized, the more smugglers have been hurt by interdiction. Others view seizures as an indicator of the quantity smuggled; this view assumes that the share of imports seized is effectively a constant. Clearly these are extreme assumptions. The quantity seized is a function of at least three factors: the quantity shipped, the relative skill of the interdictors, and the care taken by smugglers. The last element, given least attention in discussion of seizures, probably depends on the replacement cost of the drugs; if that cost goes down (e.g., because of good growing conditions in the producer country), smugglers will invest less in concealment and protection of shipments and thus the seizure rate (i.e., the share of shipments seized) is likely to rise.

Pennsylvania National Guardsmen, with the help of spotters in a helicopter, found more than 80 of these marijuana plants growing in the middle of a cornfield in York County, August 25, 1998. (AP Photo/Keith Srakocic)

Seizures of COCAINE rose throughout the 1980s, probably reflecting both the rapid increase in total shipments and the declining replacement cost of the drug. In 1989, federal authorities seized over 218,000 pounds of cocaine and that figure continued to rise during the 1990s. In 1999, cocaine seizures reached almost 291,000 pounds. MARIJUANA seizures grew dramatically during the same period. Federal authorities seized about 1.1 million pounds in 1989 and by 1999 the figure reached 2.3 million pounds. This is largely the result of increased U.S. cultivation and production of marijuana. Heroin seizures fluctuated between 1989 and 1999 but the overall trend was less dramatic than with other drugs. In 1989, federal authorities seized 2,415 pounds of heroin; in 1999, 2,788 pounds were seized. The total amount of drugs seized during this period, which also includes hashish, almost doubled. In 1989, the federal government seized a total of 1.343 million pounds of drugs. In 1999, the figure had risen to 2.62 million pounds.

Drugs are also seized by state and local police. Estimates are difficult to calculate at these levels of law enforcement, but it is believed that seizures at these levels have also grown during the 1990s. The growth of domestically grown marijuana has placed state and local police closer to the criminal activity. Likewise, the proliferation of domestic methamphetamine labs has made such facilities targets for both federal and state law enforcement.

(SEE ALSO: *Drug Interdiction; International Drug Supply Systems; Operation Intercept; Source Countries for Illicit Drugs*)

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SELF-HELP AND ANNONYMOUS GROUPS *See* Treatment Types

SENSATION AND PERCEPTION AND EFFECTS OF DRUGS Every behavior in which an organism engages involves information from the primary senses, such as vision, hearing (audition), and touch. A number of drugs of abuse alter sensory information. Mind-altering drugs can also influence perception of time, thinking, behavior, and mood. Often abusers of these drugs experience severe depression, anxiety, paranoia, confusion, and terror.

Naturally occurring drugs, such as MESCALINE from the PEYOTE cactus, increase awareness of visual and auditory sensations and also produce visual illusions and HALLUCINATIONS. The PSILOCYBIN mushroom (Mexican or Magic mush-

room) produces similar effects. Because of these sensory changes, mescaline and psilocybin have been used since pre-Columbian times in religious ceremonies by the peoples of Mexico and the American southwest.

LYSERGIC ACID DIETHYLAMIDE (LSD), an artificially-produced drug which was first synthesized in the late 1930s by the Swiss chemist Albert Hoffmann, has become well known for producing intense and colorful visual sensations. People also report changes in sensory behavior with drugs that are related to LSD (such as DMT, DOM, and MDMA, also known as "ecstasy" or the "love drug"). DMT is a short-acting (cycle takes less than one hour) crystalline powder that produces visual hallucinations. DOM, also known as STP, is more than 50 times as potent as mescaline. MDMA produces "out-of-body" sensations and acts as a stimulant. PHENCYCLIDINE (PCP) is another synthesized drug that is sometimes added to the list of drugs that alter sensory behavior; however, its sensory effects are limited to numbness in the hands and feet. Ketamine, also known as Special K, is a veterinary medicine that is chemically similar to PCP; its effects range from delirium to inability to move.

The active constituent of marijuana, TETRAHYDROCANNABINOL (THC), also produces alterations in sensory behavior; however hallucinations—such as those produced by mescaline or LSD—are less common with THC, although there is an increased risk of psychotic symptoms among users with a family or personal history of psychosis. COCAINE and AMPHETAMINE sometimes produce hallucinations and other sensory distortions, but only when they are taken for long periods of time.

Various names are used to describe drugs that alter sensory behavior. One term is *psychedelic*, which refers to mind-expansion or to experiencing events that go beyond normal boundaries; this word was coined in 1956 by Humphrey Osmond, a British psychiatrist. Another term is *psychotomimetic*, which refer to the similarities of hallucinations that occur in psychotic disorders, such as SCHIZOPHRENIA, and those produced by mescaline and LSD. The term *hallucinogenic* is slightly misleading, since not all drugs that alter sensory behavior produce hallucinations.

OBSERVATIONS IN HUMAN SUBJECTS

Most of our information about drugs and the ways in which they alter sensory behavior in people comes from individual reports (called anecdotal) rather than from well-controlled laboratory studies. People have reported vivid images, changes in perception, and hallucinations after they have taken mescaline or LSD. Synesthesias—a mixing of the senses, such as "the hearing of colors" or "the seeing of sounds"—may also occur. One of the first descriptions of LSD's effects is recounted as follows:

> I was seized by a peculiar sensation. . . . Objects, as well as the shape of my associates in the laboratory, appeared to undergo optical changes. . . . With my eyes closed, fantastic pictures of extraordinary plasticity and intensive color seemed to surge toward me. After two hours this state gradually wore off (Julien 180).

Although these sensory disturbances stop within a few hours, some people experience confusion, sensory distortions, or poor concentration for longer periods of time. For some people, drug effects recur long after the drugs have left their systems—these brief episodes are called *flashbacks.*

STUDIES IN THE LABORATORY

Since alterations in sensory behavior, such as hallucinations, cannot be observed directly, it is very difficult to examine these effects in laboratory animals. One way to investigate a drug's effect on sensory behavior is to train animals to behave differently in the presence of different types of visual or auditory stimuli. If a drug changes the animal's behavior, it is possible that these changes in behavior are due to a change in how well the animal hears or sees the stimuli. Another type of procedure examines how intense (e.g., how loud or how bright) a stimulus has to be for an organism to hear or see it. In these procedures, the intensity required to hear or see a stimulus is determined before a drug is given and then it is compared to the intensity required to hear or see the stimulus after the drug is given.

In general, drugs such as mescaline, LSD, and THC do not alter an animal's ability to tell the difference between visual or auditory stimuli—nor do they alter visual or auditory thresholds. This

lack of effect in animals suggests one of two explanations: either drugs such as LSD produce different effects in animals than they do in people, or, more likely, the procedures that are used to study alterations in sensory behavior in animals do not measure the unique ways in which drugs such as LSD alter sensory behavior.

Conversely, MDMA testing has found comparable results in both animals and humans. A late 1990s study (conducted on red squirrel monkeys) at Johns Hopkins University showed that MDMA has damaging effects on memory. Published in 2000, a British study of both current and previous MDMA users has discovered both immediate and delayed memory deficits.

(SEE ALSO: *Complications; Inhalants; Opiates/Opioids; Research; Research, Animal Model*)

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SENSATION SEEKING *See* Vulnerability as Cause of Substance Abuse

SENTENCES FOR DRUG OFFENSES *See* Mandatory Sentencing; Shock Incarceration and Boot-Camp Prisons

SEROTONIN Chemically named 5-hydroxytryptamine, this MONOAMINE transmitter is a widely distributed substance particularly prevalent in the gut, blood, platelets, and pineal gland, as well as in nine major sets of brain neurons (nerve cells). In the 1950s, chemical similarity between serotonin and the chemical HALLUCINOGEN LYSERGIC ACID DIETHYLAMIDE (LSD) focused attention on this NEUROTRANSMITTER in mental illness, a link strengthened by experimental studies in animals and humans. Neurons containing serotonin, a typical monoamine, project widely throughout the brain and spinal cord, and a large number of well-characterized serotonin-receptor subtypes mediate both direct and indirect regulation of ion channels that exist in the membranes of neurons. By regulating these channels, these serotonin RECEPTORS influence the concentration within the neuron of such ions as K^+ (potassium) and Ca^{++} (calcium) and thereby the activity of the cell.

(SEE ALSO: *Brain Structures and Drugs; Dopamine; Neurotransmission; Reward Pathways and Drugs; Serotonin-Uptake Inhibitors in Treatment of Substance Abuse*)

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FLOYD BLOOM

SEROTONIN-UPTAKE INHIBITORS IN TREATMENT OF SUBSTANCE ABUSE The development of effective pharmacological treatments for alcohol and drug abuse depends on our understanding of the biological mechanisms that start and maintain these behaviors. Studies in animals and humans have confirmed that SEROTONIN is one of several NEUROTRANSMITTERS that influence drug-reinforcing behaviors. Pharmacological agents that enhance central serotonergic

neurotransmission—in particular, serotonin-uptake inhibitors (several of which have been marketed as antidepressants)—show considerable promise, as of the early 1990s, as effective treatments for the abuse of alcohol and some other drugs. These work by blocking the re-uptake of serotonin and thereby increase its concentration in the nerve SYNAPSE.

ALCOHOL ABUSE

In the late 1980s, serotonin-uptake inhibitors were tested in various animal models of alcoholism—including selectively bred alcohol-preferring rats given a choice between water and an alcohol solution—and showed consistent decreases in the self-administration of alcohol in a dose-dependent manner. The results of these preclinical studies led to research in human alcohol abusers. In four placebo-controlled, double-blind, randomized clinical trials, serotonin-uptake inhibitors decreased short-term (1 to 4 weeks) alcohol intake by averages of 14 to 20 percent, as compared with pretreatment. No other treatment or advice was given. The effect developed rapidly after a serotonin-uptake inhibitor was administered and disappeared rapidly after discontinuation. All subjects had had mild or moderate (not severe) alcohol dependence but no current or past depression, anxiety, other psychiatric disorder, or other substance-abuse disorder. No aversive interactions with alcohol or changes in depression or anxiety levels were observed; therefore they could not account for the effects on alcohol intake. Adverse side effects were few and mild. However, concomitant decreases in desire/urge to drink were reported by subjects during treatment with serotonin-uptake inhibitors. Therefore, experimental drinking sessions, following one or two weeks of treatment with serotonin-uptake inhibitor and placebo, were incorporated into two research studies—fluoxetine (Prozac) and citalopram, each with a placebo control—to specifically measure variations in self-reported desire to drink alcohol. Desire for alcohol was lower during the experimental drinking sessions after taking serotonin-uptake inhibitors than after taking placebos. In both of these studies, the effects of serotonin-uptake inhibitors on alcohol intake were also confirmed in the outpatient weeks preceding the experimental drinking sessions.

The observation that serotonin-uptake inhibitors decrease desire to drink indicates a possible mechanism of their effects on alcohol intake. In the outpatient trials, an increase in abstinent days was often the means by which alcohol intake was reduced, and similarly, in trials with animals, serotonin-uptake inhibitors decreased their number of drinking "bouts." Therefore, serotonin uptake inhibitors may, by decreasing the desire to drink, reduce the likelihood of initiating drinking. The consistency of the pharmacological effects is quite remarkable, considering the many other factors influencing drinking behavior. In an effort to enhance the pharmacological effects of serotonin-uptake inhibitors and determine their therapeutic value, a brief psychosocial intervention was combined with citalopram in a long-term (12 week) treatment research study with sixty-two mildly/moderately dependent alcoholics. Average decreases in daily alcoholic drinks from baseline were 47.9 percent during the first week of citalopram ($n = 31$) and only 26.1 percent during the first week of placebo ($n = 31$), indicating a significant improvement with citalopram. From the second to twelfth weeks of treatment, the average decreases were similar: 33.4 percent and 40.5 percent during citalopram and placebo, respectively. Craving for alcohol also decreased similarly with both citalopram and placebo. Thus, the short-term effects of citalopram are synergistic with a brief psychosocial intervention, and serotonin-uptake inhibitors seem to facilitate the initiation of reduced drinking. The true therapeutic value of serotonin-uptake inhibitors is yet to be determined, but they may be appropriate for specific applications. For example, relapse is a frequent problem among recovering alcoholics; serotonin-uptake inhibitors, by decreasing desire or urge to drink, may be particularly suitable adjuncts for relapse-prevention strategies.

COCAINE

Abuse of COCAINE increased in the 1980s; it is also common among HEROIN addicts—some who use it alone and some together with heroin. Fluoxetine decreased cocaine craving and abuse in some heroin addicts who were in a METHADONE MAINTENANCE PROGRAM. These interesting results merit further study in a controlled trial.

CIGARETTE SMOKING

Cigarette smoking has not been affected by serotonin-uptake inhibitors in heavy drinkers who were not trying to reduce their smoking. Fluoxetine was found to prevent the weight gain that accompanies smoking cessation and, therefore, may be helpful in preventing relapse among exsmokers. The results of studies on the use of serotonin-uptake inhibitors in patients participating in smoking-cessation programs have not been reported yet.

PSYCHIATRIC DISORDERS

Individuals who abuse alcohol and/or drugs often have psychological or psychiatric disorders. The establishment of cause-and-effect relationships can be difficult. There is evidence that comorbidity (two disease processes) adversely influences outcome in treatments of substance abuse. Some patients may self-medicate symptoms of ANXIETY or DEPRESSION with a drug of abuse, such as alcohol. Therefore, successful pharmacological treatment of the anxiety or depression may reduce the need for other drugs (the alcohol).

As antidepressants, serotonin-uptake inhibitors would be particularly suitable for treating depressed substance abusers. No research studies have been conducted, but a comparison between treatment outcomes of depressed substance abusers receiving a serotonin-uptake inhibitor and those receiving other antidepressants would be of interest.

Severe cognitive deficits (memory loss) are a frequent complication of chronic ALCOHOLISM. Low brain levels of serotonin may be a factor in this type of memory loss. Fluvoxamine, a serotonin-uptake inhibitor, improved episodic memory in patients with alcohol amnestic disorder. This might greatly facilitate success in cognitively oriented treatments for alcoholism.

CONCLUSIONS

Serotonin-uptake inhibitors decrease short-term alcohol intake and desire to drink. Their effects are synergistic with a brief psychosocial intervention for alcoholism; however, their long-term efficacy and clinical importance have not been determined. One small study indicated that a serotonin-uptake inhibitor may reduce cocaine abuse. There is currently no evidence that serotonin uptake inhibitors reduce cigarette smoking or opiate abuse.

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SEXUAL AND PHYSICAL ABUSE *See* Vulnerability as Cause of Substance Abuse

SHANGHAI OPIUM CONFERENCE The 1909 Shanghai Opium Commission was the first multinational drug-control initiative. Through the encouragement of President Theodore Roosevelt and the organizational skills of Bishop Charles H. Brent, the United States convened this meeting of thirteen countries at Shanghai, including Great Britain, Japan, China, and Russia, to address the illegal production, trade, and use of OPIUM in China.

As a commission the participants could only recommend actions necessary to prevent opium trafficking and abuse but could not make binding international agreements. However, the participants passed resolutions urging national governments to enact measures to curb opium smoking in their countries, initiate regulation of opium use for nonmedical purposes, ban the export of opium to countries that prohibited importation, and control the manufacture and distribution of opium derivatives.

The commission was the first effective step taken by the international community to combat drug abuse. It served as a catalyst for countries to pass domestic legislation addressing drug problems within their borders. Most important, the commis-

sion united countries in an international cooperative effort to address the problem of the opium trade. The work of the commission led to the convening of the Hague Opium Conferences (1912–1914) and to the adoption of the 1912 International Opium Convention, sometimes called the Hague Opium Convention, and succeeding treaties that effectively restricted opium production and trade to legitimate purposes.

(SEE ALSO: *Asia, Drug Use in; International Drug Supply Systems; Opioids and Opioid Control: History; Psychotropic Substances Convention of 1971; Single Convention on Narcotic Drugs*)

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SHOCK INCARCERATION AND BOOT-CAMP PRISONS Shock incarceration programs, frequently called boot-camp prisons, are short-term prison programs run like military basic training for young offenders—adult and youthful felons (MacKenzie & Parent, 1992). Boot-camp prisons were first established in Georgia and Oklahoma in 1983 and since then all states and many counties have adopted this type of program. Boot-camp prisons have proved controversial over time, as critics argue that this type of regimen does not reduce recidivism (the tendency to return to crime). In the late 1990s, allegations of misconduct and abuse by boot-camp prison staff members against their juvenile inmates have led to criminal investigations and the closing of facilities. Nevertheless, this type of "tough love" approach remains a popular option for correctional officials.

Those sentenced to boot-camp prisons are required to arise early each day to participate in a rigorous schedule of physical training, military drill and ceremony, and hard labor. While they are in the boot camp, participants are separated from other prisoners. They are allowed few personal possessions, no televisions, and infrequent visits from relatives on the outside.

The correctional officers in the programs are referred to as drill instructors and are responsible for seeing that the inmates obey the rules and participate in all activities. When speaking to staff, inmates must refer to themselves as "this inmate" and they must proceed and follow each sentence with sir or madam as in "Sir, yes, sir." Disobedience is punished immediately using summary punishments, frequently in the form of some additional physical activity, such as pushups or situps. More serious rule violations may result in dismissal from the program.

BOOT-CAMP PRISONS AS INTERMEDIATE SANCTIONS

The boot-camp prisons were developed during the 1980s—in part, in response to the phenomenal growth in the number of convicted offenders. Correctional jurisdictions faced severe prison overcrowding, and probation caseloads grew so large that many offenders received only nominal supervision during their time in the community. Officials searched for ways to manage the offenders. There were two options—either they were sent to prison or they were supervised in the community on probation. Neither option was entirely satisfactory for the large number of young offenders. Alternative sanctions or intermediate punishments such as intensive community supervision, house arrest, or residential-community corrections centers were proposed as solutions to the problem. These options provided more control than a sentence to probation but less than a sentence to prison. Boot-camp prisons were one relatively inexpensive alternative sanction that became particularly popular.

The first boot-camp prisons were begun in 1983, in Oklahoma and Georgia. These two programs attracted a great deal of attention and other jurisdictions soon began developing similar programs. By 1999, more than fifty boot camps housed about 4,500 juveniles. Additional facilities house adult felons and other programs have been started in local jails and in juvenile-detention centers. Although the majority of the boot camps have male participants, some programs admit women into the boot camps with the male offenders. Other states have developed completely separate boot-camp

prisons for women. The Federal Bureau of Prisons developed one boot camp for males and a separate program for females. However, by 2000 several states had either ended their programs or drastically scaled back the size of the programs.

ENTERING AND EXITING

Since most boot camps have strict requirements about who is eligible for the camp, inmates are carefully evaluated prior to being sent there. Most programs require participants to sign an agreement saying they have volunteered. They are given information about the program and the difference between a boot-camp prison and a traditional prison. The major incentive for entering the boot camp is that the boot camp requires a shorter term than a traditional prison sentence.

The first day of the boot camp involves a difficult in-take process, when the drill instructors confront the inmates. Inmates are given rapid orders about the rules of the camp, when they can speak, how they are to address the drill instructors, and how to stand at attention. The men have their heads shaved; the women receive short haircuts. This early period of time in the boot camp is physically and mentally stressful for most inmates.

The programs last from 90 to 180 days. Those dismissed prior to graduation are considered program failures. They are either sent immediately to a traditional prison to serve a longer term of incarceration or they are returned to court for resentencing.

Offenders who successfully complete the boot camp are released from prison. After graduating, offenders are supervised in the community for the rest of their sentence. There is usually an elaborate graduation ceremony when inmates demonstrate the military drills they have practiced. Many programs encourage family members to attend the graduation ceremony.

A DAY IN BOOT CAMP

On a typical day, the participants arise before dawn, rapidly dress, clean their living quarters, and march in cadence to an exercise area. There they will spend an hour or more doing calisthenics and running. They march back to their quarters for a quick cleanup before breakfast. As they do at every meal, they march to breakfast and stand at parade rest while waiting to be served. They stand at attention until ordered to sit and eat without conversation. Following breakfast they may work six to eight hours. This is usually hard physical labor such as cleaning state parks or public roads. They return in the late afternoon for additional physical exercise or practice in drill and ceremony. After a quick dinner, they attend rehabilitation programs until 9 P.M. when they return to their dormitories. In the short period before bedtime, they have time to be sure their shoes are shined and their clothes are clean and ready for the morning.

SIMILARITIES AND DIFFERENCES

All the boot-camp prisons incorporate the core components of military basic training, with physical training and hard labor. Most target young offenders convicted of nonviolent crimes such as drug, burglary, or theft. Participation is limited to those who do not have an extensive past history of criminal activity.

Other than these similarities, the programs differ dramatically. Some focus only on work, military drill, and exercise. In other boot camps, offenders spend a great deal of time each day in rehabilitation programs. The camps also differ in the type of the therapeutic programming provided. Some emphasize academic education, others focus on group counseling or treatment for substance abuse.

The boot camps also differ in the ways offenders are managed after release. Some programs intensively supervise all offenders who successfully complete the boot camp; others are supervised as they would be in traditional probation caseloads. Program officials worry about the difficulty the graduates have in making the transition from the rigid structure of the boot camps to the community environment. For this reason, some boot camps developed aftercare programs to help them make the change. These aftercare programs do more than increase the surveillance over the activities of the graduates. They are designed to provide drug treatment, vocational counseling, academic education, or short-term housing to boot-camp graduates.

DRUG TREATMENT IN THE BOOT CAMPS

The earliest boot camps focused on discipline and hard work. More recently, they have begun to

Participants in the Sumter County Correctional Institution "boot camp" program arrive at their barracks in Bushnell, Florida, July 9, 1989. (© Bettmann/CORBIS)

emphasize treatment and education. It became clear that many of the entrants were drug-involved. Realizing that the punishment alone would not effectively reduce the drug use of these offenders, corrections officials introduced drug treatment or education into the daily schedule of boot-camp activities. By the late 1980s, all the camps had some type of substance-abuse treatment or education for boot-camp inmates (MacKenzie, 1994).

As happened with other aspects of the programs, the type of treatment and the amount of time devoted to substance-abuse treatment varied greatly among programs. The 90-day Florida program included only 15 days of treatment and education; in contrast, in the New York program all offenders received 180 days of treatment. Most programs reported that drug use was monitored during community supervision; however, the schedule and frequency of this monitoring varies greatly.

New York's Therapeutic Community Boot Camps. In the boot camps that include substance-abuse treatment as a component of the in-prison phase of the program, there are large differences in the way it is delivered. The boot-camp programs, developed by the New York Department of Correctional Services, use a therapeutic-community model for the program. All offenders are given a similar regimen of drug treatment while they are incarcerated (New York Department of Correctional Services, 1994). Each platoon in the boot camp forms a small community. They meet daily to solve problems and to discuss their progress in the shock program. They spend over 200 hours during the six-month program in substance-abuse treatment activity. The treatment is based on the ALCOHOLICS ANONYMOUS (AA) and NARCOTIC ANONYMOUS (NA) models of abstinence and recovery. All boot camp inmates participate in the substance-abuse treatment regardless of their history of use and abuse.

Illinois's Boot Camp with Levels of Treatment. Like New York, the Illinois boot camp also targets substance abusers. However, the delivery of treatment services is very different. In Illinois, counselors at the boot camp evaluate offenders and match the education and treatment level to the identified severity level of the offender (Illinois Department of Corrections, 1992). Three different levels of treatment are provided. Inmates identified as level-one have no substance-abuse history, therefore they receive only two weeks of education. Level-two inmates are identified as probable substance abusers. They receive four weeks of treatment in addition to the drug education. The treatment consists of group therapy focusing predominately on denial and on family-support issues. Inmates identified as level-three are considered to have serious drug addictions; they receive ten weeks of education and treatment. In addition to the drug education and group therapy, they receive group sessions on substance-abuse relapse, CODEPENDENCY, behavioral differences, family addiction, and roles within the family.

Texas's Voluntary Participation Model. A third model is represented by the Texas program (MacKenzie, 1994). In the boot camp, all participants receive five weeks of drug education. During this phase, inmates may also receive individual counseling and attend Twelve-Step fellowship meetings. More drug treatment is available for

those who volunteer (the substance-abuse counselors in this program believe that treatment should be voluntary). These volunteers receive approximately four hours per week of treatment in the form of group therapy. The meetings are held during free time, so inmates are not released from work to attend. The group sessions focus on social values, self-worth, communication skills, self-awareness, family systems, self-esteem, and goal setting. Some inmates also receive individual counseling.

DISMISSAL RATES

As occurs in many drug-treatment programs, boot camps may have high dismissal rates. Depending upon the program, rates vary from 8 percent (Georgia in 1989) to as much as 80 percent (Wisconsin in 1993). Offenders can be dismissed from the boot camp because of misbehavior or, in some boot camps, they can voluntarily ask to leave. Those who are dismissed will either be sent to a traditional prison, where they will serve a longer sentence than the one assigned to boot camp, or they will be returned to the court for resentencing. Thus, in both cases there is the threat of a longer term in prison for those who do not complete the boot camp.

There is very little information about how drug-involved offenders do in boot camp prisons. One study of the Louisiana boot camp examined the dismissal rates of drug-involved offenders and compared these rates to offenders in the boot camp who were not identified as drug-involved (Shaw & MacKenzie, 1992). Two groups of drug-involved offenders were examined: (1) those who had a legal history of drug-involvement (an arrest or conviction for a drug offense); and (2) those who were identified as drug abusers on the basis of self-report. In this program, offenders were permitted to drop out voluntarily or they could be dismissed for misbehavior. Surprisingly, in comparison to other offenders, the drug-involved offenders were *less* likely to drop out of the program.

In another study of the Louisiana boot camp, 20 percent of the participants were identified as problem drinkers on the basis of their self-reported alcohol use and problems associated with use (Shaw & MacKenzie, 1989). The problem drinkers were no more likely to drop out of the book-camp prison than were the others.

In interviews, offenders who are near graduation from boot camp report that they are drug free and physically healthy (MacKenzie and Souryal, 1994). Unlike offenders incarcerated in conventional prisons, boot-camp participants believed that their experience had been positive and that they had changed for the better. They also reported that the reason they entered the boot camp was because they believed they would spend less time in prison—not because of the treatment or therapy offered.

PERFORMANCE DURING COMMUNITY SUPERVISION

Studies have compared the performance during community supervision of graduates from the boot-camp prisons to others who served a longer time in prison or who were sentenced to probation. In most cases, there were no significant differences between these offenders in recidivism rates or in positive social activities (MacKenzie & Souryal, 1994). However, boot-camp graduates in Illinois and Louisiana had fewer revocations for new crimes. Research examining New York offenders found mixed results. Graduates had fewer new crime revocations in one study (New York Correctional Services, 1994) and fewer technical violations in another study (MacKenzie & Souryal, 1994).

All the boot-camp prisons had a military atmosphere with physical training, drill and ceremony, and hard labor. If this atmosphere alone changed offenders, we would expect all the graduates to have lower recidivism rates and better positive adjustment. The inconsistency of the results suggests that the boot-camp atmosphere alone will not successfully reduce recidivism or positively change offenders. Some other aspects of the Illinois, New York, and Louisiana programs, either with or without the boot-camp atmosphere, led to the positive impact on these offenders. After an examination of these programs, the researchers concluded that all three programs devoted a great deal of time to therapeutic activities during the boot-camp prison, a large number of entrants were dismissed, the length of time in the boot camp was longer than other boot camps, participation was voluntary, and the in-prison phase was followed by six months of intensive supervision in the community. Research as of the mid-1990s cannot separate the effect of these components from the impact of the military atmo-

sphere. Most likely, a critical component of the boot camps for drug-involved offenders is the therapy provided during the program and the transition and aftercare treatment provided during community supervision.

Performance of Drug-Involved Offenders. Shaw and MacKenzie (1992) studied the performance of drug-involved offenders during community supervision in Louisiana. In comparison to offenders who were not drug-involved, those who were drug-involved did poorer during community supervision. This was true of those on probation, parolees from traditional prisons, and parolees from the boot camp. The boot-camp parolees did not do better than others. During the first year of supervision, the drug-involved offenders were more likely to have a positive drug screen.

Problem drinkers who graduated from the Louisiana program were found to perform better, as measured by positive activities during community supervision (Shaw & MacKenzie, 1989). Their performance was, however, more varied—indicating that they may need more support and aftercare than other offenders.

In contrast to the Louisiana findings, research in New York indicated that those who were returned to prison were more apt to be alcoholics (New York Department of Correctional Services, 1994). In both Louisiana and New York, offenders who were convicted of drug offenses did better than self-confessed alcoholics during community supervision.

THE FUTURE OF BOOT-CAMP PRISONS

Boot-camp prisons are still controversial. By the late 1990s, skepticism rose about the effectiveness of this approach. Studies conducted for the U.S. Justice Department found that the national recidivism rate for boot camps ranged from 64 to 75 percent. This compared to recidivism rates from 63 to 71 percent for those who served their time in traditional detention centers. Though juveniles often responded well while in the camps, they returned to the same neighborhoods where they first got into trouble. Colorado, North Dakota and Arizona ended their programs and Georgia, where boot-camp prisons started, is phasing out its camps.

People are concerned that inmates' rights will not be observed and that they are being coerced to do something that is not good for them (Morash & Rucker, 1990). These critics argue that the summary punishments and the staff yelling at offenders may be abusive for inmates; that participants may leave the boot-camp prison angry and damaged by the experience; that the military atmosphere designed to make a cohesive fighting unit may not be appropriate for these young offenders. These concerns became public in the late 1990s, as state and federal prosecutors investigated allegations of abuse and misconduct by prison camp staff. Maryland fired its top five juvenile-justice officials in 1999 after state officials investigated reports of systematic assaults at three boot-camp prisons.

Advocates of the boot camp say that the program has many benefits. In their opinion, these offenders lack the discipline and accountability that are provided by the program. Furthermore, they argue, the strong relationship between the offenders and the drill instructors may be helpful to the inmates. Also, there may be some aspects of the boot camps that are particularly beneficial for drug-involved offenders. Although controversy exists about the boot-camp prisons, they remain a popular alternative sanction.

(SEE ALSO: *Civil Commitment; Coerced Treatment for Substance Offenders; Narcotic Addict Rehabilitation Act; Prisons and Jails; Treatment in the Federal Prison System; Treatment Types: An Overview*)

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SIDE EFFECTS *See* Complications

SINGLE CONVENTION ON NARCOTIC DRUGS The Single Convention on Narcotic Drugs of 1961 is the most comprehensive international drug control agreement ever signed. It regulates the production, trade, and use of NARCOTIC drugs, COCAINE, and cannabis (MARIJUANA).

BACKGROUND

Thirteen countries signed the first international drug control treaty in 1912 at The Hague, Netherlands. Into the 1950s, governments entered into eight multilateral treaties aimed at preventing the illicit trade and consumption of opium and other drugs. Over forty years, many of the provisions had become obsolete, had never been implemented, or required revision as world developments presented new challenges. The Single Convention consolidated the existing multilateral drug-control treaties into one agreement. Its drafters also intended to encourage governments that had not participated in earlier drug-control agreements to join the international effort. As of November 1993, 144 governments were party to the Single Convention.

PROVISIONS OF THE SINGLE CONVENTION

The Single Convention contains eight major provisions for the control of the production, trade, and use of drugs. All parties must establish or adjust national legislation to conform to these requirements of the convention.

Parties must require licenses for manufacturers, wholesalers, and other handlers of narcotic drugs, and they must maintain a system of permits, record keeping, reports, controls, and inspections to prevent diversion of drugs to the illicit traffic. A country that allows the domestic production of the OPIUM poppy, the COCA bush, or the *Cannabis* plant must establish a control agency to designate areas for the cultivation of these drugs and limit production to licensed growers.

Parties to the convention must prepare estimates (quotas) detailing the amount of drugs necessary to satisfy national medical and scientific needs, and they must provide these figures annually to the International Narcotics Control Board (INCB). Governments must also provide the INCB with quarterly and annual statistics on drug production, trade, and consumption. In addition, the Single Convention requires that parties maintain a system of import and export authorizations as well as import certificates so that the INCB and governments can monitor the flow of narcotics in and out of countries.

The Single Convention extends the control system over the opium poppy to the coca bush and the cannabis plant. Governments must uproot and destroy wild and illegally cultivated coca bushes and cannabis plants. Parties are furthermore required to ban opium smoking and eating, coca-leaf chewing, and cannabis smoking and ingestion.

A transition period is provided to overcome any difficulties that might arise for those who use such plants or drugs in ancient rituals. Countries may reserve the right to permit the quasi-medical use of opium and coca leaves as well as the nonmedical use of cannabis.

The Single Convention encourages parties to provide assistance and treatment to drug addicts. This provision distinguishes the agreement from previous international drug-control treaties, which focused exclusively on curbing the illicit flow of drugs.

INTERNATIONAL NARCOTICS CONTROL BOARD AND COMMISSION ON NARCOTIC DRUGS

Signatories to the Single Convention recognized the need for an international central monitoring and enforcement agency to oversee the production and trade of drugs. The Single Convention merged the Permanent Central Opium Board and the Drug Supervisory Board into the INCB, which serves as this central authority. The United Nations Economic and Social Council elects thirteen members to serve on the INCB.

The main responsibilities of the INCBs include limiting the cultivation, production, manufacture, and use of narcotic drugs and psychotropic substances to the amounts necessary for medical and scientific purposes, ensuring the availability of these drugs for medical purposes such as pain control. The INCB reviews estimates of opium and other drug-production figures provided by each party. These figures are formalized into production and consumption quotas. The board also analyzes information from participating countries, the United Nations, and other international organizations to ensure that there is compliance with the terms of the Single Convention. Where appropriate, it recommends that technical and financial assistance be given to those countries that may need further help. The Single Convention also provides the INCB with some direct enforcement powers, such as recommending an embargo of drug shipments to a country that is a center of drug trafficking. The INCB is more effective, however, in encouraging government to comply through confidential diplomatic initiatives than through the imposition of sanctions.

The Single Convention strengthens the role of the United Nations Commission on Narcotic Drugs (CND). The CND, which is composed of fifty governments, is the UN body that is the key information and policymaker in the drug-control area. The CND adds and deletes substances to or from the four control schedules of the convention, notifies the INCB of drug-control concerns, recommends ways to curb the illicit traffic of narcotics, and notifies nonparticipants of the actions that have been taken. It also gathers the names of the authorities that issue licenses for import and export.

DRUG SCHEDULES

In the preamble to the Single Convention, the parties recognized that "the medical use of narcotic drugs continues to be indispensable for the relief of pain and suffering and that an adequate provision must be made to ensure the availability of narcotic drugs for such purposes." In an effort to make narcotic drugs available for legitimate medical use while also curtailing drug abuse, the parties placed narcotic drugs into four schedules. Classification of a narcotic drug and the type of regulation that would be imposed on that drug substance would depend on a drug's potential for abuse as well as its medical benefit.

Schedule I is reserved for medically useful drugs exhibiting the highest potential for abuse. Examples of schedule I drugs include OPIUM, MORPHINE, and METHADONE.

Schedule II substances possess a liability for abuse that is no greater than that of CODEINE. These drugs are placed under similar controls as schedule I substances except that parties need not require prescriptions for domestic supply. Medical practitioners are not required to keep records tracking the acquisition and disposal by individuals of a controlled substance placed in schedule II. Codeine is the most commonly prescribed schedule II drug.

Drugs in schedule III are the ones intended for medical use that, as prepared, pose a negligible or nonexistent risk of abuse and a low public health risk. Schedule III drugs face substantially fewer controls than those listed in schedules I and II. Preparations of codeine and the analgesic dextropropoxyphene are two examples of drugs listed in schedule III.

To place a drug in schedules II and III governments must control the factories where these drugs

are manufactured as well as the individuals involved in their manufacture, trade, distribution, and import or export. Records of the manufacture and sale of these drugs must be maintained, and limits must be imposed to ensure that they are used exclusively for medical and scientific purposes.

The special class of drugs in schedule IV exhibit strong addiction-producing properties or a high liability of abuse that cannot be offset by medical benefits or that poses too great a risk to public health to hazard using them commonly in medical practice. Drugs in this category remain subject to the same international controls that are applicable to schedule I drugs, but governments are encouraged to limit their legitimate use. Cannabis, cannabis resin, and heroin (diamorphine) are examples of schedule IV drugs. Several medical experts have questioned the appropriateness of limiting the use of diamorphine for pain control and a number of governments permit this use.

Note that these schedules or levels of control differ from those contained in the Controlled Substances Act (CSA) of the United States. For example, in this act, drugs with a high liability for abuse and no accepted medical uses are included in Schedule I. The CSA also covers all categories of drugs including sedatives, HALLUCINOGENS, and cocaine besides other stimulants, whereas the Single Convention covers only opioid drugs, cocaine, and cannabis (marijuana). Other psychoactive drugs with abuse potential are controlled under a different international treaty, the Convention on Psychotropic Substances of 1971.

The World Health Organization (WHO) is responsible for making recommendations regarding the scheduling of drugs. In evaluating the schedule of a drug, WHO considers the "degree of liability to abuse" of a substance and the "risk to public health and social welfare" that the substance in question poses or might pose. The Convention grants WHO broad discretion in interpreting these two criteria. Ultimately, the Commission on Narcotic Drugs decides, by majority vote, whether to alter or amend a schedule, thereby reserving the right to reject WHO's recommendation.

THE 1972 PROTOCOL

The 1972 Protocol Amending the Single Convention on Narcotic Drugs confers greater powers on the International Narcotics Control Board and emphasizes the prevention of drug abuse, the distribution of drug information and education, and the treatment and rehabilitation of drug addicts. It also stresses the need to balance legitimate production of narcotics for medical and scientific purposes with prevention of illicit production, manufacture, traffic, and use of these substances.

THE SIGNIFICANCE OF THE SINGLE CONVENTION

The Single Convention has proved important in four ways. First, the aims, goals, and strategy in regard to combatting illicit drug trafficking became more focused and modernized because of its adoption. Second, the large number of participants in the Convention encourages more countries to take part in the international cooperative effort against drug abuse. Third, the placement of drugs into schedules constitutes a recognition of the differences between drug substances, and it balances the potential for abuse of the drugs with their medical benefit. The Single Convention, which openly supports the medical use of narcotics to relieve pain and suffering, states that these drugs are "indispensable" for the purpose. Narcotics with a higher potential for abuse and with a lower medical value fall subject to tighter regulation than drugs with a lower potential for abuse and a greater medical value. Fourth, the international community appreciates the need to combine strict controls of illicit drug trafficking with the treatment and rehabilitation of drug addicts. This approach, fusing strength with compassion, is now an integral part of the effort to curb the illicit production, trade, and consumption of narcotic drugs.

(SEE ALSO: *International Drug Supply Systems; Opioids and Opioid Control: History; Psychotropic Substances Convention of 1971; Shanghai Opium Conference; World Health Organization Expert Committee on Drug Dependence*)

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SKID ROW *See* Homelessness, Alcohol, and Other Drugs, History of

SKIN DAMAGE AND DRUGS *See* Complications: Dermatological

SLANG AND JARGON Slang terms in the drug subculture are constantly changing, as its ethnic, social, and demographic composition changes and as new illicit drugs roll in and roll out with the tides of fashion, including geographical variations. Yet certain terms show a remarkable durability such as some of those for heroin (trademarked Heroin in Germany, 1898)—a narcotic that has been a staple street anodyne since the early 1900s. Other drug-related terms have come into the mainstream to become a permanent part of the English language, e.g., *yen*, *hooked*, *pad*, *spaced out*, *high*, *and hip*. Many of the following words had been in use during much of the twentieth century (a few antiques of sociological or historical interest are included) and some are the product of the 1980s and 1990s. Origins, if known, are given.

a amphetamines, a stimulant

a-bomb, bomb LSD, a hallucinogen

acid [a shortening of *d*-lysergic acid diethylamide; since about 1960] LSD

Adam [originally named to connote a primordial man in a state of innocence] MDMA, a mild hallucinogen. See ecstasy below

amp [from *ampule*—the drug is sold in small glass ampules, which are broken open and the contents inhaled] amyl nitrite, a dilator of small blood vessels and used in medicine for angina pains; used illicitly to intensify orgasm or for the stimulation effect

amps amphetamines

angel dust [since the 1970s] phencyclidine (a brand name is Sernyl), an anesthetic used on animals but originally on humans; discontinued because of bizarre mental effects. See **PCP** below

bagging taking an inhalant by breathing it from a bag

base the pure alkaloid of cocaine that has been extracted from the salt (cocaine hydrochloride), in the form of a hard white crust or rock. See **crack** and **rock** below

batu crystalline methamphetamine

beamed up [from "Beam me up, Scotty," an expression used in the television series *Star Trek*; **Scotty** is also a term for **crack** cocaine; *on a mission* means looking for crack] intoxicated by crack

beamer a **crack** addict

beans dextroamphetamines

beast LSD

beat [from the idea of *beating*—cheating—someone] a bogus or mislabeled drug or a substance resembling a certain drug and sold as that drug (soap chips as **crack**; methamphetamine or baking soda as cocaine; catnip as marijuana; **PCP** as LSD, mescaline, or tetrahydrocannabinol (THC)—the active principle of marijuana; procaine as cocaine)

big H heroin

big C cocaine

blank nonpsychoactive powder sold as a drug

black beauties amphetamines

black tar heroin

blast a drag of **crack** smoke from a pipe

blotter [doses of the drug are dripped on a sheet of blotter paper for sale] LSD

blow (1) to sniff a drug (2) cocaine (3) to smoke marijuana ("blow a **stick**")

blue heavens methaqualone (a sedative) pills

bone a marijuana cigarette; a **joint**
boom marijuana
boomers hallucinogenic mushrooms containing psilocybin
booze alcohol
bottles vials or small containers for selling **crack**
boy heroin
breakfast cereal ketamine. See **K** below
brown heroin from Mexico diluted with brown milk sugar (lactose), which is less pure than **China white**. Also called **Mexican mud**
brown sugar heroin
buds [from the appearance] marijuana or sinsemilla (a hybrid variety of marijuana; see **sinse**); a quantity for sale consisting mainly of the more potent flowering tops of the marijuana plant (*Cannabis sativa*)
bump (1) cocaine. (2) **crack.** (3) fake **crack.** (4) hit of ketamine. See **K** below.
bush [from the *righteous bush*] marijuana
bust [from 1930s Harlem slang for a police raid, perhaps a shortening of *busting in*] arrest
button [from the shape of the appendages to the peyote cactus containing mescaline] peyote or San Pedro cactus
buzz, buzzed [from *buzz*, onomatopoeic equivalent of subjective feeling; the onset of the drug sometimes causes buzzing in the ears] (1) high on marijuana. (2) an inferior high from heroin
C cocaine
candy cocaine
caps hallucinogenic mushrooms
chalk [from the appearance] crystal methamphetamine or cocaine
Charlie cocaine
chasing the dragon [from a Chinese expression for inhaling fumes of heroin after heating it; the melting drug resembles a wriggling snake or dragon] (1) inhaling heroin fumes after the substance is heated on a piece of tinfoil. (2) smoking a mixture of crack and heroin
cheba marijuana
China white [from China (Indochina) **white** or **white stuff** = heroin; since the 1970s] (1) relatively pure heroin from Southeast Asia. (2) analogs of fentanyl (Sublimaze), an opioid more potent than heroin and sold on the street as **China white**
chipping, to chippy using heroin occasionally, avoiding addiction
chronic marijuana
cocoa puff [pun on the name of a chocolate-flavored breakfast cereal] a **joint**, to which cocaine has been added
coke cocaine
cola [a word play on *coke*, *cocaine*, and *Coca-Cola*, cocaine is derived from the coca (not the kola) plant] cocaine
cold turkey [from the gooseflesh that is part of abrupt withdrawal] by extension, ending a drug habit without medicinal or professional help, "going cold turkey"
coming down [from a **high**] losing the effects of a drug, all the way down to **crashing**
connect [from the *connection*, a drug pusher] cocaine importer or wholesaler, who fronts (consigns) cocaine to a supplier, who in turn distributes to a street retailer. See **dealing, mule, runner, steerer, touting**
cop [from British slang of the 1700s; to obtain, to steal, to buy; since the 1890s] to get or purchase illicit drugs
cop a buzz get **high**
copping zone an area where drugs are sold
crack [from the crackling sound when smoked in a pipe] pebbles of cocaine **base** that are smoked
crack house house or apartment (sometimes, an abandoned building) where **crack**-cocaine is sold and smoked on the premises 24/7—twenty-four hours a day, seven days a week
crank crystal methamphetamine
crank lite [from *crank*, because of the amphetamine-like stimulant effect + *lite*, meaning lighter, as in low-alcohol beer] ephedrine, a stimulant used in nonprescription medicines as a decongestant, which is lighter than amphetamines
crash, crashing to come all the way down from a drug **high**
cross roads [from the scored cross on the tablets] amphetamines
crystal [in powder form] methamphetamine or cocaine
crystal supergrass marijuana with **PCP**
cut to add adulterants to a drug—extending it to make more money in selling it (some adulterants are relatively harmless, some toxic)
date rape drug Rohypnol, called **roofies.** Women at parties may have this tasteless, odorless drug slipped into their drink. After

they lose consciousness, they may be raped and later have no memory of the incident.

deadeye blank stare produced by an overdose of phencyclidine (**PCP**) or other drug

dealing [from *dealer*, a person who sells drugs; since the 1920s] selling drugs of all kinds

designer drugs synthetic compounds or drug analogs that produce the effects of certain regulated drugs but have slight differences in chemical composition to evade the regulatory law; e.g., analogs of fentanyl (**China white**); analogs of amphetamine and methamphetamine such as MDA, MDMA (**ecstasy**), TMA, MMDA, MDE (**Eve**), MBDB; and toxic byproducts of the synthetic opiate meperidine (Demerol) such as MPTP and MPPP

dexies dextroamphetamines

ditch veins on the inside of the arm at the elbow, a site for injecting heroin. See **tracks** below

do drugs take or use illicit drugs

doobie a marijuana cigarette; a **joint**

dope [from Dutch *doop*, sauce (from *dopen*, to dip). In the late nineteenth century, the term came to be applied to opium, a black gum shaped into pellets and smoked in a pipe] (1) drugs (2) marijuana (3) heroin and other illicit drugs (4) intoxicating fumes of airplane fuel, glue (5) Coca-Cola

dope fiend [opprobrious term for narcotic and illicit drug users since the early 1900s; the term is used ironically by drug users to defy the social stigma] drug user, drug abuser, drug addict

dosing slipping a hallucinogenic drug into punch, brownies, etc., so that it will unwittingly be consumed by others

drag to draw or pull on smoke from a cigarette, pipe, or other item, "to take a drag"; to convey that smoke into one's throat and lungs. See **toke** below

drop to swallow LSD or a pill

dugie, doojee [phonetic] heroin

dust PCP

dusting (1) mixing either cocaine with tobacco in a cigarette or mixing heroin or opium with marijuana or hashish in a joint. (2) smoking **PCP**

ecstasy, extacy [from the euphoria, heightened sensuality, intensified sexual desire attributed to the drug experience] MDMA (methylenedioxymethamphetamine), a mildly hallucinogenic drug synthesized from methamphetamine and resembling mescaline and LSD in chemical structure

eightball an eighth of an ounce of cocaine

elephant tranquilizer PCP

Emilio [as in Emilio and Maria (Mary), from **Mary Jane**] marijuana

energize me give me some **crack**

equalizer pebbles of **crack**-cocaine

Eve [variant of **Adam**, MDMA or **ecstasy**] MDE, a mild hallucinogen derived from amphetamine. *Adam* **and** *Eve* is a compound of MDMA + MDE = MDEA (n-ethyl-MDA or 3,4,methylene + dioxy-N-ethylamphetamine)

exing taking **ecstasy**

fix (1) a needed drug dose to hold off withdrawal (2) a shot of heroin. See **shoot** below

flake [from the appearance] (1) cocaine hydrochloride (2) the sediment off a **rock** or chunk of cocaine

Flying Saucers [trade name] hallucinogenic seeds of a variety of morning glory

forget pill Rohypnol. See **roofies** below

freebase [the psychoactive alkaloid, the **base**, has been *freed* or extracted from the cocaine hydrochloride] (1) crystals of pure cocaine. (2) to prepare the **base**; to smoke it

frost freak one who inhales the fumes of Freon, a coolant gas, to get **high**

funky green luggage a supply of marijuana in one's baggage

G gamma-hydroxybutyrate. See **GHB** below

GHB gamma-hydroxybutyrate; clear liquid, white powder, tablet, or capsule often combined with alcohol; used mainly by adolescents and young adults, often at nightclubs and **raves**. GHB is usually abused either for its intoxicating/sedative/euphoriant effects or for its growth hormone-releasing effects, which can build muscles.

gangster marijuana

ganja [from *gaja*, Hindi word for India's potent marijuana, consisting of the flowering tops and leaves of the hemp plant, where most of the psychoactive resin is concentrated] marijuana

garbage can drug user who takes anything, everything, combinations

Georgia gamma-hydroxybutyrate. See **GHB** below

ghost LSD

girl cocaine
glass crystalline methamphetamine
gluey one who inhales glue fumes
goofing [from *goofballs* = barbiturates, and from *goof*, to act silly, stupidly, heedlessly] under the influence of barbiturates
grass marijuana chopped up line for smoking, which looks like dried grass
green [harvested hemp leaves that are not properly cured; also, the lower leaves of the hemp plant, which contain a smaller proportion of the psychoactive resin] (1) marijuana of low potency, e.g., *Chicago green*. (2) ketamine, an anesthetic similar to phencyclidine (**PCP**) but milder in its effects, which is sprinkled on parsley or marijuana and smoked
grievous bodily harm gamma-hydroxybutyrate. See **GHB** above
H heroin; also **Big H**
hash, hashish the concentrated resin of the marijuana plant, containing a high percentage of the active principle, tetrahydrocannabinol (THC).
hash oil liquid extracted from **hashish**, providing a more potent dose of the active principle and more easily transported in vials. It produces more sedation and deeper states of reverie than does hashish
Henry, Harry heroin
herb [used to connote a benign natural substance] marijuana
herbal ecstasy herbal combinations marketed as a "natural high" that can be legally purchased over the counter in drug stores, music stores, and other shops. The active ingredients include caffeine and ephedrine.
high [from the sense of euphoria, being above it all, detached from unpleasant reality] intoxicated by a drug
hip [from *laying (on) the hip*, to smoke opium—the addict lay on his side on a **pad** in an **opium den**—hence an opium user and then extended to illicit drug users. In the alienated subculture of the jazz scene of the 1930s and 1940s, using drugs was expected and made one keenly informed or *hip*—originally *hep*—until "squares" adopted the word] sophisticated, knowing, "in"; possessing taste, knowledge, awareness of the newest, and a lifestyle superior to that of conventional people
hit (1) an injection of a narcotic. (2) a **snort** of cocaine. (3) a **drag** from a crack pipe. (4) a **toke** of marijuana. (5) to adulterate (**cut**) a drug. (6) a dose of **LSD**
hog [from its original use as a veterinary anesthetic] phencyclidine (**PCP**)
home boy gamma-hydroxybutyrate. See **GHB** above
hooch alcohol
horse heroin
hot shot a potent dose of heroin sufficient to kill; heroin laced with cyanide
huff to inhale ordinary household products to get high. Users huff directly from the container or from inhalant-soaked rags, socks, or rolls of toilet paper. Inhalants include model airplane glue, nail polish remover, cleaning fluids, hair spray, gasoline, the propellant in aerosol whipped cream, spray paint, fabric protector, air conditioner fluid (freon), cooking spray and correction fluid.
ice extremely pure and addictive smokable form of crystalline methamphetamine
J, jay [from **joint**] a marijuana cigarette
jelly babies or **beans** amphetamine pills
joint [from *joint* as part of paraphernalia for injecting narcotics—particularly the needle; since the 1920s] a marijuana cigarette
jonesing [after John Jones, the British physician who first described opiate withdrawal in 1700] withdrawal from addiction; by extension, craving any drug
juice steroids
Julio marijuana. See **Emilio** and **Mary Jane**
junk [from *junker*, a pusher or peddler; since the 1920s. Also possibly from a word for *opium*—a play on *junk*, a Chinese boat—which was later extended to all narcotics] heroin (which is derived from opium)
K, super K, special K, Vitamin K ketamine, an anesthetic similar in structure to **PCP**. First synthesized by a pharmaceutical company in the early 1960s, powdered ketamine emerged as a recreational drug in the 1970s. It became **Vitamin K** in the underground club scene in the 1980s and **Special K** in the 1990s **rave** scene.
keester plant [from *keester*, rump, and *plant*, to place] drugs in a rubber container or condom concealed in the rectum
Ketaject, Ketalar ketamine. See **K** above

kick the gong (around) to smoke opium (especially in a Chinese **opium den**)

kick the habit [related to *kick it out*—to suffer withdrawal symptoms, which include muscle spasms in the legs and kicking movements from hyperactive reflexes in the spinal cord] (1) abrupt withdrawal from a drug to which one is addicted. (2) to conquer drug dependence

kif marijuana

killer joints marijuana with **PCP**

kind buds potent marijuana. See **buds** above

LA coke ketamine. See **K** above

la roche Rohypnol. See **roofies** below

lady cocaine

laughing gas nitrous oxide

lid [from the now obsolete practice of selling a measure of marijuana in a pipe tobacco tin] an ounce of marijuana, usually sold in a plastic bag

line (1) a thin stream of cocaine on a mirror or other smooth surface, which is sniffed through a *quill*—a rolled matchbook cover, tube, straw, or tightly rolled dollar bill, etc. (2) a measure of cocaine for sale

liquid ecstasy gamma-hydroxybutyrate. See **GHB** above

luding out [from *ludes*, short for Quaaludes (a brand name for methaqualone, an addictive sedative)] taking methaqualone.

Lyle [from *lysergic* acid] LSD

magic mushrooms hallucinogenic mushrooms

mainline [from *main line*, a major rail route; since the 1920s] (1) the large vein in the arm; the most accessible vein. (2) *v.* to inject morphine, heroin, or cocaine into any vein

Mary Jane, MJ, Aunt Mary marijuana

MDMA **ecstasy**

meth methamphetamine

microdot acid

Mexican brown marijuana from Mexico

Mexican mud brown heroin from Mexico. See **brown** above

mind altering the claimed mental effects of hallucinogenic drugs—altered or intensified states of perception

mind expansion [related to *psychedelic*, mindmanifesting; a descriptive term for hallucinogenic drugs coined in the 1960s] the claimed **mind-altering** effects of hallucinogenic drugs, including greater spirituality, enhanced self-awareness, and increased sensitivity to music, art, and nature; also synesthesia—cross-sensations, such as "seeing" music or "hearing" colors

Miss Emma morphine

monkey on one's back desperate desire for drugs; addiction; craving

moon [from the shape of slices of the bud of the peyote cactus] peyote

moonrock heroin mixed with **crack** for smoking

Moroccan candy [*majoun* (Arabic) is candy laced with **hashish**, sold in Morocco, Afghanistan] hashish. See **hash** above

mud heroin

mule (1) a low-level drug smuggler from Latin America; mules often swallow a condom filled with cocaine to be delivered at a destination—a dangerous practice called *bodypacking*. (2) heroin

new Ecstasy ketamine. See **K** above

night train PCP

nose candy cocaine

opium den [from *den*, an animal's lair. The term was coined by Westerners in nineteenth-century China, to have lurid connotations] a place where opium is smoked. Chinese laborers brought the practice of smoking opium to America during the gold rush of 1849 and the 1850s and the building of the transcontinental railroad

ozone **PCP**

pad [from the mats in **opium dens** on which the smokers reclined and slept. In the 1930s, Harlem apartments where marijuana was sold and smoked while reclining on couches or mattresses were called *tea pads*] (1) private place for taking drugs; a variant is **crash pad**, a place for recovering from the effects of a methamphetamine *run* (period of extended use); the user collapses (**crashes**) into an exhausted sleep. (2) by extension, since the 1950s, any dwelling place, room, apartment

PCP [from *PeaCe Pill*] phencyclidine (brand name Sernyl), a veterinary anesthetic that induces bizarre mental states in humans

peace pill **PCP**

pearls [medical nickname] amyl nitrite ampules

Persian white fentanyl. See **China white** above

p-funk, p-dope [*p* stands for pure] fentanyl. See **China white** above

PG paregoric, a traditional diarrhea remedy containing opium.

piece hashish, a form of marijuana. See **hash** above

pill popping [from *popping* something into one's mouth] promiscuous use of amphetamine and barbiturate pills or capsules. One who does this is a *popper* and may be a **garbage can**

pit veins on the inside of the arm at the elbow, a main site for injecting heroin and the place to look for **tracks**. See **ditch** above

pop to inject. See **shoot** below

poppers [the glass ampule is *popped* open and the contents inhaled] amyl nitrite ampules

pot [from *potaguaya*, a Mexican-Indian word for marijuana] marijuana

psychedelic heroin ketamine. See **K** above

pusher [extension from *pusher*—a person who circulates counterfeit money; since the 1920s] drug seller, drug dealer. See **dealing** above

quas, quacks [from Quaalude, a brand name of methaqualone] methaqualone pills, an addictive sedative

Raoul cocaine

rave an all-night underground party, usually frequented by teens and college students. Raves are characterized by techno music and often designer drugs, especially Ecstasy.

reds, red birds [also called red devils, red jackets, red caps—from the color of the capsules] Seconal (a brand of secobarbital) capsules

reefer [from *grifa*, a Mexican-Spanish word for marijuana] (1) a marijuana cigarette. (2) marijuana

rhoids steroids

rib Rohypnol. See **roofies** below

righteous bush marijuana plant

ringer [from the idea of "hearing bells"; *bells* is a term for **crack**] powerful effect from a **hit** of crack

roach [from its resemblance to a cockroach] the butt (end) of a marijuana cigarette

rock [from the appearance](1) large crystals or a chunk of pure cocaine hydrochloride. (2) **crack**. See **base** above

rocket fuel **PCP**

roofies, rophies, ruffies, roach, R2, roofenol Rohypnol, the brand name for the powerful sedative flunitrazepam. The pills are often used in combination with alcohol and other drugs.

rope Rohypnol. See **roofies** above

runner a messenger (often a juvenile) who delivers drugs from the seller to the buyer (not to be confused with a *drug runner*, a smuggler)

rush the quick initial onset of orgasmic sensations—of warmth, euphoria, and relaxation after injecting or inhaling heroin, cocaine, or methamphetamine

scag heroin

schoolboy (1) codeine, a derivative of opium with relatively low potency, used as a cough suppressant and analgesic. (2) morphine

Scotty crack-cocaine. See **beamed up** above

script prescription for a drug, often forged by addicts

script doctor a physician who will provide a drug prescription for a price—or one who is deceived into providing one

shabu crystalline methamphetamine

shake [the mixture is made by shaking the drug and the adulterant] (1) cocaine adulterated (**cut**) with a harmless substance such as mannitol. (2) loose marijuana left at the bottom of a bag that held a pressed block of marijuana.

sheet (acid) [from decorated blotter paper containing doses of the drug] LSD

shit heroin

shoot inject a drug; also *shoot up* a **fix** or a shot (usually of heroin)

shooting gallery place where heroin addicts **shoot up** and share needles and other **works** (paraphernalia)

shoot the breeze inhale nitrous oxide (called **laughing gas**).

shrooming high on hallucinogenic mushrooms

shrooms hallucinogenic mushrooms

Sid a play on the *s-d* sound of LSD

sinse [from *sinsemilla*, without seeds] a hybrid variety of marijuana; also called *ses*

skin popping [from **pop**, to inject] injecting heroin or any psychoactive drug subcutaneously (rather than into a vein), a practice of casual (**chippy**) users.

skunk marijuana

smack [perhaps from *shmek*, Yiddish word for sniff, whiff, pinch of snuff; since the 1910s, when heroin users sniffed the drug; in the

1920s and 1930s, some Jewish mobsters were involved in heroin trafficking] heroin

smoke marijuana

snappers [the ampule containing the drug is *snapped* open] amyl nitrite capsules

snob [from the idea of an elite—expensive—drug] cocaine.

snop marijuana

snort to sniff a drug

snow [from the appearance; also, the drug is a topical anesthetic and numbs the mucous membranes] cocaine hydrochloride.

snowbirds cocaine

soapers [from Sopor, the brand name of a sedative, now taken off the market] methaqualone pills

space basing or **space blasting** smoking a mixture of **crack** and phencyclidine (**PCP**)

speed (1) amphetamines (2) caffeine pills (3) diet pills

speedball [first used by GIs during the Korean War] injected mixture of heroin and cocaine.

splif a fat marijuana cigarette

spook heroin

squirrel a mixture of **PCP** and marijuana sprinkled with cocaine and smoked

stash extension of *stash*, hobo argot for hiding place; since the 1800s (1) hiding place for drugs. (2) a supply of drugs. (3) *v.* to hide drugs

steerer member of a cocaine or heroin crew who directs people to the seller

stepped on adulterated or **cut**

stick a marijuana cigarette

street drugs drugs purchased from sellers on the street; hence, of dubious quality

strung out severely addicted

sugar cubes **LSD**

sunshine [from the type sold as an orange-colored tablet] LSD

super grass [the powder is sometimes mixed with parsley or marijuana and smoked] ketamine. See **green**.

tabs [from *tablet*, a form in which the drug is sold] LSD

tea marijuana

Thai stick potent marijuana from Thailand

thing (1) heroin. (2) *pl.* an addict's **works**—the hypodermic needle (needle and syringe)

tic [from *THC*] fake tetrahydrocannabinol

toke a **drag** on a marijuana cigarette

tooies [from Tuinal, a brand name for a preparation containing amobarbital and secobarbital] sedative capsules

toot (1) to sniff cocaine. (2) cocaine. (3) a binge, especially a drinking bout or spree (since the late 1700s)

touting (1) purchasing drugs for someone else. (2) advertising, *hawking*, drugs that one is selling

tracks a line of scabs and scars from frequent intravenous injections. See **pit** and **ditch** above

tripping [from *trip*, in the sense of a psychic "journey"] taking LSD

trips (1) LSD tablets (2) periods under the influence of various drugs, usually hallucinogens

turkey [from *turkey*, a jerk; or from a theatrical failure or flop] (1) a nonpsychoactive substance sold as a drug. (2) the seller of such phony substances

turn on take drugs, especially hallucinogens

ups, uppers amphetamines

V, Vs Valium (a brand name for diazepam, a tranquilizer) tablets

wasted [from *waste*, a street-gang term since the 1950s, meaning to kill, beat up, destroy] (1) severely addicted to the point of mental and physical depletion (2) extremely intoxicated—out of it, beyond caring

weed marijuana

whack (1) to adulterate heroin, cocaine, or other drugs. (2) an adulterant (3) phencyclidine (**PCP**). (4) to kill

whiff [from the idea of smelling or shiffing] cocaine

white or **white stuff** heroin

white beanies amphetamines

white lady, white [from the color] cocaine

window pane [the drug is sometimes sold in a clear plastic square; also of a greater potency, providing a more intense experience and nonstructured sensations—"opening a window on reality"] LSD

wired (1) extremely intoxicated by cocaine. (2) anxious and jittery from stimulants (may be related to *amped*, a play on amphetamines and amperes)

woola [phonetic spelling] a **joint** containing a mixture of marijuana and **crack**

works equipment or paraphernalia for injecting drugs

X, the X, XTC [from **ecstasy**] MDMA.
yellow jackets [from the color of the capsules] Nembutal brand of pentobarbital
yen [from English slang *yen-yen*, the opium habit, based on Cantonese *in-yan* (*in*, opium + *yan*, craving); since the 1800s] any strong craving
zenes [short for Thorazine, a brand name for chlorpromazine] tranquilizer pills
zombie (1) **crack** cocaine. (2) phencyclidine (**PCP**)
zooted up high on **crack**-cocaine

(SEE ALSO: *Argot; Yippies*)

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SLEEP, DREAMING, AND DRUGS The use of "mind-altering" drugs and intoxicating drinks to hasten the onset of sleep and to enhance the experience of dreaming is a worldwide phenomenon and goes back to prehistory. The ancient Greeks used hallucinatory substances for religious purposes. The priestesses at Delphi, for example, chewed certain leaves while sitting in a smoke-filled chamber and going into a trance. On returning to consciousness, they would bring forth a divine prophecy. The various Dionysian cults encouraged their celebrants into ecstatic dream-like states through the use of wine and perhaps other drugs (Cohen, 1977).

The ancient Hindus imbibed a sacred drink called "soma," and MARIJUANA was used in practices of meditation. For the Arabs, HASHISH (a form of marijuana) was the substance of choice, while the Incas chewed the leaves of the COCA plant (from which COCAINE may be made). The OPIUM poppy was used in Asia, and the ancient Mexicans used a variety of powerful PSYCHOACTIVE substances, including PEYOTE, sacred mushrooms, and seeds from the Mexican MORNING GLORY plant, to enter the realm of dreams. The Australian aboriginals used the pituri, a psychoactive substance, to take them into "dream time," as they referred to it.

Belladonna and OPIATES have historically been used for the specific purpose of producing vivid dreams. The most famous illustration is the story of the English poet Samuel Taylor Coleridge (1772–1834), who allegedly wrote his most celebrated work, "Kubla Khan," during a drug-induced dream (Cohen, 1977). LYSERGIC ACID DIETHYLAMIDE (LSD) became popular in the United States and Europe during the 1960s for ostensibly facilitating higher states of consciousness and creativity. The writer John Lilley used a sensory-deprivation tank to emulate the state of sleep while taking LSD to induce creative dreaming (Cohen, 1977).

Reference to the effects of drugs and ALCOHOL on sleep and dreaming are also found in popular literature. It was a mixture made from poppies that caused Dorothy and her companions to fall into deep sleep in the *Wizard of Oz* (Baum, 1956). After ingesting a series of pills and liquids, in *Through the Looking Glass*, Alice finds herself in "Wonderland," where she has a conversation with an opium-smoking caterpillar who is sitting on a magic mushroom that alters the state of one who eats of it. After returning to the reality of her home in England, Alice realizes that she had, of course, fallen asleep and been dreaming (Carroll, 1951).

Modern study of the effects of drugs and alcohol on sleep and dreams dates to the mid-1950s. With the use of electrophysiological machines, including electroencephalograms (EEGs), electrooculograms, and electromyograms, the state of sleep most closely associated with dreaming was discovered, studied, and named REM, for the *r*apid *e*ye movements unique to that sleep state. In humans, REM sleep recurs in approximately 90-minute cycles throughout the sleep period, resulting in 4 or 5 REM episodes per night, each lasting from 10 to 30 minutes. Adults spend about 20 to 25 percent of their sleep period in REM sleep. Abrupt, but not gradual, awakening from REM sleep is consistently associated with the recall of vivid dreaming. While the function of REM sleep is unknown, it appears to serve a necessary function. Deprivation of REM sleep by awakenings or by the administration of REM-suppressing drugs leads to a compensatory or rebound effect-specifically, a more rapid onset and a greater amount and intensity of REM sleep.

Most psychoactive substances have profound effects on sleep and particularly on REM sleep. While the effects of drugs on REM sleep are known, their effects on dreaming are being studied. Given the association of REM sleep and dreaming, one might think that REM-enhancing drugs would increase dreaming, while REM-suppressing drugs would decrease dreaming. But no data suggest such a simple relationship. After the discontinuation of REM-suppressing drugs, a REM rebound occurs, which is reported to be associated with increased and unpleasant dreams. Some have hypothesized that the visual HALLUCINATIONS experienced during discontinuation of some drugs (e.g., alcohol) is a REM rebound intruding into wakefulness. It is too simplistic to think of dreaming and REM in a one-to-one correspondence, but it is reasonable to assume that drugs affecting REM will also affect the frequency and nature of dreams.

The effects of ethanol (alcohol) on sleep are complex and somewhat paradoxical. The acute bedtime administration of ethanol to healthy, nonalcoholic volunteers shortens the latency to sleep onset and, depending on dose, may initially increase the amount of relaxed, deep slow-wave (delta-wave) sleep (Williams & Salamy, 1972). Additionally, ethanol reduces the amount of REM sleep, usually affecting the flint or second REM period. An ethanol concentration in the blood of 50-milligram percent (mg%) or greater (100-mg% is legal intoxication in most states) is necessary for observing these sleep effects. The sleep effects of ethanol are observed only during the first half of an 8-hour sleep period. Ethanol is metabolized at a constant rate, and consequently the usual dose of ethanol (50-90 mg%) given in these studies is almost completely eliminated from the body after 4 or 5 hours.

Following elimination of ethanol, an apparent compensatory effect on sleep occurs. During the latter half of sleep, increased amounts of REM sleep and increased wakefulness or light sleep is found (Williams & Salamy, 1972). Within three to four nights of repeated administration of the same dose, the initial effects on sleep are lost (e.g., tolerance occurs), while the secondary disruption of sleep during the latter half of the night remains. REM sleep time and sleep latency return to their basal levels, and the effects on slow-wave sleep, when initially present, do not persist. When nightly administration of ethanol is discontinued, a REM rebound is seen. But the REM rebound after repeated nightly ethanol administration in healthy, nonalcoholic subjects is not a particularly consistent result (Vogel et al., 1990). In alcoholics, however, the REM rebound is intense and persistent (Williams & Salamy, 1972). Some believe the presence of a REM rebound is a characteristic of drugs with a high addictive potential.

MORPHINE, the opiate ANALGESIC (derived from the opium poppy), decreases the number and the duration of REM sleep episodes and delays the onset of the first REM period (Kay et al., 1969). It also increases awakenings and light sleep and suppresses slow-wave sheep. HEROIN, a semisynthetic opiate, also suppresses REM sleep and slow-wave sleep and increases wakefulness and light sleep, producing a disruption of the usual continuity of

sleep. Heroin appears to be more potent than morphine in its sleep effects. The synthetic opiate, METHADONE, has similar effects on sleep and wakefulness, with a potency more comparable to that of morphine. When an opiate is administered just before the onset of sleep, the EEG pattern shows isolated bursts of delta waves on the background of a waking pattern. Animal studies have correlated these delta bursts with the behavior of head nodding (a possible physiological correlate to the street term "being on the nod"). Repeated administration of the opiates at the same dose leads to tolerance of the sleep effects of these drugs, particularly the REM sleep effects (Kay et al., 1969). The cessation of opiate use leads to a protracted REM rebound, increased REM sleep, and a shortened latency to the first REM episode.

Among the stimulants, AMPHETAMINE, when administered before sleep, delays sleep onset, increases wakefulness during the sleep period, and specifically suppresses REM sleep (Rechtschaffen & Maron, 1964). Cessation of chronic amphetamine use is associated with an increase in slow-wave sleep on the first recovery night and, on subsequent nights, with increased amounts of REM sleep and a reduced latency to the first episode of REM sleep, a REM rebound.

Caffeine interferes with sleep in most nontolerant individuals (Greden, 1997). Once tolerance has developed, people are much less likely to report sleep disturbances, or they may sense that their inability to sleep because of caffeine intake has completely disappeared. To illustrate, 53 percent of those consuming less than 250mg per day (about 2 to 3 cups of coffee) agreed that caffeine before bedtime would prevent sleep, compared to 43 percent of those consuming 250 to 749 mg per day, and only 22 percent of those taking 750 mg per day or more. Even though the higher level caffeine consumers denied that caffeine interferes with their sleep, studies done in sleep laboratories confirm that caffeine consumers do have greater sleep latency, more frequent awakenings, and altered sleep architecture, and that these effects are dose-related (Greden, 1997). One study that investigated the effects of day-long consumption of coffee and tea on sleep onset and sleep quality demonstrated that caffeinated beverages had a dose dependent negative effect on sleep onset ($P<.001$), sleep time (P[.001) and sleep quality ($P<.001$) (Hindmarch, 2000).

Nicotine has a paradoxical effect on sleep. In a study using rats, the higher the dose of nicotine that was administered, the lower the total sleep time (Salin-Pascual, 1999). In a study that observed the effects of nicotine transdermal patches on depressed patients, nicotine increased REM sleep time and alleviated some symptoms of depression (Salin-Pascual, 1998). Yet, another study that assessed the effects of 24-hour transdermal nicotine replacement, at four different doses, on sleep showed no changes in sleep efficiency from baseline for any of the four doses used (Wolter, 1996). Sleep disturbances are possible when a person is attempting to withdraw from nicotine addiction, along with ability to concentrate. Research has demonstrated that such withdrawal symptoms are lessened by maintaining an adequate blood level of nicotine, as can be supplied by transdermal patches. In that regard, sleep can appear to be enhanced by the administration of 24-hour nicotine patches (Tsoh, 1996).

Cocaine also has stimulant effects on the central nervous system, and its effects on electroencephalogram readings were first studied by Berger in 1931; he was the researcher who developed the EEG (Berger, 1931). Cocaine was found to increase fast-frequency EEG activity, suggesting an alerting effect. The self-reported use of cocaine during the late afternoon and early evening is associated with reduced nocturnal sleep time. Systematic electrophysiological studies show a reduction of REM sleep (Watson et al., 1989). Cessation of chronic cocaine abuse is followed by increased sleep time and a REM rebound.

The three classic HALLUCINOGENS are LSD, MESCALINE, and PSILOCYBIN. The state experienced following use of hallucinogens is somewhat similar to dreaming. Since REM sleep is highly correlated with dreaming, scientists expected the hallucinogens to facilitate REM sleep, but LSD is the only hallucinogen that has been studied for its effects on sleep. One study done in humans showed that LSD enhanced REM sleep early in the night, although it did not alter the total amount of REM sleep for the night (Muzio et al., 1966). However, studies done in animals all indicate that LSD increases wakefulness and decreases REM sleep (Kay & Martin, 1978). The frequency changes seen in the waking EEG of animals (similar among all three hallucinogens) suggest an arousing effect. Thus the REM suppression in animals may not be a specific REM

effect but rather a sleep-suppressing effect (Fairchild et al., 1979).

Another drug with hallucinogenic effects is marijuana, its active ingredient being TETRAHYDROCANNABINOL (THC). The effects of THC on the waking EEG pattern are quite distinct from the effects of the classic hallucinogens cited above (Fairchild et al., 1979). THC has sedating effects at lower doses and hallucinatory effects at higher doses. The acute administration of marijuana or THC to humans is associated with an increase in slow-wave sleep and a reduction in REM sleep (Pivik et al., 1972). When THC is administered chronically (long-term), the effects on slow-wave and REM sleep diminish, indicating the presence of tolerance. Discontinuing the use of marijuana is associated with increased wakefulness and increased REM sleep time (Feinberg et al., 1976).

Most of these drugs, which are also drugs of abuse, seem to alter sleep and specifically the amount and timing of REM sleep. Each affects chemicals in the brain that control sleep and wake and, with chronic use, some adaptation seems to occur. A characteristic REM rebound is seen on discontinuation of dependent drug use. (It may be that the ancients' experience of enhanced dreaming was the REM rebound that is typically associated with protracted drug use.) Some studies indicate that, in the former drug dependent, the occurrence and intensity of the REM rebound has been predictive of relapse to drug use. How the sleep-wake pattern changes, and specifically the REM changes associated with these drugs, contribute to abused drugs' excessive use needs further study.

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(SEE ALSO: *Addiction: Concepts and Definitions; Benzodiazepines: Complications; Sedative-Hypnotics; Sedatives: Adverse Consequences of Chronic Use; Tolerance and Physical Dependence*)

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SLEEPING PILLS This is a general term applied to a number of different drugs in pill form that help induce sleep, i.e. sedative-hypnotic agents. There is a wide range of such medication and many require a doctor's prescription, but some can be purchased as OVER-THE-COUNTER drugs at a pharmacy. These latter preparations generally contain an antihistamine such as chlorpheniramine maleate, which produces drowsiness.

The prescription medications are much stronger. They include barbiturates, benzodiazepines, and a number of other compounds. However, due to the risk for fatal overdose, especially in combination with alcohol or other CNS depressants, the barbiturates are no longer widely prescribed for this indication. In general, the shorter-acting sleeping pills are used to help one relax enough to get to sleep, while the longer-acting ones are used to help prevent frequent awakenings during the night. Long-term or inappropriate use can cause TOLERANCE AND PHYSICAL DEPENDENCE.

(SEE ALSO: *Sedative-Hypnotic; Sedatives: Adverse Consequences of Chronic Use*)

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SCOTT E. LUKAS

SMOKING *See* Nicotine; Tobacco

SMOKING CESSATION *See* Tobacco; Treatment: Tobacco

SMOKING CESSATION AND WEIGHT GAIN *See* Tobacco: Smoking Cessation and Weight Gain

SNUFF *See* Tobacco: Smokeless

SOBRIETY The term *sobriety* is not defined in current medical or psychiatric literature. The term *abstinence* is found more often and is generally agreed upon as the treatment goal for severe alcoholics. Abstinence is defined as nonuse of the substance to which a person was addicted.

SOBRIETY AND SUBSTANCE ABUSE

The term "sobriety" is used by members of ALCOHOLICS ANONYMOUS (AA) and NARCOTICS ANONYMOUS (NA), and also by members of other Twelve-Step groups and recovery groups not affiliated with AA. In AA and NA, "sobriety" is often preceded by the adjectives "stable" or "serene." Abstinence—the condition of being sober—is a necessary but insufficient condition for sobriety. Sobriety means something different from the *initial* abstinence so often achieved by alcoholics and other drug addicts. This initial abstinence is recognized as a time of vulnerability to RELAPSE, often referred to as a "dry drunk" or "white knuckle sobriety."

Sobriety in NA and AA. According to AA beliefs, recovery from ALCOHOLISM and other addictions calls for more than just abstinence. The addict's central nervous system must undergo a substantial readaptation. This means that the CRAVING, drug-seeking, dysphoria (unhappiness), and negative cognitions that characterize early abstinence must not only diminish but must also be replaced by more normal positive behavior. This readaptation requires time and substitute activities. The activities most associated with successful readaptation are found in TREATMENT programs and in AA or NA.

Sobriety, as used by most recovering people in AA and NA, refers to abstinence plus a program of activity designed to make the abstinence comfortable and to improve functioning in relationships and in other aspects of life. The program of recovery that leads to stable sobriety usually includes: (1) attending AA and/or NA meetings; (2) "working" the Twelve Steps and continuing to use steps 10, 11, and 12 for the maintenance of sobriety; (3) working with a sponsor who acts as a mentor in maintaining sobriety; (4) belonging to a home group and engaging in service activities that help others with their sobriety; and (5) other activities that enhance or support sobriety (e.g., exercise, hobbies, and psychotherapy). A program of recovery recognizes that any activity has potential to either enhance or interfere with the recovering individual's sobriety. In addition, Twelve-Step programs emphasize the importance of basing sobriety on positive beliefs and ideals. "Shotgun sobriety" is defined in AA as a type of sobriety based only on fear of drinking. "Long-term sobriety must be based on spiritual principles, not on fear of alcohol."

Sobriety in Non-AA Recovery Groups. Secular Organization for Sobriety (SOS), Women for Sobriety (WFS), LifeRing Secular Recovery (LSR), and similar recovery groups for substance abusers also define sobriety in terms of abstinence from drugs and alcohol. A LifeRing pamphlet states, "Please look elsewhere for support if your intention is to keep drinking or using, but not so much, or to stop drinking but continue using, or stop using but continue drinking. The successful LifeRing participant practices the Sobriety Priority, meaning that nothing is allowed to interfere with staying abstinent from alcohol and drugs."

SOBRIETY AND BEHAVIORAL ADDICTIONS

One complication of the term "sobriety" has been the difficulty of defining it in the context of the so-called "process addictions" or "behavioral addictions," terms that have been used to distinguish addictions to such activities or behaviors as gambling, shopping, overeating, sexual acting-out, etc. from substance addictions in the strict sense. Unlike alcoholics and drug abusers, people with behavioral addictions cannot always define "sobriety" as simple abstinence. A compulsive overeater, for example, must learn to consume food in moderation, not avoid it. Persons addicted to compulsive spending or shopping cannot simply abstain from making purchases. Members of Sex Addicts Anonymous (SAA) rarely define sexual sobriety as "complete abstinence from sex," although at times recovering persons may practice complete abstinence (celibacy) for a period of time in order to gain perspective on their life. In this Twelve-Step group, sexual sobriety is most often defined as "a contract that the sexual addict makes between him/herself and their 12-step recovery support and/or their therapist/clergy. These contracts . . . are always written and involve clearly defined concrete behaviors from which the sexual addict has committed to abstain in order to define their sobriety." Comparable abstinence contracts are used by recovering binge eaters, compulsive spenders, relationship addicts, etc.

One benefit of attempts to redefine sobriety in the context of behavioral addictions is that they have called attention to the problem of substitute addictions, which are addictions that develop when a recovering alcoholic or drug abuser substitutes food, tobacco, or certain activities (including exercise) for their drug of choice. Many members of Twelve-Step groups have found that sobriety requires a re-examination of addictive beliefs and attitudes in general as well as abstinence from alcohol or specific drugs.

SPONTANEOUS RECOVERY

One question that has arisen in recent years is whether some alcoholics can achieve sobriety through spontaneous recovery. G. G. May (1988) uses the term "deliverance" for this phenomenon and defines it as "healing [that] takes the form of empowerment that enables people to modify addictive behavior." Some researchers suggest that spontaneous remission and recovery is more common among alcoholics than was once believed, and that it is connected to growth and maturity in the course of the adult life cycle. G. E. Vaillant (1983) found that most alcoholics in his study outgrew their drinking problem, more often than not without going into treatment or joining AA. Stanton Peele (1992) is perhaps the best-known proponent of the view that ". . . some people who appear completely out of control of their actions at one point significantly change their outlooks and ability

to regulate their behavior later in life." He likens spontaneous recovery of sobriety to the ability of some smokers to suddenly quit using tobacco.

SUMMARY

Despite these problems of precise definition, the concept of sobriety (abstinence or its equivalent for nonchemical addictions, plus a program of activity designed to make abstinence comfortable) is a useful one for health-care professionals.

(SEE ALSO: *Addiction: Concepts and Definitions; Treatment Types: Minnesota Model; Treatment Types: Self-Help and Anonymous Groups*)

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SOCIAL COSTS OF ALCOHOL AND DRUG ABUSE Drinking, smoking, and the use of psychotropic drugs have a variety of consequences for those who partake of them, for their families and associates, and for society at large. A number of these consequences are negative. Smokers die young from heart or lung disease, drinkers get into traffic accidents and fights, drug injectors spread the HIV virus. In the context of public policymaking, where priorities must be set for the use of scarce resources, it seems important to have a measure of the overall magnitude of the social burden engendered by such consequences. One familiar approach is to express the magnitude of the problem in terms of the number of people who die each year. When we learn that there are 107,400 deaths per year in the United States from ALCOHOL abuse (Harwood et al., 1998) and perhaps four times that number from TOBACCO use, we know that the stakes are very high in devising sound policies for controlling drinking and smoking. Such statistics, compelling as they are, tell only part of the story. In addition to causing early death, substance abuse makes for a variety of consequences that reduce the quality of life, both for users and other people.

To capture this broad array of consequences in a single number, analysts have estimated various measures of social cost. The estimates are important because they figure in the political process by which federal funds are allocated to the National Institutes of Health and to other agencies that play a role in combating substance abuse. The most prominent estimates of social costs for substance abuse have utilized a conceptual apparatus developed by a task force of the U.S. Public Health Service chaired by Dorothy Rice (Hodgson & Meiners, 1979). In 1994, the International Symposium on the Economic and Social Costs of Substance Abuse issued guidelines recommending the use of this cost-of-illness method in an attempt to establish a common foundation and enhance the comparability of cost studies conducted in different countries (ICAP, 1999).

Although prominent in policy debate, the cost-of-illness (COI) method has been faulted for its emphasis on production as the measure of social welfare. Economists favor a quite different approach that measures social welfare from the perspective of the consumer. The economists' pre-

ferred accounting framework is referred to in this article as the "external social-cost" approach.

THE TWO FRAMEWORKS APPLIED TO SUBSTANCE ABUSE

A coherent assessment of the social costs of substance abuse requires an accounting framework that specifies criteria for judging which of the myriad effects are properly deemed to be of public concern. For example, in the case of drinking, on any one drinking occasion there may be unwanted, harmful consequences: social embarrassment, loss of reputation or affection, failure to discharge some responsibility at work or home, physical injury from an accident, victimization by a mugger or rapist, and nausea or hangover. Chronic heavy drinking may result in still other consequences, including rejection by family and friends, loss of a job or of an opportunity for promotion, progressive deterioration in physical health, and an early death. In order to capture these and other negative consequences in a single number, the list of consequences must be reviewed to determine which should be considered in establishing priorities for substance abuse policy. The consequences deemed relevant must then be quantified, translated into a standard unit of account (dollars), and summed.

The Cost-of-Illness Framework. The COI approach is concerned with measuring the loss or diversion of productive resources resulting from an illness or activity. In the case of alcohol abuse, human capital resources are lost and the gross national product reduced by the morbidity and early death suffered by some drinkers, whether as a result of injuries sustained in alcohol-related traffic accidents or violent crime or as a result of organ damage and other diseases stemming from chronic heavy drinking. The loss to society in these cases is equal to the loss of the marginal product of the victims' labor, valued at the market wage. Unpaid work at home, including housework and child care, is included in the computation, with values being assigned according to how much households pay for such services when they are performed by paid help.

The COI approach also takes account of the diversion of resources from other productive uses necessitated by alcohol abuse. Thus the costs of medical care for alcohol-related illness, treatment for ALCOHOLISM, and research on prevention and treatment are incorporated in the social-cost estimate. Similarly, the value of law-enforcement and justice resources devoted to alcohol-related crimes are included, as are the costs of replacing property damaged in traffic crashes and fires caused by drinking.

Several prominent estimates of the total costs of alcohol abuse for the United States have utilized the COI framework (Berry & Boland, 1977; Harwood et al., 1984 & 1998). In 1998, Harwood et al. published the most complete COI study to date. Using figures from 1992, the most recent year for which complete data were available, they found that the economic costs to society of alcohol abuse totaled $148 billion, broken down as follows:

About three quarters ($107 billion) of the total cost in this tabulation is the value of labor PRODUCTIVITY lost as the result of illness, injury, or early death. The human capital lost as a result of alcohol-related mortality was computed for all those who died in 1992 from causes in which intoxication or chronic heavy drinking played a role. These include traffic fatalities and deaths from liver cirrhosis, among other causes. The lost human capital was valued by estimating how much the deceased would have earned if they had lived and worked until retirement age.

The human capital lost as a result of morbidity was calculated by estimating the reduction in the productivity of the labor force resulting from alcohol dependence or abuse. Harwood et al. combined two sets of estimates to arrive at this number: first, the percentage of the labor force in 1992 that was or had ever been subject to a diagnosis of alcohol dependence or abuse; and second, an estimate of the loss in earnings associated with such a diagnosis.

Critique. Estimates of this sort have been challenged for two reasons. The first challenge is to the statistical methods used to generate the estimates of morbidity, mortality, and lost earnings (Cook, 1991). The second challenge is more fundamental, for it concerns the basic principles that inform the COI accounting framework.

The COI procedure estimates the cost of morbidity and mortality in terms of lost productivity, but this emphasis on production as the measure of social welfare seems misplaced. A more liberal perspective, favored by economists among others, shifts the emphasis to consumption and interprets the task of measuring social welfare in terms of

aggregating individual preferences. Consumers are the best judges of their own welfare, and if sometimes they make choices that fail to maximize their productivity, that should not in itself be regarded as problematic. In this view, the choices that people make concerning how hard to work and when to retire are of little public concern. The same goes for choices that place one's own health and safety at risk. Thus in economics there is a strong presumption in favor of consumer sovereignty, the principle that the individual consumer is in the best position to define what is best for him or her, and that social welfare is enhanced by free choice within certain limits. A negative consequence is deemed to be of *public* concern only when the actions of one individual impinge negatively on the welfare of others. The basic distinction, then, is between *internal* and *external* consequences of individual decisions, where the latter impose an involuntary cost on other people.

In the case of alcohol abuse, the internal costs include those suffered by drinkers and are foreseeable as a natural consequence of their choices. A small example explains the reasoning here. Suppose a woman decides to drink heavily tonight despite knowing that she may be tired and unproductive tomorrow. By making this decision, she is indicating that for her the pleasure of partying outweighs the "morning-after" costs. If no one else is harmed by this decision, the external costs are zero. If she were to drive after drinking, however, the accounting would change. She would be risking serious injury to herself and to others on the highway. Her injury would have external costs to the extent that a third party (group insurance or Medicaid) paid her medical expenses. The risk that she might injure other people while driving is also a negative externality, to be valued at the expected loss to them. That cost, incidentally, is not limited to their lost earnings, but also includes their pain and suffering and the suffering of those who care about them.

In sum, the most fundamental challenge to the COI framework relates to its presumption that social welfare is synonymous with national product. Economists argue instead that the preferences of individuals are the proper measure of their well-being and that social welfare is the sum total of individual welfare. Some of the major costs in the COI framework, especially lost earnings, are less important in the external social-cost view, whereas a number of costs that are ignored in COI become important when the focus is on external costs.

The External Social-Cost Framework. In a study at the Rand Corporation, economists applied the ESC framework to alcohol abuse and other poor health habits (Manning et al., 1989, 1991). Their estimate for alcohol abuse amounted to about $30 billion in 1985, less than half the COI estimate presented above for the same year. The accounting procedures used to generate this estimate of the ESC can be briefly summarized:

1. **Earnings**. Heavy drinkers might earn less than they otherwise would have during their careers and might have their careers cut short by poor health and early death. Although the most obvious effect was a reduced standard of living, which was properly considered a private cost, a number of programs created a collective interest in the productivity of each individual. For example, those who died young saved their fellow citizens the expense of years of pension payments and medical costs. Those who retired early (perhaps because of poor health) imposed financial costs on others in the sense that their contributions to the Social Security system were reduced. Thus these collective financing arrangements had the effect of creating both external costs and benefits in relation to heavy drinking. The net effect, according to Manning et al. (1991), was negative, and equaled about 22 percent of the total external cost.
2. **Traffic Fatalities**. Heien (1996) reported that about 3,765 of the 13,984 people who died in alcohol-related traffic accidents in 1993 were "innocent," in the sense that they had not been drinking at the time. Their lives had value not because their work increased the size of the national product, but because they enjoyed life. People are willing to pay to reduce the risk of a fatal accident, and the social cost of these innocent deaths is in principle equal to the total amount the public would be willing to pay to eliminate the threat of being killed by a drunk driver. Manning et al. (1991) employed this willingness-to-pay approach and found that nearly half of the social cost of alcohol abuse stemmed from traffic fatalities.
3. **Other Costs**. The remaining $7.2 billion in Manning et al.'s (1991) social cost estimate stemmed primarily from the burden of alcohol-

related cases on the criminal justice system, and the share of collision insurance costs accounted for by the property damage caused by drunk drivers.

It appears that in several respects these estimates are incomplete. The costs of alcohol-related injuries to innocent victims are far higher than indicated by Manning et al., since they omitted the financial and personal costs of nonfatal injuries in traffic accidents (Miller & Blincoe, 1993), and also the costs of both fatal and nonfatal injuries from violent crimes perpetrated by drunks. In addition, recent research has suggested that moderate alcohol consumption carries measurable health benefits, which must also be figured into any equation attempting to assess social costs (ICAP, 1999).

An even more interesting controversy has arisen over the basic perspective that informs these external social-cost estimates. Some critics reject outright the liberal doctrine that individual preferences are to be accorded primacy in the definition of social welfare and social cost. They postulate a collective interest that can somehow be defined without reference to the choices made by individuals (Beauchamp, 1980). The COI approach reflects one such definition. Other critics accept the liberal doctrine but argue about its application. A particularly difficult set of philosophical and practical issues arise in setting the boundary between internal and external costs in the context of the family. Manning et al. (1991) view the family as a unit and accept the presumption that each member of the family will internalize the concerns of the others and act accordingly. Harwood et al. found that, in 1992, abusers and their households bore $66.8 billion of the total cost of alcohol abuse. If the father is a heavy drinker or smoker, it is not because he is unaware or unconcerned about the consequences for his wife and children of his drinking or smoking, but because his enjoyment of these activities in some sense outweighs the costs to them. That presumption may seem particularly problematic in the case where the mother's substance abuse causes her baby to be born defective.

COSTS OF SMOKING AND DRUG ABUSE

Manning et al. (1989) provided an estimate of the social costs of smoking that utilized the same general approach as their estimate of drinking costs. They found that over their lifetime smokers experienced higher medical costs than they would have if they had never smoked, amounting to an average of $0.38 per pack. Since these costs were for the most part paid by insurance, government programs, or other collective sources, they included them in the external social-cost estimate. Other important external costs were the reduced contributions to the Social Security system and related programs ($0.65 per pack) resulting from the early termination of the average smoker's career, and the increased cost to group life insurance programs resulting from the reduced life expectancy of smokers ($0.11 per pack). Interestingly, these external costs were much less than the external benefits conferred by smoking. Because smokers died young, the pension payments were much less than they would have been otherwise ($1.82 per pack), and the likelihood that they would be housed in a collectively financed nursing home was also substantially reduced ($0.26 per pack). The result was that each pack of cigarettes smoked conferred a net social benefit amounting to $0.91.

The calculations used to arrive at these figures are quite complex. Cigarettes smoked in different years may have variant health effects. Tar content in cigarettes, for example, has decreased three to four percent since World War II. It is generally believed that cigarettes containing lower amounts of tar cause fewer health problems. Since over the course of a smoking career the social costs generally precede the benefits, the net benefit to society was reduced if future costs and benefits were discounted (standard practice in accounting). The appropriate discount rate to be applied to these calculations is a matter of some dispute. It turned out that with a discount rate of five percent, the lifetime present value of the external effects of smoking amounted to a net external cost of $0.15. Manning et al. point out that smokers more than pay this cost in the form of the state and federal excise taxes imposed on tobacco. The external effects in this calculation are all financial; they stem from private and government programs that have the effect of forcing us to pay for each other's medical care, retirement, and other benefits. Smoking, however, also causes external effects directly, since smoke pollutes the air we all breathe. The value of clean air for nonsmokers could in principle be estimated and added to the total external cost. Manning et al. chose not

to do so, in part because they believe that the bulk of the costs of secondhand smoke is borne by those in the same household as smokers. However, in 1995, taking into account the consequences of second-hand smoke, Viscusi brought the estimate of the net social benefit of smoking down to a more modest, but still beneficial, $0.07 per pack, assessing the costs of second-hand smoke at $0.25 per pack.

Applying the external social-cost framework to smoking and other harmfully addictive activities raises another issue. The vast majority of smokers begin their habit as adolescents, so the obvious question is whether people at that age are making well-informed decisions that take proper account of the lifetime consequences (Goodin, 1989). Adolescents tend to be as well informed about the health risks of smoking as adults, and both groups, if anything, exaggerate these risks (Viscusi, 1992). However well informed they are, most people who acquire a smoking habit nevertheless end up wishing they could quit.

In considering the social costs of illicit drug use, the illegal status of these drugs makes an enormous difference (Kleiman, 1992). The consequences of criminalizing transactions in these drugs include the bloody wars between rival drug-dealing organizations, crime by addicts seeking funds for their next fix, and the spread of disease through use of unclean needles, as well as the billions of dollars spent in law-enforcement efforts. Harwood et al. estimated that, in 1992, drug abuse problems incurred a social cost of $97.7 billion.

CONCLUSION

In conclusion, the effort to produce estimates of the social costs of drinking, smoking, and drug abuse is motivated by an interest in establishing a scientific basis for setting priorities in government programs. This effort has produced some useful results and a good deal of controversy surrounding the issue of what is to be counted and how. The task of estimating the social costs of substance abuse requires an accounting framework, and the choice of a framework is not a technical, scientific issue but rather a matter of political philosophy. This is surely one area where the numbers do not speak for themselves.

(SEE ALSO: *Accidents and Injuries; Complications; Economic Costs of Alcohol Abuse and Alcohol Dependence; Productivity: Effects of Drugs and Alcohol on*)

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SOCIAL MODEL *See* Disease Concept of Alcoholism and Drug Abuse

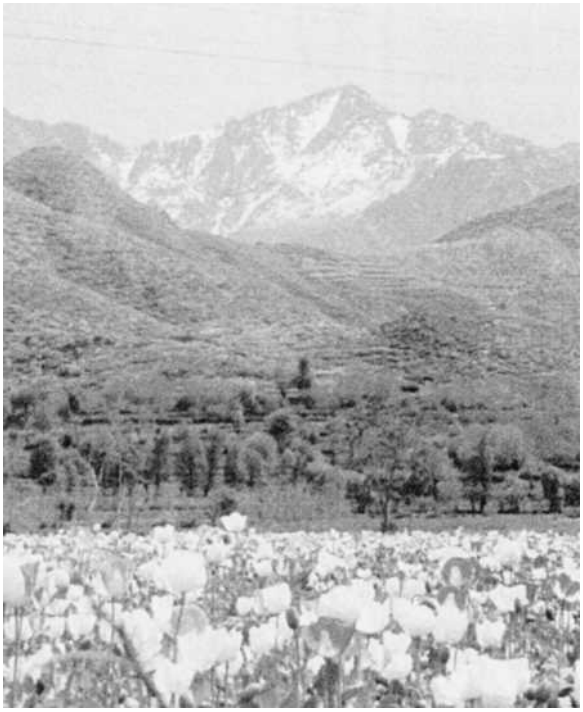
SOCIETY OF AMERICANS FOR RECOVERY (SOAR) *See* Treatment Programs/Centers/Organizations: An Historical Perspective

SOLVENTS *See* Inhalants

SOURCE COUNTRIES FOR ILLICIT DRUGS The 1987 Omnibus Drug Bill requires the U.S. Department of State to develop a list of all major illicit drug-producing and drug-transit countries. Inclusion on the list has an immediate effect, because sanctions include cutting off foreign assistance, other than humanitarian and counternarcotics aid. In addition, the U.S. will block loans by the World Bank to countries that are included on the list.

Major illicit drug producing country is defined in the statute as any country producing "during a fiscal year five (5) metric tons or more of OPIUM or opium derivative, 500 metric tons or more of coca, and 500 metric tons or more of MARIJUANA." (One metric ton equals 1.102 tons.)

The major source countries for HEROIN are Afghanistan, Pakistan, Iran, and Lebanon; Myanmar (formerly Burma), Thailand, and Laos; Mexico, Guatemala, and Colombia. Heroin production rose dramatically in South America in the 1990s. Colombian and Mexican heroin have supplanted Southeast and Southwest Asian heroin in much of the United States. Major source countries for COCAINE are BOLIVIA, Colombia, Peru, and Ecuador. However, in the 1990s, the governments of Bolivia and Peru substantially reduced those countries' cultivation of coca plants. Despite these efforts, drug traffickers shifted their production to Colombia, which by 2000 had become the dominant producer of cocaine. Major source countries for marijuana are MEXICO, Belize, COLOMBIA, and Jamaica. However, the U.S. Drug Enforcement Administration estimates that much of the marijuana consumed in the United States is grown domestically, qualifying the U.S. as a source country. Major source countries for HASHISH are Lebanon, Pakistan, Afghanistan, and Morocco.



Over 60 percent of the heroin that is sold in the United States originates in the poppy fields of Southeast Asia, particularly Myanmar, Thailand, and Laos. (Drug Enforcement Administration)

Measured in U.S. dollar value, at least 80 percent of all illegal drugs consumed in the United States are of foreign origin, including all the cocaine and heroin and significant amounts of marijuana. The opium poppy flower, coca bush, and marijuana plant represent cash crops for indigenous populations—who use the proceeds of sale for subsistence, improvements in lifestyle, and/or means to procure weapons to engage in antigovernment activities. The cultivation of illicit drug crops often represents the most viable—at

times the only viable—economic alternative available to otherwise impoverished farmers and political refugees.

CERTIFICATION

Chapter 8, Section 481 (h) of the Foreign Assistance Act, known as the Certification Law, links the provision of foreign aid to positive drug-control performance. The law also requires the president to certify whether major drug-producing and drug-transit countries have "cooperated fully" with the United States, or have taken adequate steps on their own, to prevent illicit drug production, drug trafficking, drug-related MONEY LAUNDERING, and drug-related corruption. A later amendment to the act requires countries to take adequate steps to implement the 1988 United Nations Drug Convention. Four outcomes of the certification statute deliberation are possible: (1) full and unconditional certification; (2) qualified certification for countries that would not otherwise qualify on the grounds that the national interest of the United States requires the provision of foreign assistance; (3) denial of certification; or (4) congressional disapproval of a presidential certification, which causes statutory sanctions to be imposed.

The annual International Narcotics Control Strategy Report (INCSR) is prepared by the U.S. Department of State and provides the factual basis for the president's decision on certification. The certification statute introduces the concept of variability, by using phrases such as "cooperated fully," "taken adequate steps," and "maximum achievable reductions." Judgments on a country's relative capability to perform are important factors in making certification decisions; each March, these generate spirited debate between the legislative and executive branches of the U.S. government. In addition, this very public decision-making produces tensions between the U.S. and the countries in question.

(SEE ALSO: *Drug Interdiction; International Drug Supply Systems; Transit Countries for Illicit Drugs*)

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SOUTHEAST ASIA, DRUGS AND *See* Asia, Drug Use in; Golden Triangle; Source Countries for Illicit Drugs

SPECIAL ACTION OFFICE FOR DRUG ABUSE PREVENTION (SAODAP) *See* U.S. Government Agencies

SPORTS AND DRUG USE *See* Anabolic Steroids

STATE DRUG PROGRAMS *See* Appendix, Volume 4

STEROIDS *See* Anabolic Steroids

STILL Still is the colloquial term for distillery, a device used for DISTILLATION—to extract ethyl alcohol (ethanol) from various plants and food products. The simplest ones contain a cooking pot and a tightly fitted cap from which a long arm extends in a downward direction. A mash is boiled, the ethyl alcohol rises to the top and is deposited as a vapor which then condenses as it cools and passes through the arm.

(SEE ALSO: *Alcohol: History of Drinking*)

SCOTT E. LUKAS

STIMULANTS *See* Drug Types

STP *See* DOM

STRAIGHT, INC. *See* Appendix, Volume 4

STREET DRUGS *See* Slang and Jargon

STREET VALUE When drugs are seized by a police or interdiction agency, the significance of the seizure is often measured in terms of its street value, that is, the revenues that would be fetched if each gram were sold at the current retail price. Such measures are routine among police and customs service agents in the United States and in most other nations, although large price fluctuations can occur from one area to another and within short time frames.

The use of the term *street value* is potentially misleading when it is intended to convey the significance of the seizure as a loss to the traffickers. The price of drugs rises steeply as they move down the distribution chain from point of importation. In the 1990s, for example, a gram of cocaine could sell on the streets of a U.S. city for about $75. That gram (1,000 milligrams) contained approximately 700 milligrams (mg) of pure cocaine—so that the "pure gram" price was about $106. Yet when sold in 100-kilogram (kg) units at the point of import, the cocaine could have sold for a pure-gram price of about $20. Thus it would cost drug traders $2 million to replace the 100 kilograms. That figure is the total value of payments that would have to be made to growers, refiners, and smugglers in order to obtain another 100 kilograms and bring the drug to the same point in the distribution system.

Valuing a 100-kg seizure at street value would then imply that the government had inflicted a $10.6 million blow to the drug industry, more than five times as much as the true value of the loss. The extent of overstatement increases with the size of the seizure, since the price of drugs goes down as the volume increases in a given transaction.

(SEE ALSO: *Drug Interdiction; Drug Laws: Prosecution of; Seizures of Drugs*)

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STRESS *Stress* is best thought of as a negative emotional state—a psychophysiological experience that is both a product of the appraisal of situational and psychological factors as well as an impetus for coping (Baum, 1990). Stressors—events posing threat or challenge or otherwise demanding effort and attention for adaptation—are judged in terms of the situational variables and one's personal attributes and assets. Negative affect may ensue; and stress responses, which appear directed at the mobilization of bodily systems as a means of coping, strengthen specific problem solving aimed at eliminating the sources of threat or demand and at reducing emotional distress (Baum, Cohen, & Hall 1993).

(SEE ALSO: *Vulnerability As Cause of Substance Abuse*)

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STRUCTURED CLINICAL INTERVIEW FOR DSM-IV (SCID) This is a diagnostic interview designed for use by mental health professionals. It assesses thirty-three of the more commonly occurring psychiatric disorders described in the fourth edition of the DIAGNOSTIC AND STATISTICAL MANUAL (DSM-IV) of the American Psychiatric Association (1994). Among these are MOOD DISORDERS (including MAJOR DEPRESSIVE DISORDER), PSYCHOTIC DISORDERS (including SCHIZOPHRENIA), ANXIETY DISORDERS (including PANIC DISORDER) and the substance-use disorders. The SCID is a semi-structured interview that allows the experienced clinician to tailor questions to fit the patient's

understanding; to ask additional questions that clarify ambiguities; to challenge inconsistencies; and to make clinical judgments about the seriousness of symptoms. The main uses of the SCID are for diagnostic evaluation, research, and the training of mental-health professionals.

The SCID is modeled on the standard clinical interview practiced by many mental-health professionals. It begins with an overview section that includes questions about basic demographic information (e.g., age, marital status), educational history, and work history, followed by questions about the chief complaint, past episodes of psychiatric disturbance, treatment history, and current functioning. The remainder of the interview is organized into the following sections: mood episodes, psychotic symptoms, differential diagnosis of psychotic disorders, differential diagnosis of mood disorders, substance-use disorders, anxiety disorders, somatoform disorders, eating disorders, and adjustment disorder. A separate interview, the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) is available for the assessment of personality disorders.

The SCID comes in two basic versions: the research version (known as the SCID-I) and the clinician version (SCID-CV). The research version contains the full complement of disorders, subtypes and specifiers that are of interest to researchers. It is provided by the Biometrics Research Department at Columbia University as an unbound packet of pages so that the investigator has the ability to leave out pages covering disorders or subtypes that are not relevant to a particular study. The bound clinician version (published by American Psychiatric Press) includes only those disorders and specifiers that are the most clinically relevant. Training materials and computerized versions are also available. Additional detailed information about the SCID (including differences between the research and clinician versions, ordering information, training materials, references) is available on the SCID web site (www.scid4.org).

The substance use disorders covered in the SCID are dependence and abuse for seven classes of substances: alcohol, sedative-hypnotics-anxiolytics, Cannabis (marijuana), Stimulants, Opioids, Cocaine, and Hallucinogens/PCP. For each substance, the interviewer determines whether the symptoms of dependence or abuse have ever been present during the subject's lifetime; whether they have been present during the last month; and age when the first symptoms appeared. If dependence is current, the interviewer rates the current severity as mild, moderate, or severe. If dependence is in partial or full remission, the appropriate DSM-IV remission specifier is noted (e.g., early partial remission, sustained full remission, etc.). Because alcohol use is so much more common the other substance use, the assessment for alcohol dependence and abuse is conducted first, followed by an assessment of dependence or abuse on the remaining categories of substances.

The *ALCOHOL* (ethanol) section of the SCID begins with some overview questions about the subject's drinking history (e.g., "has there ever been a period when you had five or more drinks on one occasion?" "has anyone ever objected to your drinking?"). The subject's answers to these initial questions allow the interviewer to sequence the assessment questions to match the subject's drinking history as follows: If a history of dependence seems likely (e.g., the subject reports a history of detoxification from alcohol or attendence at AA), the interviewer begins with the assessment of the individual DSM-IV dependence criteria. (If criteria are met for dependence, the assessment of abuse is skipped since a DSM-IV diagnosis of dependence pre-empts a diagnosis of abuse; if criteria are not met for dependence, then the interviewer continues with the assessment of abuse). If the history is not suggestive of dependence but is indicative of excessive drinking or problematic use, the interviewer commences with the individual DSM-IV criteria for abuse. (If the criteria are met for abuse, the interviewer must then continue the assessment to see if the problematic drinking is sufficiently severe to qualify for dependence). Only if there have never been any episodes of excessive drinking and there is no evidence of alcohol-related problems can the interviewer skip the alcohol section and move on to the assessment of other substances.

The drug section of the SCID is similarly structured to tailor the sequence of questions to the subject's drug-taking history. If, for any class of substance, the subject reports having used the substance on at least 10 occasions in any one month period, the interviewer starts with the assessment for dependence. If the subject reports using a substance at least twice, but less than 10 times in any month, the assessment focuses on abuse. (As with the assessment for alcohol, if criteria are met for

abuse, the interviewer follows up with the assessment for dependence). For prescribed medications, the interviewer checks for dependence if the subject reports taking having been "hooked" on the medication or reports often taking more of it than prescribed.

(SEE ALSO: *Addiction: Concepts and Definitions; Complications: Mental Disorders; Disease Concept of Alcoholism and Drug Abuse; Epidemiology of Drug Abuse; International Classification of Diseases*)

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STUDENTS AGAINST DESTRUCTIVE DECISIONS (SADD) In 1981, Robert Anastas, a health educator and hockey coach in Wayland, Massachusetts, stood helplessly by as two of his students died of injuries sustained in two separate alcohol-related traffic crashes. Anastas decided to fight back and developed a fifteen-session high school course on driving while impaired. Rather than a curriculum focusing solely on the effects of alcohol while driving, he taught strategies for preventing driving after drinking, and he emphasized the legal consequences of getting caught. In this sense, the curriculum was a significant departure from traditional driver-education approaches.

Students who took Anastas's course reacted enthusiastically and formed an organization to reduce alcohol-related traffic deaths among their peers. They initially called the organization Students Against Driving Drunk (SADD) in order to focus attention on the act of drunk driving, not on the drivers themselves. An anecdote related by Peggy Mann (1983) captures SADD's approach and philosophy: When a student jokingly suggested that SADD involve the governor, Anastas replied, "I believe that if you dream it, it can be done," and when the governor became the honorary chairman of SADD, its motto became "If You Dream It, It Can Be Done." Within a year, chapters had been formed throughout Massachusetts and the program was gaining national attention.

Members of the early SADD chapters had a number of goals. They sought to raise awareness of impaired driving among students through the curriculum developed by Anastas. They also sought to change norms related to impaired driving. Because they realized that most of their peers did not think of drinking and driving as wrong or risky, they reasoned that changing these norms was an important component of reducing impaired driving problems. As the students put it, they wanted to change the "drinking and driving is cool" image to another image: "Drinking and driving is dumb." Finally, students in the SADD chapters undertook to simulate discussion between high school students and their parents concerning drinking and driving. To meet this goal, they developed a "Contract for Life." The contract stipulated that a student would call a parent if he or she had been drinking or if the person responsible for driving had been drinking, and the parent, in turn, agreed to provide a ride or taxi fare.

SADD was significant in three important ways. First, it was among the earliest prevention programs to emphasize student leadership. Other programs had used peer educators or peer counselors trained and supervised by adults, but SADD chapters were run by students who planned activities and took responsibility for making them happen. Second, SADD was among the first youth programs to recognize the importance of norms in impaired-driving prevention. Earlier programs had emphasized education, attitude change, or scare tactics. Third, SADD was one of the first school-based prevention programs to venture outside the classroom. Although SADD had a curriculum, it also entailed

extracurricular, community, and family involvement. In this sense, SADD was the first of the so-called comprehensive school-based prevention programs.

SADD's early growth was rapid. By the mid 1980s, there were SADD chapters in every state in the United States and chapters in Europe. SADD received considerable media attention and was the only alcohol-prevention program ever to be the subject of a nationally broadcast made-for-television movie ("Contract for Life: The Bob Anastas Story").

SADD was also controversial. Some vocal critics argued that SADD's emphasis on preventing drinking and driving implicitly condoned drinking by young people. They were particularly concerned about the Contract for Life— they argued that by insuring safe transportation, parents were communicating the message that drinking itself was not a problem. Similar charges were leveled at Safe Rides and other programs that provided sober transportation for youth. Anastas and others countered that although drinking itself *was* a problem, young people were dying from traffic crashes, not just from drinking.

This debate, which resulted in the refusal by some funding agencies to allow grant money to be used to support SADD chapters, raged throughout the 1980s. SADD was also subject to criticism because of its acceptance of funding from the alcoholic beverage industry. In 1989, SADD divorced itself from this source of funds. It also adopted a strong "No Use" message and amended its Contract for Life to emphasize its commitment to a drug- and alcohol-free lifestyle. The organization specifically disassociates itself from "safe rides" and "designated driver" programs. However, it continues to characterize itself as an "inclusive, not exclusive" organization, recognizing that teenagers make mistakes and should not be punished for them.

Over the years, SADD has evolved. Junior high school and college programs have been added, as has an emphasis on seat-belt use. In 1997, in response to calls from its chapters, the organization amended its popular name to Students Against Destructive Decisions, incorporating in its mandate other potentially destructive behaviors such as underage drinking and drug use, teen suicide, violence, and HIV/AIDS. Today, SADD chapters focus primarily on education, awareness and peer support activities on a range of issues around risky behaviors. In recent years, several student safety clubs with very similar approaches to that of SADD have emerged. Members of these clubs, like SADD members, encourage students reaching out to other students to reduce highway deaths.

As is the case with many widespread, visible prevention efforts, little measurable data can be summoned to show whether or not SADD is effective in reducing drinking and driving among youth. In 1995, the Preusser Research Group, with funding from the National Highway Traffic Safety Administration, performed an evaluation of SADD's effectiveness and concluded that students attending a SADD school were exposed to substantially more activities and information about the risks of underage drinking and drinking and driving. The survey also found that students at SADD schools were more likely to hold positive attitudes against drinking and driving.

(SEE ALSO: *Accidents and Injuries from Alcohol; Dramshop Liability Laws; Drunk Driving; Mothers Against Drunk Driving; Prevention Movement*)

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SUBSTANCE ABUSE *See* Addiction: Concepts and Definition

SUBSTANCE ABUSE AND AIDS AIDS stands for acquired immunodeficiency syndrome: AIDS is a life-threatening disease that results from severe damage to part of the body's cellular immune system—the defense system against opportunistic infections and some cancers. The disease is acquired (as opposed to genetic or hereditary) and presents a myriad of clinical manifestations (syndromes) that result from severe damage to the immune system. AIDS was first identified in 1981

among homosexual men in California and New York, and among illicit injected-drug abusers in New York City. After 1981, the numbers and types of AIDS patients increased rapidly; it was diagnosed in millions of persons throughout the world. In the United States alone, the Centers for Disease Control (CDC) estimated in 1996 that 1 million persons were HIV-positive and 223,000 were living with AIDS.

By 1996, injecting drug abusers accounted for 26 percent of cases among men, 70 percent of cases among women, and about 55 percent of pediatric cases—the children of mothers who are either injecting drug abusers or the sexual partners of male injecting drug abusers. As of 1997, it was estimated that 84 percent of HIV-positive women were of childbearing age; 41 percent of them were drug abusers. AIDS is one of the 10 leading causes of death in children between one and four years of age. Women with AIDS do not live as long as men, though the reasons for this finding are still unclear. The finding has been attributed to the hormonal changes of pregnancy, to poverty, and to violence against women. AIDS has been diagnosed among injectors of various illicit substances, including OPIATES, COCAINE, AMPHETAMINES, and ANABOLIC STEROIDS. AIDS has also been reported among noninjecting drug abusers, such as alcoholics, cocaine "snorters," and crack (cocaine) smokers, who have been infected through sexual contact. An epidemic like AIDS that spans the continents is appropriately called a pandemic.

CAUSE

AIDS is caused by a viral infection. In the United States, the virus is called HIV (for human immunodeficiency virus); it is one of a group of viruses called retroviruses (so-called because they can make DNA copies of their RNA—the reverse of what typically occurs in animal cells). In 1983, French researchers discovered the virus, which they had linked to an outbreak of enlarged lymph nodes (one early sign of HIV infection) that had been reported among French male homosexuals. The French named it the lymphadenopathy-associated virus (LAV). In 1984, U.S. researchers isolated HIV from AIDS patients and named it human T-lymphotropic virus type III (HTLV-III). American investigators found a way to grow HIV in laboratories in large amounts, which led to the development of laboratory tests that detect HIV infection.

HIV gradually destroys certain white blood cells called T-helper lymphocytes or CD4+ cells. The loss of these cells results in the body's inability to control microbial organisms that the normal immune system controls easily. These infections are called opportunistic because they take advantage of damage to part of the immune system. A few select cancers are also frequently diagnosed, such as Kaposi's sarcoma, a cancer of blood vessels, which appears as purplish spots on the skin or mucous membranes.

The sharing of needles contaminated with HIV for injecting drugs of abuse may lead to infection with HIV—but drug abuse may also act as a cofactor with HIV, affecting the development of AIDS. A co-factor in AIDS is a non-HIV-related influence operating in conjunction with HIV to affect the cause of the disease. For example, HIV-infected individuals who continue to inject drugs and/or continue tobacco use may not survive as long as those who do not abuse those substances. The abuse of nitrite INHALANTS ("poppers") among HIV-infected homosexual men may promote the development of Kaposi's sarcoma.

SIGNS AND SYMPTOMS

Early HIV Infection. The natural history of HIV disease and the time intervals between clinical events vary greatly from individual to individual. The general course, however, is one of exposure to HIV, which leads to infection. Within a few weeks or months of infection, laboratory evidence of infection can be detected as the presence of virus in the blood (viremia) or the appearance of the p24 antigen. Antibodies to HIV are found in the blood and indicate that infection has occurred. Some patients develop flulike symptoms resembling mononucleosis or peripheral nerve abnormalities that are self-limited. This first stage of HIV infection is called the acute retroviral syndrome. Most patients have no symptoms during this period.

Latency Period. Over the ensuing years of a second, or latency, period (1&endash;15 or more years), laboratory evidence of a decreasing number of helper T-lymphocytes can be measured. As the helper T-lymphocyte count decreases, patients are more likely to develop such signs and symptoms as enlarged lymph glands, fatigue, unexplained fever,

weight loss, diarrhea, and night sweats. At about the same time or later, patients develop opportunistic infections or cancers. The diagnosis of one of the opportunistic infections or cancers indicates that the patient has developed AIDS. *Pneumocystis carinii* pneumonia, a fungal infection of the lung, is the most common opportunistic infection among AIDS patients. Other opportunistic infections include candidiasis of the mouth (thrush), cryptococcal meningitis, amebiasis, and cryptosporidiosis. Tuberculosis is another serious infection that has become increasingly common because of the AIDS pandemic.

Late-Stage AIDS. Late-stage AIDS is usually marked by a sharp decline in the number of lymphocytes, followed by a rise in the number of opportunistic infections and cancers. Kaposi's sarcoma is the most common cancer among AIDS patients. Kaposi's sarcoma usually arises in the skin and looks like a bruise or an area of bruises, but it grows and spreads to the internal organs. Another common type of cancer in late-stage AIDS is a form of lymphoma, or a tumor of the lymphatic system. Patients with late-stage AIDS may also develop inflammations of the muscles, arthritis-like pain in the joints, and AIDS dementia complex. AIDS dementia complex is marked by loss of reasoning ability, apathy and loss of initiative, loss of memory, and unsteadiness or weakness in walking.

DIAGNOSIS AND TREATMENT

Infection with HIV can be diagnosed with a blood test measuring antibodies to the virus. Antibodies are proteins produced by certain white blood cells in response to injection. The HIV antibody test became widely available in 1985. As of the late 1990s patients were usually given an enzyme-linked immunosorbent assay (ELISA) test for the presence of HIV antibody. Positive ELISA results are then tested with a western blot assay for confirmation. The polymerase chain reaction (PCR) test can be used to detect the presence of nucleic acids from HIV in the very small number of patients who have false-negative results on the ELISA and Western blot tests. The use of these tests by blood banks has greatly reduced the chances of contracting infection from transfusions.

Although a cure or vaccine for AIDS had not been discovered as of 2000, three groups of antiviral drugs are used to treat HIV infection.

Nucleoside Analogues. These drugs work by interfering with the replication process of the HIV virus. They include zidovudine (ZDV, AZT), didanosine (ddI), zalcitabine (ddC), stavudine (d4T), and lamivudine (3TC).

Nonnucleoside Reverse Transcriptase Inhibitors. These drugs work by blocking the activities of the RNA and DNA in infected cells. They include nevirapine and delavirdine. The drawback of this group of drugs is that the virus quickly develops resistance to them.

Protease Inhibitors. These are considered the most potent antiviral drugs. They inhibit the viral proteinase enzyme, which results in noninfectious particles of virus. The protease inhibitors include saquinavir, ritonavir, indinavir, and nelfinavir.

As of 1999 through 2000, these drugs were usually given in combinations of at least two and preferably three compounds. Triple combinations including one of the protease inhibitors are considered the most powerful antiviral regimens. All antiviral treatment regimens must be individualized to the patient.

HIV TRANSMISSION

HIV can be transmitted from person to person in three ways: (1) by contact with infected blood or blood components; (2) through intimate sexual contact; and (3) from an infected pregnant mother to her fetus. Drug abusers commonly become infected by sharing needles, syringes, and other injecting paraphernalia; injecting substances—such as heroin, cocaine, and amphetamines—after an HIV-infected person uses the needle and syringe causes direct inoculation of HIV. Using any paraphernalia contaminated with blood (even in quantities too small to see) can result in HIV or hepatitis B virus transmission. Sexual contact is a common route of transmission from drug abusers to their sex partners (who can transmit the virus to other sex partners, other drug abusers, or to unborn children). Health-care workers have also been exposed to HIV through unprotected or accidental direct contact with blood of infected patients in health-care settings.

We do not know how many individuals are HIV infected worldwide. The World Health Organization (WHO) estimated in 1995 that 18 million adults and 1.5 million children had been infected worldwide, producing about 4.5 million cases of

AIDS. Most of these cases are in the developing countries of Asia and Africa. Numerous HIV surveys have been conducted among injecting drug abusers in several parts of the world. As those currently HIV infected progress to AIDS, the healthcare systems and social fabric of many nations will be severely challenged.

HIV does not appear to be contagious in other settings. No known cases of AIDS have been linked to transmission in nonsexual social or household situations, through air, food, or water, or by mosquito bites.

PREVENTION AMONG DRUG ABUSERS

Methadone Maintenance Treatment (MMT). Because no reliable cure or vaccine for HIV infection exists now (nor is one expected to exist in the near future), the hope for slowing the spread of HIV infection is through education and behavior-changing strategies. Among injecting drug abusers, the most effective way to avoid HIV infection is to stop sharing infected needles, or, better yet, stop injecting drugs, and to avoid sexual contact with individuals who may be HIV-infected. Former drug abusers in drug-abuse treatment have been consistently found to have lower HIV infection rates than those on the streets. Methadone maintenance therapy has been shown to be an effective therapy for opiate addicts and has decreased HIV transmission among compliant patients. As of 2000, the rates of patient compliance among patients in maintenance methadone treatment were higher among women than men; higher among Caucasians than among minorities; and higher among older than younger patients. The National Institute on Drug Abuse (NIDA) continues to conduct research on innovative treatment for drug abuse.

HIV Counseling. The use of HIV antibody tests, counseling about HIV infection, and partner notification projects in drug-abuse treatment programs have thus far met with limited success. A *Morbidity and Mortality Weekly Report* issued in June 2000 noted that men who have sex with men and also abuse drugs (MSM/IDU) still pose unique challenges to slowing the AIDS epidemic because they have multiple risks for HIV infection and transmission. The findings for the period 1985 to 1998 show that over half of MSM/IDU with AIDS were non-Hispanic blacks and Hispanics; that most came from large metropolitan areas; and that the incidence of AIDS has slowly declined since 1996.

Needle Exchange Programs. Some investigators recommend that injecting drug abusers employ "safer" needles and syringes. One approach to reduce HIV transmission among injecting drug abusers is to educate addicts about cleaning needles and syringes between each use. Mechanical cleansing to remove any visible evidence of blood or other debris in the paraphernalia is followed by rinsing with a disinfectant. Of the various disinfectants tested, household bleach appears to be the most effective against HIV. Another approach has been the establishment of needle/syringe exchange programs. Rigorous studies of the effects of such programs on (1) HIV transmission and (2) the recruitment of "new" injectors of drugs will help to show how useful this strategy is.

Newer Strategies. A more recent proposal concerns evaluation of injecting drug abusers for concurrent psychiatric disorders, particularly major depression and antisocial personality disorder, as drug abusers with these disorders are at higher risk of HIV infection. Another strategy is the extension of HIV prevention efforts to abusers of other drugs, most notably cocaine and amphetamines. Lastly, the high rates of HIV infection among Native Americans and Spanish-speaking drug injectors born outside the United States, respectively, have led to concerted efforts to develop group-specific interventions and to recruit outreach workers from these affected groups.

(SEE ALSO: *Alcohol and AIDS; Complications: Route of Administration; Injecting Drug Users and HIV; Needle and Syringe Exchanges and HIV/AIDS; Sweden, Drug Use in*)

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SUBSTANCE ABUSE AND MENTAL HEALTH SERVICES ADMINISTRATION (SAMHSA) *See* U.S. Government Agencies

SUDDEN INFANT DEATH SYNDROME *See* Fetus: Effects of Drugs on; Tobacco: Medical Complications

SUICIDE AND SUBSTANCE ABUSE

With 29,000 annual victims, SUICIDE is the eighth leading cause of death in the United States. Alcohol and illicit drugs are involved in about 50 percent of all suicide attempts. About 25 percent of completed suicides occur among alcoholics and drug abusers. Substance abuse among young adults is largely responsible for the increased suicide rates under age thirty.

The relationship between substance abuse and suicidal behavior has been more extensively studied for alcoholism than for drug abuse. To evaluate this relationship, it is helpful to understand the statistical association between ALCOHOL and drug abuse and suicide, to learn which substance abusers are at particular risk to attempt or commit suicide, and to appreciate how this knowledge may be used to prevent suicide.

SUBSTANCE ABUSE INCREASES SUICIDE RISK

Suicides are not random; each occurs in a particular context. The association between specific psychiatric syndromes—such as DEPRESSION or abuse of alcohol or drugs—and suicidal behavior has been studied by epidemiologists using both retrospective and prospective methods. Since interviews with suicide completers are impossible, retrospective reviews of the circumstances predating suicides have been conducted. By using interviews of relatives and others familiar with the suicide victim, together with study of medical records, suicide notes, and coroner reports, each suicide case is subjected to a "psychologic autopsy." Factors that distinguish successful suicide cases from suicide attempters and substance abusers who have never attempted suicide are compared in the hope that differences in these factors may identify those at particular risk of attempted or completed suicide. A limitation of retrospective studies is termed *recall bias*: informants may provide information about the suicide victim that is distorted by their attempt to explain the suicide event. Although written records and use of standardized methods to collect diagnostic information can reduce this bias, prospective studies are more

reliable. Prospective studies in the general population are not feasible, because suicide is rare, occurring in only about 1 in 10,000 annually; however, about 10 percent of suicide attempters, 15 percent of depressed people, and 3 percent of alcoholics eventually commit suicide. By prospective study of such high-risk groups, additional risk factors can be identified during a follow-up period.

Although most heavy drinkers are not alcoholic, heavy drinking in young adulthood is associated with suicide in middle adulthood. A prospective study of Swedish military conscripts found that those who drank more than twenty drinks weekly had three times the death rate, prior to age forty, of light drinkers. Most of these premature deaths were due to suicide or accidents. Those who develop alcohol dependence or abuse are, together with drug abusers, at increased risk of death from accidents, liver disease, pancreatitis, respiratory disease, and other illnesses; however, suicide is among the most significant causes of death in both male and female substance abusers. U.S. and Swedish prospective studies, for example, found that alcoholism increased the risk of suicide fourfold in men and twentyfold in women.

Next to depression, alcoholism and drug abuse are the psychiatric conditions most strongly associated with suicide attempts. In the U.S. Epidemiologic Catchment Area (ECA) Study conducted in the 1980s, the risk of suicide attempts was increased forty-onefold by depression and eighteenfold by alcoholism. While COCAINE users had increased rates of suicide attempts, users of MARIJUANA, SEDATIVE-HYPNOTICS, and AMPHETAMINES did not.

Among completed suicides, the proportion who were alcoholics or drug abusers is large: Prior to 1980, ALCOHOLISM accounted for about 20 to 35 percent, and drug abuse for less than 5 percent, of suicides in a variety of countries. In the San Diego Suicide Study, conducted in the early 1980s, well over 50 percent of 274 consecutive suicides had alcoholism or drug abuse or dependence. Much of the increase in young-adult suicide rates since the 1960s is attributable to alcoholism and drug abuse or dependence.

RISK FACTORS FOR SUICIDE ATTEMPTS

Alcoholics and drug abusers frequently threaten to kill themselves. Many, particularly women and young adults, actually attempt it. Among alcoholics studied in the ECA communities, 32.5 percent had attempted suicide during a period of active alcoholism. About 15 to 25 percent of alcoholics in treatment programs report having previously attempted suicide. In a group of treated opiate addicts, 17 percent had attempted suicide. This represents at least a fivefold increased frequency of suicide attempts compared to those among nonsubstance abusers.

Although only about 10 percent of substance abusers who attempt suicide will die in a subsequent attempt, most substance abusers who commit suicide have attempted suicide at least once before. Thus, a review of the risks of suicide attempts may guide the identification of those substance abusers at risk of suicidal death. The risk of attempting suicide by an alcoholic or drug abuser is increased by coexisting depression, ANTISOCIAL PERSONALITY disorder (ASP), and a history of parental alcoholism.

Even among people who do not abuse alcohol or drugs, major depression increases the risk of attempting suicide. Major depression is itself 50 percent more common among alcoholics than nonalcoholics: it was found among 5 percent of male and 19 percent of female alcoholics living in the five ECA communities. Depressive feelings (but not necessarily the syndrome of major depression) often motivate alcoholics and drug addicts to enter a treatment program. Typically 20 to 40 percent of alcoholics in such programs have had a period of major depression during their lifetime. While many people drink alcohol or use drugs such as cocaine to reduce feelings of depression, experiments show that consumption produces an initial state of euphoria, followed within a few hours by anxiety, depression, and enhanced suicide ideas. Retrospective studies have found that depressive symptoms are more common among alcoholics who have made a suicide attempt.

Several studies have found that alcoholism in a parent is associated with suicide attempts among alcoholics. In addition, antisocial personality disorder (ASP) and drug abuse, which commonly occur in genetically predisposed males who develop alcoholism early in life, are associated with suicide attempts. Many clinicians have noted the repetitive high-risk behaviors of intravenous drug addicts, who often are quite aware that they may acquire infection or die by overdose with each injection.

Overdoses occur more commonly among HEROIN addicts who have attempted suicide than among those who have not. Highly impulsive and aggressive alcoholics or drug abusers with ASP may be a subgroup at elevated risk of attempting suicide. Transient but intense dysphoria (feeling unwell or unhappy), though not of sufficient scope or duration to meet criteria for major depression, may nonetheless increase this group's risk of attempting suicide.

Prospective studies have found that depression, anxiety, and histories of violence and legal problems were predictive of suicide attempts in previously nonsuicidal drug addicts. Retrospective studies of alcoholics and drug addicts have found that poor social supports, occupational losses, personal losses such as divorce, and other family problems increase their risk of making a suicide attempt.

RISK FACTORS FOR COMPLETED SUICIDE

Although in the general population there is considerable overlap between those who attempt suicide and those who complete suicide, substantial differences exist between these groups. For example, women are three times more likely than men to attempt suicide, while men are three times more likely to commit suicide. Despite these differences, suicide attempters are at higher risk of completed suicide. What, then, are the risk factors for completed suicide in substance abusers?

Depression. Depressed people, particularly men, typically kill themselves in young adulthood. Among pure alcoholics, over 90 percent of suicides occur among men. In contrast to depressives, alcoholic men typically commit suicide in their fifth and sixth decades; usually this follows about twenty years of alcoholism. Men with depression, but not those with alcoholism, continue to be at elevated suicide risk beyond age sixty. Drug abuse shortens the interval preceding suicide: in the San Diego Suicide Study, drug addicts committed suicide after an average of only nine years of heavy use. They typically did so in young adulthood. This suggests that factors other than alcoholism may shorten the suicide risk period in this group. About three of four alcoholic suicides communicate their suicidal intent prior to their deaths. Thus, middle-aged male alcoholics and young polysubstance abusers, especially those who talk of suicide, are at high risk of suicide.

Long-term Use. Ongoing substance use makes suicide more likely. Nearly all alcoholic suicides occur among active drinkers, and alcohol consumption often immediately precedes the suicide. The abstinent alcoholic is only partly protected from suicide, however, for 3 percent of suicides among alcoholics occur among those who are abstinent. It is likely that impulsiveness and transient or syndromal depression contribute to these suicides.

Psychiatric Conditions. Coexisting psychiatric conditions, particularly depression, play an important and perhaps crucial role in the suicide of alcoholics and drug abusers. The vast majority of suicide victims have depressive symptoms at the time of their death. Concurrent depression is the leading factor in at least 50 percent of suicides among alcoholics and drug abusers. SCHIZOPHRENIA, mania, and ASP are also associated with suicide in substance abusers.

Timing. What determines the timing of suicide among substance abusers? Substance abusers often accumulate interpersonal problems throughout their drinking or drug-use careers, but one-third of those who commit suicide sustain a major interpersonal disruption (such as separation or divorce) within the six weeks preceding their deaths. They often are unemployed, living alone, and unsupported by family and friends at the time of this final and most severe disruption. In contrast, only 3 percent of nonalcoholics with depression suffer such a loss in the period before they commit suicide. Beyond psychiatric diagnoses, the strongest indicator of suicide risk in substance abusers is such an interpersonal loss. Beyond these actual losses, anticipated losses, such as impending legal, financial, or physical demise may also increase the risk of suicide among substance abusers. Among alcoholics, those who develop serious medical problems, such as liver disease, pancreatitis, or peptic ulcers, are also at higher risk of suicide.

Summary. Which of these risk factors is the most important, and how do they interact to affect the risk of suicide? To partly answer these questions, Murphy and colleagues studied 173 white male alcoholics, 67 of whom committed suicide. After adjusting for age, the most potent risk factor for suicide was (1) current drinking, followed by (2) major depression, (3) suicidal thoughts, (4) poor social support, (5) living alone, and

(6) unemployment. All suicide cases had at least one, and 69 percent had at least four, of these six risk factors. These factors act cumulatively to increase the risk of suicide in male alcoholics significantly. Their relative roles in other groups of substance abusers have not been reported.

CLINICAL FEATURES

Substance abusers who commit suicide often see a physician or are psychiatrically hospitalized in the months prior to their deaths. Those who talk of suicide may be ambivalent about their wish to die. They may thus be amenable to clinical interventions such as detoxification, substance-abuse rehabilitation, or psychiatric hospitalization. Conversely, those who take special precautions against discovery during a prior suicide attempt are much more likely to die in a subsequent suicide attempt.

Feelings of hopelessness are common in depression. While suicide attempters who are depressed and who report hopelessness are more likely to die of suicide, hopelessness is not a particular risk for completion of suicide among alcoholics. This may occur because substance abusers are motivated to commit suicide less by persistent hopelessness and more by impulsive anger, dysphoria, or feelings of isolation or abandonment.

PREVENTION

Prediction of those who will complete suicide remains poor in individual cases, even among high-risk groups such as substance abusers. Despite their high prevalence, alcoholism and drug abuse often go unrecognized by physicians and other health-care professionals. Recognition of alcohol and drug use disorders and of risk factors such as major depression that increase the risk of suicide may assist clinicians with preventive interventions. The substance abuser with active suicide plans or a recent suicide attempt may need hospitalization, detoxification, and/or rehabilitation designed to foster abstinence from alcohol and drugs of abuse. Firearms should be removed from the homes of substance abusers with active suicide ideation, especially adolescents and young adults. Treatments designed to enhance social supports and foster abstinence from alcohol and drugs, together with those directed at resolution of major depression, often reduce the risk of suicide.

(SEE ALSO: *Accidents and Injuries; Complications: Mental Disorders; Epidemiology of Drug Abuse; Social Costs of Alcohol and Drug Abuse*)

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MICHAEL J. BOHN

SURGEON GENERAL, REPORT OF THE

See Tobacco: Medical Complications; Treatment: Tobacco

SWEDEN, DRUG USE IN Sweden is roughly the size of California—or twice that of the United Kingdom. Sweden's capital city, Stockholm, has a population of about 1.3 million, and the country as a whole has some 8.8 million inhabitants. The first well-documented example of drug abuse in Sweden arose during the 1940s, when the technique of injecting AMPHETAMINE began to spread among criminal elements and bohemians in Stockholm. This form of intravenous (IV) drug abuse quickly spread to other major towns and cities and also to the neighboring countries of Finland, Norway, and Denmark. In 1944, central nervous system (CNS) stimulants were subjected to the same strict prescription control regulations as narcotic drugs in general. In Sweden, CNS stimulants were formally scheduled as narcotics in 1958. The classification of CNS stimulants as psychotropic substances in the international convention of 1971 was largely a result of Sweden's efforts.

MARIJUANA (*Cannabis* leaves), declared an illicit drug in Sweden in 1930, enjoyed its first popularity around 1954, when the habit of smoking a "joint" was started by American jazz musicians who were performing in Sweden. HASHISH (*Cannabis* resin) was introduced in the early 1960s and became popular among young people as the habit of smoking "pot" (marijuana) emerged along with the youth rebellion. In the 1990s, the domestic growing of *Cannabis* plants started on a small scale.

The intravenous use of heroin stems from the mid-1970s, and this mode of drug abuse quickly attracted attention from the news media when several overdose deaths were reported. COCAINE was introduced into Sweden in the late 1970s, but on a small scale.

LEGISLATION

In Sweden, the term *narcotic drugs* refers to all pharmaceutical substances controlled under the provisions of the Narcotic Drugs Act (1968) and listed on the Narcotic Drug Schedules issued by the Swedish Medical Products Agency. These schedules contain all internationally controlled substances and some additional substances, such as KHAT (leaves and branches from *Catha edulis*). The use of Schedule I drugs (*Cannabis*, LSD, HEROIN, MDMA, khat, etc.) is prohibited, even for medical purposes.

Narcotic offenses in Sweden fall into three classes:

1. Petty offenses involving possession of small amounts of the drug punishable with a fine or imprisonment for a maximum of six months.
2. Narcotic offenses, which might entail selling ("pushing") drugs on the streets, carry a maximum of three years imprisonment.
3. Grave (serious) narcotic offenses, such as the import of large amounts of illicit drugs or the production and sale of narcotics. These offenses are punishable by imprisonment for two to ten years.

Compulsory (coercive) treatment of drug abusers is allowable under the 1988 law for Treatment of Alcoholics and Drug Misusers. Young offenders may be subjected to compulsory treatment under the Care of Young Persons Act of 1990. The decision to invoke this treatment for young drug abusers is made by the county administrative courts. METHADONE MAINTENANCE treatment for opiate addicts, using very strict admission criteria, is currently available at three university hospital clinics—at Stockholm, Uppsala, and Malmö-Lund.

Doping compounds, such as ANABOLIC STEROIDS, are regulated under the Doping Compounds Act of 1992. These substances cannot be imported, produced, traded, or possessed without special permits; however, use of anabolic steroids is not a punishable offense at the present time.

CURRENT SITUATION AND TRENDS

Since the 1970s, hashish has been the most widespread of the illicit drugs used in Sweden; it is often considered the starting point, or gateway, into abuse of other drugs. During the screening of job applicants in 1986, as many as 4 percent had traces of TETRAHYDROCANNABINOL (THC) in their urine. An estimated 50,000 people regularly smoke hashish in Sweden as of the mid-1990s. A study conducted by UNO (Utredningen om narkotikamissbrukets omfattning, or Commission on the Extent of Drug Abuse) in 1979 revealed somewhere between 10,000 and 14,000 severe drug abusers, or *tung missbrukare*, that is, users who take drugs either on a daily basis or intravenously, exclusive of frequency. A similar study in 1992 found this number had increased to between 14,000 and 20,000.

Amphetamine, which is relatively easily obtained throughout the country, is the most popular drug of abuse for intravenous use; about 10,000 people are currently using this CNS stimulant. Injection of heroin seems to be mainly concentrated in the southern and central metropolitan areas, where some 2,000 to 3,000 are known to indulge in this form of drug abuse. The abuse of cocaine is primarily seen within jetset circles in the major cities. The smoking of CRACK-cocaine is uncommon in Sweden. HALLUCINOGENS (such as LSD and Ecstasy) are used to some extent by adolescents who follow the "rave" culture. Plant hallucinogens such as PSILOCYBIN are rarely encountered, as are PHENCYCLIDINE (PCP), "ice" (crystallized METHAMPHETAMINE) and phentanyl (e.g., fentanyl, sufentanil) opioids. Solvent (inhalant) abuse is on the rise in Sweden, with 10 percent of 16-year-old boys and 6 percent of 16-year-old girls reporting usage in 1999. Those who use this type of product for the purpose of intoxication can be treated under the Care of Young Persons Act or the Care of

Alcoholics, Drug Abusers and Abusers of Volatile Solvents (Special Provisions) Act.

Increased immigration into Sweden during the 1980s brought the development of new subpopulations of drug users, with use patterns derived from their home drug cultures. These included the smoking of opium and heroin, which is common to the Middle East, or the chewing of khat from East Africa. The relaxing of border controls with the Eastern bloc led to new smuggling routes for drugs into Sweden—hashish from Russia and amphetamine from Poland.

According to figures obtained from the Stockholm Remand Prisons, human immunodeficiency virus (HIV) infection rates in the early 1990s were approximately 30 percent among IV abusers of heroin and 5 percent among IV abusers of amphetamine. About 600 individuals are apprehended each year in Sweden on suspicion of driving under the influence of drugs. The most common drug encountered in people suspected of driving under the influence of narcotics is amphetamine, followed by *Cannabis* and then various SEDATIVE-HYPNOTIC prescription drugs belonging to the BENZODIAZEPINE family.

Annual studies of drug use by school children (aged 16) and military conscripts (aged 18) have been conducted in Sweden for some time by CAN, the Swedish Council for Information on Alcohol and Other Drugs. In 1998, CAN reported that 9 percent of 16-year-old boys and 6 percent of 16-year-old girls had tried drugs, a number roughly double that reported in 1991. Among the military conscripts, 16 percent reported having experimented with drugs at least once, up from 6 percent in 1991. Two-thirds of those who reported having tried drugs had used only cannabis, with amphetamine following as the second most-tried drug.

SHIFTS IN CONTROL POLICY

Sweden has experienced dramatic shifts in public policy concerning the control of illicit drugs. In 1965, after a turbulent media campaign, the medical authorities were obliged to allow certain doctors to prescribe what were illicit drugs to registered addicts for their personal use, as part of the so-called legal prescription experiment. Over a two-year period, about 4 million doses of amphetamine and 600,000 doses of morphine had been distributed to a total of only 150 addicts. The project rapidly became unmanageable; it was stopped as the IV drug habit began to spread widely and several fatal overdoses were reported. During the final twelve months of the project, the prevalence of IV drug use among the arrestee population in Stockholm had doubled.

In 1969, a nationwide police offensive against all sorts of drug-related crime brought about a dramatic decrease in drug abuse in Sweden. The tendency among public prosecutors to dismiss petty drug offenses during the 1970s led to an escalation in drug abuse once again. Since 1980, all drug offenses have been either referred to the courts for trial or, if the suspects plead guilty to petty offenses, they are fined directly. In the late 1980s, the police began a new strategy against drug abuse, by focusing more attention on all kinds of drug activity on the streets—with the aim of decreasing the demand for drugs.

The fight against drug abuse in Sweden grew progressively stricter between 1983 and 1993. In 1988, the taking of illicit drugs was made a punishable offense. Since July 1, 1993, the police have been allowed to order chemical analyses of body fluids for evidence that a suspect has been taking illicit drugs. The primary goal of Swedish drug policy is to establish and maintain a narcotics-free Sweden. Measures employed in this effort include information campaigns (prevention), strict border controls to minimize smuggling, mandatory treatment programs for offenders, street-level interventions, and legal restrictions on sale, use, and production of drugs. Sweden's drug policy is often held up as model for other European nations, but has recently come under attack by those alarmed by the steady increase in drug use despite these strict controls.

(SEE ALSO: *Amphetamine Epidemics; Britain, Drug Use In; Drug Testing and Analysis; Italy, Drug Use in; Netherlands, Drug Use in the*)

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SYNAPSE, BRAIN The term synapse is from the Greek word *synaptein*, for "juncture" or "fasten together," by way of the Latin *synapsis*. It refers to the specialized junction found between nerve cells. It was conceived by the British pioneer neurophysiologist Sir Charles Sherrington (1857–1952) to describe the then-novel microscopic observations that the "end-feet" of one neuron physically contacted, in an intimate manner, other NEU-

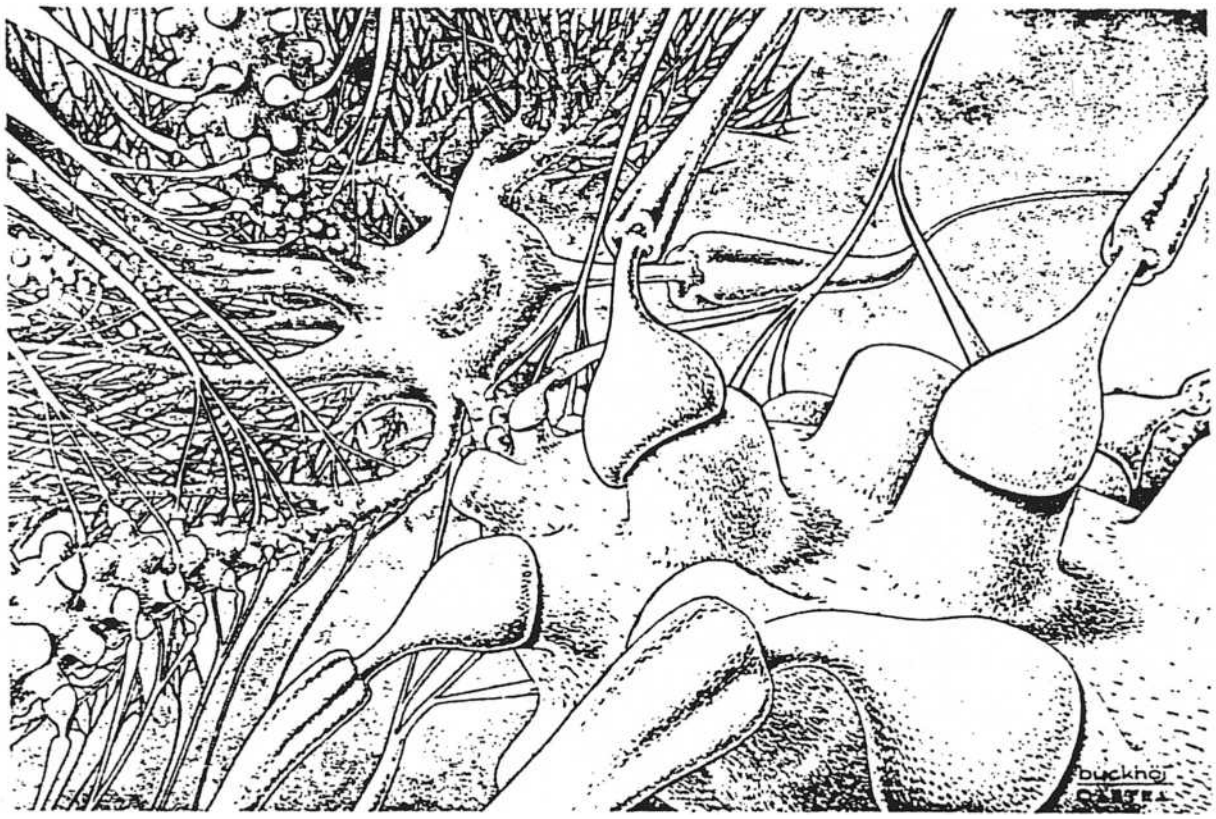

Figure 1
Synapse. The nerve ending from one neuron forms a junction, the synapse, with another neuron (the postsynaptic neuron). The synaptic junction is actually a small space, sometimes called the synaptic cleft. Neurotransmitter molecules are synthesized by enzymes in the nerve terminal, stored in vesicles, and released into the synaptic cleft when an electrical impulse invades the nerve terminal. The electrical impulse originates in the neuronal cell body and travels down the axon. The released neurotransmitter combines with receptors on postsynaptic neurons, which are then activated. To terminate neurotransmission, transporters remove the neurotransmitter from the synaptic cleft by pumping it back into the nerve terminal that released it.
SOURCE: Figures 1 and 2 have been modified from Figure 1, in M. J. Kuhar's "Introduction to Neurotransmitters and Neuroreceptors," in Quantitative Imaging, edited by J. J. Frost and H. N. Wagner. Raven Press, New York, 1990.

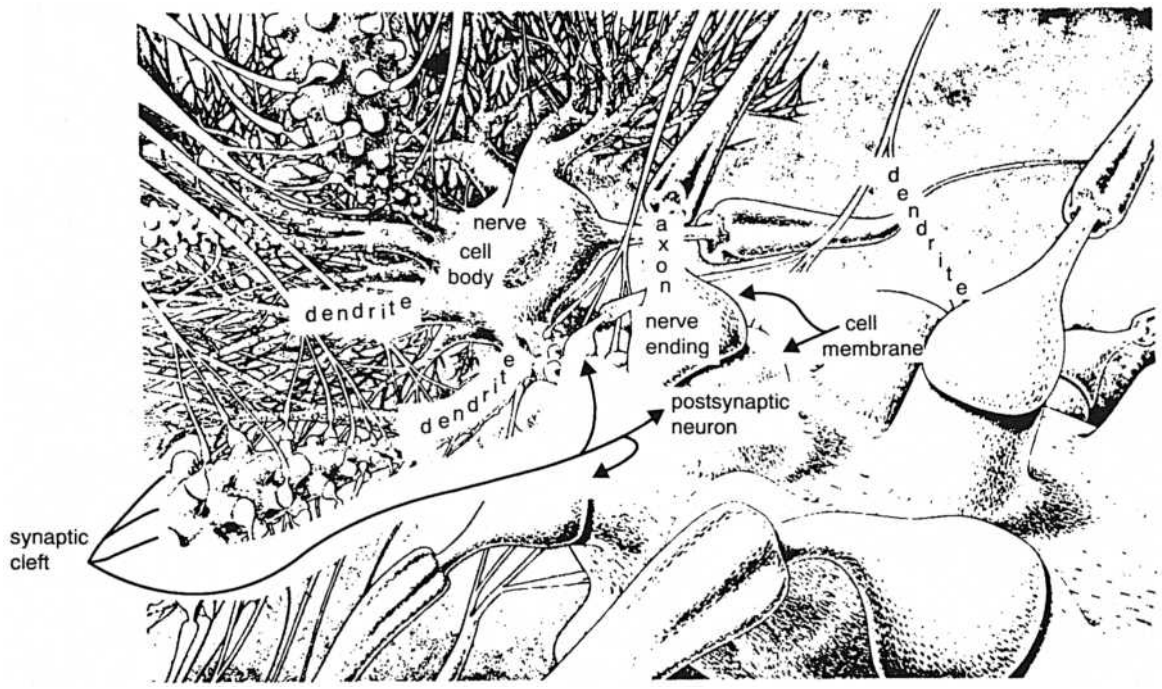

Figure 2
Neuronal Network. Synapses can be seen here with their narrow synaptic clefts, only 20 micrometers wide, across which a nerve impulse is transmitted from one neuron to the next. Hundreds of thousands of nerve endings may form synapses on the cell body and dendrites of a single neuron. As an electrical impulse reaches the synaptic cleft, it cannot be transmitted because of a discontinuation in the cell membrane. To bridge this cleft, another type of transmission, a chemical transmission, begins, mediated by a chemical compound—the transmitter substance or a neurotransmitter.

RONS to which it was structurally connected. A similar point of connection between peripheral nerves and their targets is usually referred to as a *junction*.

Synapses in the brain (see Figures 1 and 2) are morphologically typed by several features (1) a dilation of the presynaptic terminal (nerve ending) that contains accumulations of synaptic vesicles in various sizes, shapes, and chemical reactivities; (2) mitochondria; (3) a specialized zone of modified thickness and electron opacity in the presynaptic membrane, in which a presynaptic grid is perforated to provide maximum access of transmitter-containing vesicles to the presumptive sites of transmitter release; and (4) a specialized zone of altered thickness and opacity in the postsynaptic membrane termed the *active zone* and believed to be the site of initial response.

The synaptic vesicles have been shown to contain the NEUROTRANSMITTERS by a series of extensive analyses of meticuously purified vesicles. The vesicles differ in their protein content and may include the transmitter's synthetic enzymes, as well as the transporters that can concentrate the transmitter within the vesicles. For MONOAMINE neurons, the vesicles also contain specific proteins (named for their sites of discovery in the adrenal medulla as *chromogranins* but now termed more generally *secretogranins*. These are assumed to facilitate storage and release. Superficially, synapses with a thinner postsynaptic specialization, of about the same thickness as that at the presynaptic membrane (hence termed *symmetrical*), are often inhibitory;

those with a thickened postsynaptic membrane (*asymmetrical*) are often excitatory.

Monoaminergic synapses, however, are often asymmetrical, as are those for peptide-containing neurons that do not obey these simple physiological categorizations. Synapses can also be discriminated on the basis of the pairs of neuronal structures that come together at this site of functional transmission. Most typical is the *axo-dendritic* synapse in which the axon of the presynaptic neuron contacts either the smooth or spiny surface of the dendrite of the post-synaptic neuron. A second common form is the *axo-somatic* synapse in which the presynaptic axon contacts the surface of the post-synaptic neuron's cell body (or somata). Less frequently observed are axo-axonic relationships in which one axon contacts a second axon-terminal that is in its own axo-dendritic relationship; such triads of axo-axo-dendritic synapses are found most frequently in spinal cord and certain midbrain structures, in which channels of information flow are necessarily highly constrained. Most rarely, junctions between cell bodies (somato-somatic) and dendrites (dendro-dendritic) have also been described.

The nature of the proteins that provide for the thickened appearances of the active zones by electron microscopy are not completely known, but they include the postsynaptic receptors and associated molecules that can transduce the signals from the activate receptors, as well as those molecules that serve to concentrate the receptors in such locations.

(SEE ALSO: *Brain Structures and Drugs; Neurotransmission; Reward Pathways and Drugs*)

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FLOYD BLOOM

SYNANON *See* Treatment Programs/Centers/Organizations: An Historical Perspective

SYRINGE EXCHANGE AND AIDS *See* Needle and Syringe Exchanges and HIV/AIDS

T

TASC *See* Treatment Alternatives to Street Crime

TAX LAWS AND ALCOHOL The first internal revenue measure adopted by the U.S. Congress, in 1790, was an excise tax on domestic whiskey; a subsequent increase in that tax from 9 to 25 cents per gallon led to an armed insurrection by the farmers of western Pennsylvania during the summer of 1794, the so-called Whiskey Rebellion.

This matter of the appropriate level for alcoholic beverage taxes has remained contentious to this day; although there is consensus that alcoholic beverages should be subject to higher taxes than other commodities, substantial disagreement remains concerning the appropriate level for such taxes. The principal impetus for raising tax rates has always been the quest for increased government revenue. Since the 1970s, however, increasing attention has been paid to the public health benefits of alcohol taxes, as research has demonstrated that raising the excise tax rates, and hence the prices of alcoholic beverages, reduces traffic fatalities and other costly consequences of alcohol abuse.

HISTORY

Alcoholic beverage taxes were a major source of revenues for the federal government throughout much of U.S. history. As recently as 1907, this source accounted for 80 percent of federal internal tax collections and was still as high as 10 percent on the eve of U.S. entry into World War II. Currently, the federal excise taxes and import duties continue to have a considerable effect on the prices of alcoholic beverages, but figure very lightly (less than 1%) in overall federal tax collections.

Because federal excise taxes are set in dollar terms per unit of liquid, rather than as a percentage of the price, inflation gradually erodes the real value of these taxes. For example, while Congress increased the tax per fifth of 80-proof spirits by 29 percent (to $2.16) between 1951 and 2000, the overall level of consumer prices increased by over 550 percent during this same period. The result is that the *real* value of the federal liquor tax had declined by 2000 to just one-fifth of its value in 1951. A considerable reduction in the average price of whiskey and other spirits relative to the prices of other commodities has been the inevitable result.

The states also impose special excise taxes on alcoholic beverages, as do some local governments. In addition, alcoholic beverages are generally subject to state and local sales taxes. The relative importance of these tax collections in state budgets differs widely, but as of 2000 is everywhere less than 10 percent of government revenues.

TAX EFFECTS

When a legislature raises the excise tax rates on alcoholic beverages, the resulting cost to distributors is passed along to consumers in the form of

higher prices. As is true for other commodities, the sales of alcoholic beverages tend to fall when prices increase. This is not to say that price is all that matters. For example, the steady decline in sales and consumption of alcohol during the 1980s cannot be explained by increased prices, since the prices of alcoholic beverages remained more or less constant (in real terms) during this period. The downward trend in consumption presumably resulted from the aging of the population and increasing public concern with healthy lifestyles, among other factors. Per capita sales and consumption of alcohol are nevertheless negatively affected by alcohol beverage prices, and if Congress had increased federal excise taxes substantially during the 1980s, sales would have declined still more rapidly than they did.

Although they differ somewhat, a number of published estimates of the price elasticity of demand for beer, wine, and liquor tend to confirm that price is one of the important variables influencing sales. One review of these estimates concluded that the price elasticity for liquor is approximately -1.0; this implies that, other things being equal, a percentage increase in the average price of liquor will result in an equal percentage reduction in the quantity of liquor sold. Beer and wine sales tend to be somewhat less responsive to price, with estimated price elasticities in the neighborhood of -0.5 (Leung & Phelps, 1993). Estimates for other developed countries are quite consistent with these conclusions (Edwards et al., 1994; Cook & Moore, 2000).

These results do not in themselves imply that a general price increase for alcoholic beverages will reduce consumption of ethyl alcohol (ethanol), the intoxicating substance in all these beverages. In the face of higher prices, consumers can switch to higher- proof brands, reduce wastage, and attempt home production of beer or wine. But in practice, research suggests that these substitutions are not large enough to negate the price effect. Ethanol consumption does tend to fall in response to a general increase in the price of alcoholic beverages.

Given the fact that higher alcohol excise taxes increase prices and reduce ethanol consumption, there remains the vital question of whether alcohol taxes are effective instruments in preventing alcohol-related harms. Of public concern are both the harms associated with the *acute* effects of inebriation—injuries stemming from accidents and violent crime—and the harms resulting from *chronic* heavy drinking, most notably the long-term deterioration in health and productivity.

There is considerable evidence that the incidence of both inebriation and chronic heavy drinking, and the associated harms, are sensitive to the prices of alcoholic beverages. For the acute effects, Cook (1981) studied 39 instances in which states increased their liquor tax between 1960 and 1975, finding strong evidence that traffic fatalities in those states fell as a result. This result was confirmed for the beer excise tax by Ruhm (1996) and Saffer & Grossman (1987), both using panel data on state traffic fatality rates. Cook & Moore (1993), also using panel data on states, found a close link between per capita ethanol consumption and violent crime rates, and direct evidence that an increase in the beer tax helped suppress rape and robbery. And, Chesson et al. (2000) use a similar method to demonstrate that the incidence of sexually transmitted disease is inversely related to the beer tax. This literature is not without dissenters (see Dee, 1999), but the bulk of the published research results provide support for the conclusion that alcohol excises influence the incidence of inebriation and the costly consequences thereof.

There is also evidence of a link between alcohol prices and the prevalence of chronic heavy drinking. Cook & Tauchen (1982) demonstrated that changes in state liquor taxes had a statistically discernible effect on the mortality rate from cirrhosis of the liver. Since a large percentage of liver cirrhosis deaths result from many years of heavy drinking, it appears that chronic heavy drinkers are quite responsive to the price of alcohol. This conclusion is supported by evidence from clinical experiments and other sources (Vuchinich & Tucker, 1988).

Thus, there is indeed evidence that alcohol taxes are an effective instrument for preventing alcohol-related harms. The claim that alcohol taxes promote the public health is increasingly important in the public debate over raising federal and state alcohol taxes.

FAIRNESS

Although alcohol taxes reduce consumption and save some lives that would otherwise be lost to alcohol-related accidents, there remains a question of whether they are "fair." Fairness is largely in the

eye of the beholder (or taxpayer); nevertheless, several standards are commonly used as bases for judging the fairness of a tax. Two of the most notable standards are that a tax should fall equally on households which are in some sense equally situated, and that it should not be regressive.

If equals are to be treated equally, is it fair that alcohol taxes force drinkers to pay more taxes than nondrinkers of similar incomes? Indeed, the bulk of all alcohol taxes are paid by the small minority who drink heavily: Half of all alcohol consumption is accounted for by just 6 or 7 percent of the adult population. One response is that it is fair for drinkers to pay more, because drinking imposes costs on others. One estimate suggests that drinkers impose an average cost on others amounting to about 25 cents per drink (Manning et al., 1990); Miller et al. (1998) provide a much higher estimate. Thus, if the alcohol tax is considered a sort of "user fee," whereby the drinker pays in proportion to the amount of alcohol consumed, then it may seem fair.

Another concern is that alcohol taxes may be regressive, meaning that on the average, wealthier households spend a smaller fraction of their income on alcohol taxes than poorer households. Although it is often taken as self-evident in political debates over raising beer taxes, the evidence on this matter is not clear (Sammartino, 1990; Cook & Moore, 1993).

Another debated issue is that of uniform taxation. A can of beer, a glass of wine, and a shot of spirits all contain approximately the same amount of ethanol, but are taxed quite differently; the federal excise tax on a shot of spirits exceeds the tax on a can of beer by a factor of 2, and on a glass of wine by a factor of 3. If special taxes on alcoholic beverages are ultimately justified by the fact that such beverages are intoxicating, then these disparities are difficult to explain. Part of the explanation may be the widespread belief that spirits are in some sense more intoxicating than beer or wine, and hence more subject to abuse, whereas beer is the "drink of moderation" and wine "the drink of connoisseurs." But much of the evidence works against this view. Indeed, beer consumption may be more costly to society (per drink) than spirits because of the demographics of beverage choice: young men, a group that consumes most of their ethanol in the form of beer, has by far the highest incidence of alcohol-related traffic accidents and violent crimes.

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PHILIP J. COOK

TEA Tea is the most widely consumed beverage in the world, except for water, and provides over 40 percent of the world's dietary CAFFEINE. In the United States, caffeine from tea accounts for about 17 percent of caffeine consumed; per capita caffeine consumption from tea is about 35 milligrams per day, which is a little over one-third of the daily caffeine provided by coffee beverages. Tea consumption in the United Kingdom is substantially higher, averaging 320 milligrams per capita per day and accounting for 72 percent of the United Kingdom's caffeine consumption.

Although tea contains a large number of chemical compounds, the relatively high content of polyphenols and caffeine is responsible for tea's pharmacological effects. The primary psychoactive component of tea is caffeine. Tea also contains two compounds that are structurally related to caffeine, theophylline and THEOBROMINE, however, these compounds are found in relatively insignificant amounts. On average, a 6-ounce (177-milliliter) cup of leaf or bag tea contains about 48 milligrams of caffeine, a little less than half the caffeine in the same amount of ground roasted coffee, and only slightly more than the amount found in 12 ounces of a typical COLA soft drink. Six ounces of instant tea contain 36 milligrams caffeine, on average. Individual servings of tea contain amounts of caffeine that can affect mood and performance of adult humans.

Figure 1
Tea

Although the term *tea* has been used to refer to extracts from a large number of plants, only teas derived from leaves of *Camellia sinensis* plants are of special interest here, because they contain caffeine. The term *tea* has come to be used especially for extracts of *Camellia sinensis* and that restricted usage is maintained in this entry.

Consumption of *Camellia sinensis* was first documented in *China* (where tea is called *cha* or *chai*) in 350 A.D., although there is some suggestion that the Chinese consumed tea as early as 2700 B.C. Tea was introduced to Japan around 600 A.D. but did not become widely used there until the 1400s. Through the China trade, tea became available in England in the 1600s, where it became the national drink. Tea was introduced into the American colonies around 1650 but in 1773 became a symbol of British rule. Americans protested the British tax on tea by raiding ships anchored in Boston Harbor and dumping boxes of tea into the water. This event, referred to as the Boston Tea Party, along with other similar protests that followed, became important in shifting the predominant caffeinated beverage in North America from tea to coffee.

India, China, and Sri Lanka are the major producers and exporters of tea—producing about 60 percent of the world's tea and providing about 55 percent of world tea exports. The United Kingdom, the United States, and Pakistan are the leading importers of tea.

Two types of tea, black and green tea, account for almost all of the tea consumed in the world. Black tea makes up over 75 percent of the world's

tea; green tea accounts for about 22 percent. The method by which tea is manufactured determines whether black or green tea is produced. Black tea is dark brown in color and is produced by promoting oxidation of a key tea constituent. Green tea is yellow-green in color and is produced by preventing such oxidation, a less processed tea. Oolong tea, a less common type, is partially oxidized and is intermediate in appearance to that of black and green tea. Flavored teas were originally prepared by adding a range of fruits, flowers, and other plant substances to the tea prior to final packaging, although artificial flavors are often added today.

(SEE ALSO: *Chocolate; Plants, Drugs from*)

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TEMPERANCE MOVEMENT Many temperance movements and societies emerged in the United States during the nineteenth century. These movements began in the early 1800s and gained ascendancy during the mid-to-late 1800s, culminating in the Prohibition Movement, the Prohibition Amendment (Article 18) to the U.S. Constitution in 1919, and the start of Prohibition in 1920. Gusfield (1986), an eminent scholar of the temperance movement, has argued that the term *temperance* is not appropriate, because the broad reformist ideology of the movement focused mainly on abstinence—not moderation—in the intake of alcoholic beverages. Blocker (1989) observed that the many temperance movements that emerged in the United States represented men and women from varying ethnic, religious, social, economic, and political groups who selected out temperance as the solution to what they perceived as problems in their own lives and in those of others. By the end of the nineteenth century, the temperance movement had evolved through several phases, and the strategies used by the proponents changed from persuasive efforts to moderate the intake of alcoholic beverages to more coercive strategies, even laws, to bring about the control of all drinking.

EARLY PHASE: 1800–1840

In colonial America and during the early 1800s, alcoholic beverages (brewed, fermented, and distilled) were a staple of the American diet, were often homemade, and were viewed as "the good creature of God." Among the colonists, the drinking of alcoholic beverages was integrated with social norms; all social groups and ages drank alcoholic beverages, and the consumption rate was very high. Alcohol was also traded, sold, and given to Native Americans, who had no long history of daily drinking, with almost immediate negative consequences for these peoples.

By 1840, a revolution in American social attitudes had occurred, in which alcohol came to be seen as "the root of all evil" and the cause of the major problems of the early republic, such as the crime, poverty, immorality, and insanity of the Jacksonian era (Tyrell, 1979). Temperance was advocated as the ideal solution for these problems by such people as Anthony Benezet, a popular Quaker reformer; Thomas Jefferson; and Dr. Benjamin Rush, the surgeon general of the Continental Army and a signer of the Declaration of Independence. Temperance-reform organizations, such as the American Temperance Society, emerged, committed to the eradication of these social problems.

The American Temperance Society (ATS), founded in Boston in 1826 as the American Society for the Promotion of Temperance, was the first national (as opposed to local) temperance organization. It had its roots in the processes of industrialization and the commercialization of agriculture. The people who developed the movement were committed to hastening the processes of economic and social change. These processes involved the educating of Americans to value sobriety and industry, in order to create the conditions for the development of an industrial-commercial society. The movement was supported by entrepreneurs who needed a disciplined and sober work force to help create the economic change necessary for the material improvement of the young republic.

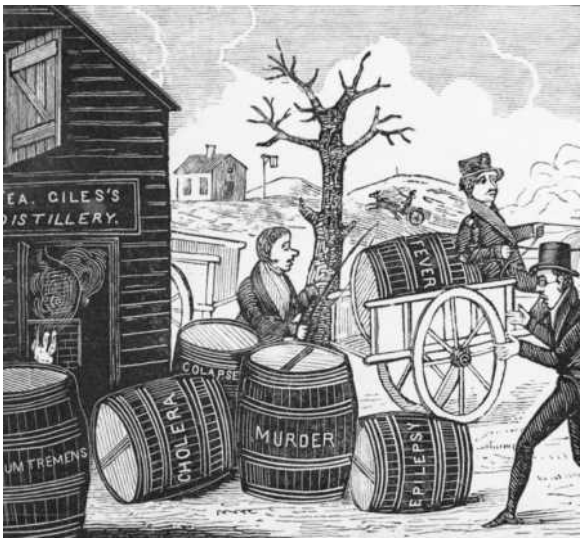
During the so-called Great Awakening the evangelical clergy as well as that of other U.S. Protestant

groups supported temperance as a means of promoting the morality needed for building a "Christian nation," through social and economic progress. According to Gusfield, these groups helped to place the issue of drinking on the public and political agenda, providing their personnel as authorities on the cognitive aspects of drinking and becoming the legitimate source of public policies on drinking. Also, in the early 1820s and 1830s, small-scale farmers and rural groups were active in promoting the temperance movement; they saw temperance as a way to promote social progress in a time of transition from a rural to an urban-industrial order, from small-scale farming to entrepreneurial forms of agriculture.

By 1836, the American Temperance Society had become an abstinence society, and ideas about problems associated with alcohol had begun to change—inebriety or habitual drunkenness was being called a disease. The ideology of the movement placed the source of alcohol addiction in the substance itself—alcohol was inherently addicting—a finding supported by research conducted by Rush, who in 1785 wrote *Inquiry into the Effects of Ardent Spirits upon the Human Body and Mind* (approximately 200,000 copies were published between 1800 and 1840). Blocker (1989) observed that the general focus of the American Temperance Society was on persuading the already temperate to become abstinent, rather than persuading drunkards to reform their drinking behavior. According to Gusfield (1986), abstinence became a symbol that enabled society to distinguish the industrious, steady American worker from other people—which resulted in the movement becoming democratized instead of associated only with the New England upper classes. Attempts to reform and save drunkards was the focus of another temperance movement, the Washingtonians.

MIDDLE PHASE: 1840–1860

Where well-to-do groups and Protestant evangelical clergy dominated the early phase of temperance reform, the middle phase included the efforts of artisans and women of the lower and lower-middle classes, who promoted self-help groups among largely working-class drunkards trying to give up drinking (Tyrell, 1979). These artisans organized into the Washingtonian societies (named for George Washington), dedicated to helping



A woodcut dating to the early phase of the temperance movement illustrates the physical and moral afflictions attributed to alcohol. Circa 1820. (© Bettmann/CORBIS)

working-class drunkards who were trying to reform.

In 1840, the (first) Washingtonian Temperance Society was established in Baltimore. Members took a pledge against the use of all alcoholic beverages and attempted to convert drunkards to the pledge of teetotalism (c. 1834, derived from *t*otal + *total* = abstinence). By the end of 1841, Washingtonian societies were active in Baltimore, Boston, New York, and other areas throughout the North. These groups were not socially homogeneous. Tyrell (1979) observed that the relationships between the old organizations and the new societies culminated in various struggles for control over the Washingtonian societies, with fragmentation of these groups occurring.

Washingtonian members who wanted respect from the middle-class temperance reformers, including the evangelical reformers, elected to remain with the mainstream temperance movement. The wage earners and reformed drunkards remained in their own societies, and they opposed early efforts at legal coercion—for example, the passage of the Maine Law of 1851. Gusfield (1986) has interpreted support for this law as a reaction against the drinking practices of the Irish and German immigrants to the United States between 1845 and 1855. He argued that temperance reform in this

period represented a "symbolic crusade" to impose existing cultural values on immigrant groups. Tyrell interpreted the Maine Law as a way for middle-class reformers to control and reform the laboring poor. From 1851 on, many local laws were passed that attempted to limit the consumption of alcohol; however, throughout the remainder of the century, these statutes were repealed, liberalized, or unenforced.

LATE PHASE: 1860–1920

The Civil War, World War I, and the rapid demographic changes that accompanied immigration during this period contributed to the support of abstinence during the last phase of the temperance movements. Urban areas were expanding, factory towns were a reality, and there was an increase in the socializing at the end of the workday as well as at the end of the workweek; consequently there was an increase in the production and consumption of alcoholic beverages. Several temperance societies that emerged during this period included the active participation of women and children—since wives and children were often neglected or abused by drunken husbands and fathers. Irish-American Catholics formed the Catholic Total Abstention Union in 1872; the WOMEN'S CHRISTIAN TEMPERANCE UNION (WCTU) was formed in 1874; and the Anti-Saloon League (ASL) emerged in 1896. These societies were able to mobilize tremendous support for abstinence, rather than mere moderation in the intake of alcoholic beverages. At this time, the ideology of the temperance movements centered upon the evil effects of all alcohol, espousing the view that alcohol had become the central problem in American life and that abstinence was the only solution for this problem.

The WCTU was founded in Cleveland in 1874 and emerged as the first mainstream organization in which women and children were systematically involved in the temperance movement. Annie Wittenmeyer, Frances Willard, and Carrie Nation provided this temperance-reform movement with creative and dynamic leadership. The WCTU—a crusade to shut down saloons and promote morality—took a radical stance, criticizing American institutions by aligning itself with the feminist movement, the Populist party, and Christian Socialism. Gusfield (1986) argues that, although, under the leadership of Frances Willard (1879–1898), the WCTU was unsuccessful in establishing these alliances, it did achieve the following: It united the Populist and more conservative wings of the movement and it united the political forces of "conservatism, progressivism, and radicalism in the same movement." In addition, the WCTU provided backing for Prohibitionist candidates, including workers for their campaigns as well as audiences to listen to their positions on alcohol use. The WCTU still exists, based in Evanston, Illinois, and lists about 100,000 members as of 1990.

By the late 1800s, coercive reform became the dominant theme of the temperance movement. In 1893, the ASL of Ohio was organized by Howard H. Russell, a Congregational minister and temperance activist. In 1895, this group combined with a similar group in the District of Columbia, establishing a national society in 1896. By the end of the 1800s, the ASL, which represented a skillful political leadership resource for the Prohibition movement, mobilized tremendous support for abstinence instead of just temperance. In 1896, the movement began to separate itself from a number of economic and social reforms, concentrating on the struggle of traditional rural Protestant society against developing urban systems and industrialization.

Part of the success of the ASL was its determination to remain a single-issue (prohibition) pressure group that cut across all political party lines; the ASL also maintained a strong relationship with the Protestant clergy. It always put its own issue first but worked peacefully with the major political parties and especially with legislators (Blocker, 1989). By 1912, local prohibition laws had been passed to render most of the South legally dry.

In 1917, a major event boosted the cause of national prohibition. The United States entered into World War I, which prompted the ASL to push for the suspension of the industrial distilling of alcohol (ethanol). Very shortly after the U.S. entry into the war, the selling of liquor near military bases and to servicemen in uniform was prohibited (Blocker, 1989). By 1918, the Eighteenth Amendment to the U.S. Constitution had been proposed and the ASL had pushed prohibition through 33 state legislatures. Consequently, the Volstead Act—called Prohibition—was ratified on January 16, 1919. It went into effect one year later, on January 16, 1920, prohibiting the manufacture, sale, or transportation of alcoholic beverages.

CONCLUSION

Where the temperance movement was a middle-class reform movement, because it articulated the theme of self-control that was central to the middle-class ideology of the nineteenth century, some members of the working class also supported reform (Blocker, 1989). An ideology of ABSTINENCE became a rallying point for middle-class people who saw the rich as greedy, the working class as increasingly restless, and the poor as uneducated immigrants. Thus, they felt the need to restore a coherent moral order, especially after the upheaval of the Civil War and the ensuing period of industrial greed. At this time, the United States was undergoing economic expansion and deepening division along class lines. Other reform groups, such as the Progressive political party, joined the prohibitionists in their commitment to rid cities of saloons so that the United States could move toward becoming a virtuous and moral republic. At the end of the nineteenth century, Americans seemed to be more receptive to moral than scientific arguments for temperance reform and abstinence from alcohol.

Members of the temperance movements were concerned not only with changing the behavior of other social classes and groups but also about changing themselves (Levine, 1978). They were concerned that the pernicious effects of alcohol were also destroying the lives of Protestant middle-class people. While some of these reform groups were not complete supporters of an abstinence ideology, they were concerned with rebuilding a national community and promoting the common welfare. Abstinence became the governing ideology of the many diverse groups that had mobilized to promote a new social order.

As more scholars turn their attention to the study of the temperance era and the various temperance movements and societies, additional knowledge and interpretations will continue to be published. The bibliography that follows provides examples of some new interpretations of this period.

(SEE ALSO: *Alcohol; Prohibition: Pro and Con; Treatment*)

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PHYLLIS A. LANGTON

TEMPOSIL *See* Calcium Carbimide

TERRORISM AND DRUGS The term *narcoterrorism* has entered the popular lexicon as a shorthand to refer to the complex relationship between the illicit drug trade and terrorism. The term, however, has often been used interchangeably to refer to two distinct aspects of this issue.

EXPLOITING THE DRUG TRADE

Narcoterrorism refers, first, to the activities of a number of guerrilla groups worldwide. These groups engage in terrorism and insurgency and also exploit the drug trade for financial gain. In most

cases this exploitation involves rural-based guerrillas. Guerrillas and the drug trade (especially cultivation and processing) both tend to thrive in rugged, remote areas where government control is weak and where a nationally integrated economic infrastructure is lacking.

Rural-based guerrillas make money primarily by extorting "war taxes" from growers and traffickers. Thus the relationship between guerrillas, on the one hand, and the growers and the traffickers, on the other, is frequently rooted in coercion and conflict.

Nevertheless, guerrillas, growers, and traffickers sometimes cooperate in a marriage of convenience. The degree of government pressure exerted in an area can at times act as a unifying factor. Local family and/or personal relationships in a drug region can bring guerrillas, growers, and traffickers together, at least for periods of time.

A number of guerrilla groups have used both coercion and cooperation to exploit the drug trade. Examples include the following: The Revolutionary Armed Forces of Colombia (FARC), the country's largest and oldest insurgent group, and Colombia's National Liberation Army (ELN); Peru's Sendero Luminoso (Shining Path) and the Revolutionary Movement Tupac Amaru (MRTA); and the Kurdish Workers' Party (PKK) in the Middle East.

In addition to or apart from "taxation" and "protection" arrangements, various groups themselves have been directly involved in the drug trade:

In COLOMBIA, the FARC controls its own coca fields and processing laboratories for COCAINE. FARC may have some drug distribution networks, although evidence for this is fragmentary.

In Southeast Asia's GOLDEN TRIANGLE of Thailand, Burma, and Laos, guerrillas have long been actively involved in every stage of the OPIUM/HEROIN pipeline. They have frequently devolved into warlord trafficking organizations and dominate the drug business in the area.

Some guerrillas in the South Asian subcontinent (the Indian peninsula of Bangladesh, Bhutan, Nepal, Pakistan, Sikkim, and India), such as the Tamil Tigers (LTTE) and the Sikhs, have used expatriate communities abroad to smuggle heroin.

Lebanon's Hizballah reportedly smuggles drugs as a result of a *fatwah* (an Islamic religious decree). In 1987, the police uncovered narcotics in a Hizballah terrorist arms cache near Paris, France.

USING THE TACTICS OF TERROR

The second aspect covered under the rubric of *narcoterrorism* has been the drug traffickers' use of the tactics of political terrorism—such as the car bomb, kidnapping, and selective assassination—to undermine the resolve of various governments at the highest levels to fight the drug trade.

Traffickers usually use members of their own organization to carry out such attacks. Sometimes, however, traffickers have subcontracted to guerrillas. In late 1990, Colombia's Pablo Escobar used the ELN to help conduct kidnappings to pressure the Colombian government into negotiating with him.

Colombia has been hardest hit by the traffickers' use of terrorist tactics. Escobar's Medellín trafficking group was responsible for a string of vicious attacks in the 1980s and early 1990s. Among the victims and targets were a justice minister, an attorney general, Supreme Court justices, the editor of a leading newspaper, several presidential candidates, a commercial airliner, and the headquarters of Colombia's equivalent of the FBI.

Escobar scored a major victory by using narcoterrorism along with bribery to ensure the banning of extradition between Colombia and the United States in 1991. With the aid of corrupt officials, Escobar escaped from a jail in 1992 and continued to carry out sporadic attacks until he was killed by Colombian authorities in December 1993.

Escobar's death, however, did not end the relationship between terrorist groups and drug traffickers. During the 1990s, Peru and Bolivia successfully reduced the amount of coca production, but this led to a dramatic rise in production in rural Colombia. Guerrilla and paramilitary groups control the major drug-producing regions, mostly in southern Colombia. Drug money enables these groups to purchase sophisticated weapons on the black market that are used against government forces. The situation in Columbia continued to deteriorate in the late 1990s, to the point that the U.S. government gave Colombia $1.3 billion in emergency aid in 2000 to help fight the narcoterrorists. However, it remains to be seen whether this funding and additional military aid will turn the tide against narcoterrorism.

Mexico also saw an upsurge of terrorist acts in the 1990s. However, these acts were committed by drug traffickers and were not the product of revolu-

tionary groups. The assassination of political candidates and government officials demonstrated the vulnerability of the government to terrorist acts. For example, in February 2000, the police chief of Tijuana, Alfredo de la Torre, was assassinated as he drove to his office without bodyguards. The assassination came two days after the government announced a new attack on drug trafficking in the state of Baja California, where Tijuana is located.

Italy too has suffered from drug violence. During the 1980s and early 1990s, the Sicilian Mafia retaliated for government crackdowns by killing a number of the country's leading prosecutors and law enforcement officers—often with car bombs, in spectacular fashion.

IMPLICATIONS

Narcoterrorism in both its incarnations challenges government efforts to control political violence, organized crime, and the drug trade.

Although involvement in the drug trade may sometimes decrease the revolutionary fervor of a guerrila group, the ability to derive income from this lucrative source strengthens the resources and capabilities of the groups to oppose the central government either as subversives or as a criminal element. Whether or not the guerrillas obtain the funding through coercion or cooperation with growers and traffickers, the result is usually a more formidable foe. Most observers, for example, believe that exploitation of the drug trade is the chief source of funding for Peru's Sendero Luminoso. In general, the presence of guerrillas with an economic stake in the survival of the drug trade makes counternarcotics efforts an even more risky undertaking.

The willingness and ability of drug barons in some countries to use the tactics of terrorism adds a dangerous dimension to the threat posed by the drug trade. In Colombia, narcoterrorism has pushed the country to the brink of civil war and threatens to move the conflict into neighboring countries. In other countries, such as Mexico and some of the newly independent states of the former Soviet Union, there is growing concern about the volatile mix of drugs, violence, and organized crime.

(SEE ALSO: *Crop-Control Policies; International Drug Supply Systems*)

MARK S. STEINITZ
REVISED BY FREDERICK K. GRITTNER

TERRY & PELLENS STUDY In a time when the use of many drugs is illegal in the United States and the public is inundated with information on such drug use, it is probably surprising that this set of circumstances is a historically recent phenomenon. Throughout most of the history of the United States, the manufacture, possession, and use of most drugs now considered addictive were legal, and very little was known about these drugs, their use or abuse.

Other than ALCOHOL (through the TEMPERANCE MOVEMENT), the drug that first captured the attention of policymakers and medical and public-health sciences was OPIUM. An interest in the addiction to opiates in the United States can be found as far back as 1877, when Dr. Marshall conducted a study of the number of opiate addicts in Michigan. However, this and the handful of similar efforts at epidemiological research conducted through 1920 were plagued with methodological problems. Generally these studies were conducted by sending short questionnaires to physicians or pharmacists who, at that time, legally supplied people with OPIUM and opium-based products. These physicians or druggists were simply asked to report the number of opium addicts they saw in their communities. All these studies were done in only one city, county, or state—with one exception. The exception was a study done by the U.S. Department of the Treasury, in an attempt to provide direct estimates of the number of opium-addicted people in the nation. Unfortunately, none of these studies would come close to meeting the requirements of sampling or of measures taken that would be required today.

A very important step forward in the study of drug addiction or dependency in general, and opiate addiction in particular, took place in a now classic study done for the Committee on Drug Addictions of the Bureau of Social Hygiene, in cooperation with the U.S. Public Health Service, by Charles E. Terry and Mildred Pellens from 1923 to 1924 (Terry & Pellens, 1924, 1927, 1928). This study was groundbreaking in several ways. First, rather than sending questionnaires to physicians and pharmacists, only about 30 percent of whom had responded in any of the previous studies, Terry and Pellens used field study techniques—their staff went to the sites of data collection. Second, rather than relying on self-reports, Terry and Pellens took advantage of official records that physicians, den-

tists, veterinarians, institutions, and laboratories were required to keep for all opium distribution, as mandated by the HARRISON NARCOTIC ACT of 1914. Third, and perhaps most important, Terry and Pellens conducted their study in six sites across the United States: Sioux City, Iowa; Montgomery, Alabama; Tacoma, Washington; Gary, Indiana; Elmira, New York; and El Paso, Texas. Although no known precedent existed for such a research strategy, they selected these six cities on the basis of racial characteristics, occupations, geographic region, and other social demographic factors, so that in aggregate these six sites could represent the United States as a whole.

As a consequence of these efforts, Terry and Pellens not only attempted to collect data more accurately but also produced the first study of the EPIDEMIOLOGY of drug addiction or dependence that tried to take into account social and demographic factors that, now as then, affect the number and distribution of people who are addicted to or dependent upon chemical substances. Their book, *The Opium Problem*, which contains chapters on the history of the problem, theories of its etiology, and contemporary treatments, is considered a classic in the field.

(SEE ALSO: *Epidemiology of Drug Abuse; High School Senior Survey; National Household Survey on Drug Abuse; Treatment*)

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ERIC O. JOHNSON

TETRAHYDROCANNABINOL (THC)

Tetrahydrocannabinol, or THC, is a chemical found in the HEMP plant, CANNABIS SATIVA, that causes the PSYCHOACTIVE effects in MARIJUANA, BHANG, HASHISH, and GANJA. Hashish is derived from the resin that oozes from the flowering tips of the female plant; bhang comes from the dried leaves and flowering shoots of the female plant; and ganja comes from small leaves. THC is one of the three natural cannabinoids—chemical constituents of *Cannabis*—the other two being cannabinol (CBN) and cannabidiol (CBD).

As of 2000, marijuana is the most commonly used nonlegal drug in the United States. Its usage peaked during the late 1970s, when about 60 percent of high school seniors reported having tried marijuana, with 11 percent reporting daily use. Usage has declined since 1979; as of 1999, 2 to 3 percent of the 70 million Americans who had tried cannabis described themselves as daily users.

PHARMACOLOGICAL EFFECTS

For more than 30 years, the discovery of the mechanism of THC's action had eluded the best researchers. The problem seems finally to have been resolved by the detection of specific cannabinoid-binding sites (RECEPTORS) in the brain. A further step in unraveling the mechanism of THC's action has been the cloning of the cannabinoid receptor.

The pharmacological effects of THC vary with the dose, the method of administration, the user's degree of experience with THC, the setting, and the user's vulnerability to the psychoactive effects of the drug. Most users seek to experience a "high," or "mellowing out." The high begins about 10 to 20 minutes after smoking and lasts about 2 hours. The psychological effects obtained during the high are often related to the setting in which the drug is taken.

Inhalation. THC is most commonly taken into the body by inhaling the smoke from marijuana "joints." A joint of good quality contains about 500 milligrams of marijuana, which in turn contains between 5 and 15 milligrams of THC. Blood levels of THC rise almost as rapidly after inhaling smoke as they do after intravenous administration of THC. That the drug should be so rapidly absorbed is an indication of the efficiency of the lung as a trap for the drug. THC is quickly redistributed into other tissues so that blood levels decline over the course of 3 hours to negligible amounts. The usual

symptoms of marijuana intoxication are almost completely gone by that time.

Ingestion. THC is absorbed slowly and unreliably from the gut after oral administration. Blood levels of the drug peak between 1 and 2 hours after ingestion. These peak concentrations are also considerably lower than those following smoking.

THC is easily soluble in fats. It is taken up and stored in the fatty tissues of the body and in the gray matter of the brain. This pattern of storage is one reason why THC remains so long in the body.

Withdrawal. THC does not produce a severe withdrawal syndrome. Heavy users, however, frequently report insomnia, nervousness, mild stomach upset, and achy muscles— particularly if they stop their use suddenly.

DRUG TESTING AND FORENSIC ISSUES

Drug testing is an issue with respect to marijuana because of the effects of THC on coordination, sense of timing, and impairment of depth perception as well as short-term memory. It is hazardous for someone who has taken a moderate dosage of marijuana to drive or to operate heavy equipment in the workplace.

Urine testing, however, is hardly useful for determining impairment, since the metabolic products of THC are detectable for as long as 50 days in chronic users. Urine tests are also of little use in determining the patient's pattern of use.

EFFECTS OF THC

THC produces a variety of complex sensations and behavioral effects in humans. The effects on memory, coordination, and sense of time have already been noted. Some studies indicate that THC produces impairment of human cognitive functions as well. In addition, many users experience increased appetite. Psychological effects range from a pleasant sense of mellowness to negative effects that include panic reactions, anxiety, hallucinations, and schizophrenic symptoms. THC can also cause relapses in schizophrenic patients, even those who are taking antipsychotic medications. These negative effects are more common with high doses of the drug and with oral ingestion rather than smoking.

The physical effects of THC include dry mouth, abnormalities in heart rhythm, and abnormal precancerous changes in the tissues that line the airway and the lungs. People who are heavy users of marijuana often develop bronchitis and laryngitis. As of 1999, however, it was not definitely known whether persons who smoke only marijuana have an increased risk of lung cancer, as compared to those who smoke tobacco. THC lowers the sperm count in males and may produce abnormal menstrual cycles in females. Women who are pregnant or nursing are advised to avoid marijuana, as THC is secreted in human breast milk.

MEDICAL USES OF THC

THC has been used in medicine to treat the nausea that many cancer patients experience after chemotherapy. It has also been used to prevent convulsions and to lower the fluid pressure inside the eye in treating glaucoma.

In recent years, THC has been replaced in medical use by a synthetic derivative called dronabinol (Marinol). Dronabinol is used as an antinausea drug, an appetite stimulant in AIDS patients, and an antiglaucoma medication.

(SEE ALSO: *Drug Metabolism; Drug Testing and Analysis; Pharmacokinetics*)

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THC *See* Tetrahydrocannabinol

THEOBROMINE This ALKALOID belongs to the class of drugs called methylxanthines; it is similar to theophylline and to CAFFEINE. Theobromine (3,7-dimethylxanthine), however, is somewhat weaker than these two compounds and currently has almost no practical use in medicine.

Theobromine is found in the seeds of the plant *Theobroma cacao*, which is the well-known source of CHOCOLATE and cocoa. The cacao seeds have caffeine too (as does TEA, which contains small amounts of theobromine and theophylline); caffeine has powerful stimulant effects on the brain, whereas theobromine has very little (although popular articles alleged for years that theobromine makes one feel "happy"). High doses of theobromine can, however, affect several physiological functions in the body, such as increasing the formation of urine in the kidney.

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THERAPEUTIC COMMUNITIES *See* Treatment Types: Theraputic Communities

TOBACCO: DEPENDENCE In the United States as of 1999, there were about 57 million cigarette smokers-representing 25 percent of the adult population. Another 5 percent (men) use smokeless tobacco (chewing tobacco or snuff). Most (70-80%) say they would like to quit. Unfortunately, they are dependent on (addicted to) nicotine, an alkaloid that makes it difficult to stop using tobacco. Most of them will have to try to quit several times before they are successful. Both the direct effects of nicotine on the body and behavioral associations with those effects learned over the years of tobacco use keep people going back for more even when they want to quit.

The role of nicotine in tobacco use is complex. Nicotine acts on the body directly to produce effects such as pleasure, arousal, enhanced vigilance, relief of anxiety, reduced hunger, and body-weight reduction. It may also reverse the withdrawal who is symptoms that occur in a nicotine-dependent person trying to quit, when nicotine levels in the body fall. These symptoms include anxiety, irritability, difficulty concentrating, restlessness, hunger, depression, sleep disturbance, and craving for tobacco. When this happens, the use of nicotine (whether tobacco or nicotine-containing medications) usually makes people feel better by reversing the unpleasant withdrawal symptoms.

Nicotine also acts *indirectly*, through a learning process that occurs when the direct effects of nicotine occur repeatedly in the presence of certain features of the environment. As a result of the learning process, called conditioning, formerly insignificant environmental factors become cues for the direct actions of nicotine. These factors can become either pleasurable in themselves or they can serve as a triggering mechanism for lighting up a cigarette. For example, the taste, smell, and feel of tobacco often evoke a neutral response and sometimes repugnance in a nonsmoker. After years of experiencing the direct effects of nicotine in the presence of tobacco, however, a smoker finds the sensory aspects of tobacco pleasurable.

The indirect or conditioned effects of nicotine are responsible for much more complicated learning than the learning associated with nicotine's direct effects. Conditioning is also the process whereby the situations in which people often smoke such as after a meal, with a cup of coffee, with an alcoholic beverage, while doing a task at work, while talking on the phone, or with friends who also smoke become in themselves powerful cues for the urge to smoke. When people stop using tobacco, therefore, the direct effects of nicotine are not the only pleasures they must give up. They must also learn to forgo the indirect effects of nicotine: those experiences that, through learning, have become either pleasurable in themselves or a cue to smoke.

MOTIVATION FOR QUITTING

Most Americans who use tobacco would like to quit, and the reasons for wanting to quit vary. The most common include (1) a concern for one's health; (2) a concern for the health of one's family and friends (this may entail concern about the harmful effects on children of secondhand smoke or concern about setting a bad example for them); (3) social pressure; (4) and economic factors (cigarettes are expensive).

STAGES OF QUITTING

Successful quitting of tobacco use usually occurs as a process over time, a series of mental stages or steps that the smoker goes through in quitting: 1. *Precontemplation*. The person is smoking and is not motivated to stop smoking during this stage. 2. *Contemplation*. The person is still using tobacco and is motivated to quit but has not settled on a quit date that is within one month. 3. *Action*. The person has a stop date and a plan that was either already implemented or will be implemented within one month. 4. *Maintenance*. The person has discontinued the regular, daily use of tobacco for a minimum of one month.

RELAPSE

Most tobacco users who try to quit agree with Mark Twain, who said, "To cease smoking is the easiest thing I ever did; I ought to know because I've done it is a thousand times." People who are addicted to tobacco and who try to quit are able to do so for a brief period of time, but most resume smoking. For example, 66 percent of smokers who try to quit on their own or with minimal outside help relapse within 2 days, 90 percent relapse within 3 months, and 95 percent to 97 percent relapse within 1 year of quitting. The key to successful smoking cessation is an understanding of what triggers relapse, and what strategies are effective in preventing relapse. Some of the most important triggers for lighting up a cigarette are withdrawal symptoms, environmental cues acquired through learned associations, and emotional upset. Relapse is promoted by such common withdrawal symptoms as difficulty concentrating, irritability, and weight gain. Environmental cues to relapse include the presence of other smokers such as a spouse, friends, or coworkers who smoke and occasions when alcoholic beverages are consumed. Emotional upset and depression are also commonly reported cues for lighting up.

MANAGING URGES TO SMOKE

A smoker who contemplates quitting often thinks that smoking cessation is a simple matter of refraining from smoking during a period of nicotine withdrawal. Urges to smoke are powerful, however, and occur long after the period of nicotine withdrawal has ended. Tobacco users must not only not smoke but must, in fact, learn a new, tobacco-free lifestyle. Some learn on their own; others seek professional help. Key aspects of learning a tobacco-free lifestyle include anticipating and managing withdrawal symptoms and environmental triggers for smoking. The environment might be managed to minimize smoking triggers by, for example, (1) sitting in nonsmoking sections of restaurants; (2) removing ashtrays from one's home and office; (3) leaving the table as soon as possible after meals and engaging in other activities such as talking, walking, or doing the dishes; (4) avoiding (at least temporarily) situations that trigger smoking, such as drinking alcohol or coffee when smokers are around and going to places, parties, or bars where people smoke; (5) actively seeking social support for smoking cessation. The encouragement of a husband or wife, or of friends and others who have quit or are in the process of quitting, also makes it easier. Smokers who enjoy handling cigarettes or having something in their mouths need to substitute something for these smoking-related behaviors. They may chew gum, toothpicks, sunflower seeds, or something similar; munch food or low-calorie snacks; exercise to take up time they might otherwise spend smoking and to reduce any weight gain; snap, roll, or twist rubber bands on their wrist. What people think about while quitting is an important factor in relapse. They need to teach themselves to maintain thoughts that may be useful in overcoming urges to smoke. Instead of thinking about the expected pleasures of a cigarette, the would-be quitter can substitute a stream of thoughts about the risks of smoking, the benefits of not smoking, the commitment to not smoking, the pleasures of an anticipated reward for not smoking, or the day's next activity. Stress management is also important for successful quitting. Smokers soon recognize that giving up smoking is a substan-

tial stress in itself. They can resort to some strategies that may reduce stress, such as meditation, relaxation, and physical exercises. Other aspects of self-management during smoking cessation include setting realistic goals and some sensible rewards for behavior that leads to reducing tobacco use. Some days a realistic goal is a short-term one and involves just getting through each urge to smoke without succumbing. The smoker who is quitting can use any of the already mentioned substitution or distraction strategies while remembering that urges to smoke are likely to continue to come and go for some time. Rewarding oneself for meeting even the short-term goals is important. Rewards for not using tobacco can include new clothes, a new book, time to develop a new hobby, or anything else the former smoker might enjoy. Many rewards can be paid for from money saved by not buying tobacco.

INDEPENDENT QUITTING

Most smokers quit smoking without professional help. People who quit on their own can benefit by (1) clearly identifying the reasons they want to quit (i.e., health, cost of cigarettes, etc.); (2) anticipating potential barriers to or problems with quitting and how to manage them; (3) setting a firm quit date and on that date removing all cigarettes and ashtrays from the home or office. In addition, any friends or family members who smoke should be asked not to offer cigarettes. Persistence in trying to quit almost always works. Smoking a cigarette in the course of trying to quit should not become the end of the smoking-cessation effort. Most smokers try to quit several times before they are successful. Many aids are available to tobacco users who quit on their own. Smoking-cessation program guides and motivational and educational tapes—audiotapes and videotapes—may be obtained from physicians, hospitals, or organizations such as the American Lung Association, the American Cancer Society, or the American Heart Association, or they may be found in bookstores and libraries.

ASSISTED QUITTING

Smoking-Cessation Programs. These programs are available to help smokers in most communities. They usually involve attending meetings made up of small groups of quitting smokers who discuss their reasons for not smoking, their problems with quitting, and how they manage these problems. Participants in the programs can pick up practical skills in managing their smoking-cessation attempts and also obtain social support for their efforts. The cessation programs are offered by public-health organizations such as the American Lung Association and the American Cancer Society, and also by private companies such as Smokestoppers and Smokenders.

Physician- and Clinic-Assisted Quitting. Many physicians' offices and some hospital clinics offer assistance in smoking cessation. The clinics are particularly useful for people who have medical problems that need to be treated at the same time, for people who have tried before and failed to quit, or for people who may benefit from taking nicotine-replacement medications. Smokers can turn to these health-care facilities for advice on how to quit and for self-help material as well as for support and information during the different stages of quitting.

Pharmacotherapies for Tobacco Dependence. Medications for tobacco dependence are categorized as first-line or second-line depending on the level of evidence supporting their efficacy. First-line medications include the nicotine replacement systems, i.e., nicotine chewing gum, nicotine patch, nicotine nasal spray, and nicotine inhaler, and bupropion. Second-line medications include nortriptyline and clonidine, and combination nicotine replacement therapy.

Nicotine replacement treatments. Recent research has shown that nicotine replacement increases by about twofold the likelihood of a person successfully quitting smoking. Nicotine-replacement therapy can reduce the severity of nicotine withdrawal. Some tobacco users are concerned about the hazards of taking in nicotine, but the hazards of nicotine-replacement therapy are much less than those associated with smoking. In the first place, the amount of nicotine ingested in replacement therapies is less than that taken in from cigarettes. In the second place, nicotine-replacement medications do not expose smokers to the other hazards of cigarette smoke which include carbon monoxide, tar, cyanide, and a number of other toxic substances. On balance, using the nicotine replacement systems is much safer than smoking cigarettes.

The nicotine-replacement medications are particularly useful with more seriously addicted

smokers, but they are not a simple cure; rather, they must be used as part of a program of learning to live a tobacco-free lifestyle. Currently, four nicotine-replacement products are marketed in the United States: nicotine chewing gum (also called Nicorette), nicotine patches (also called transdermal Nicotine Delivery Systems), nicotine nasal spray, and nicotine inhaler.

Nicotine Chewing Gum. Nicotine chewing gum contains nicotine (bound to a resin, a chemical substance that binds other chemicals) and sodium bicarbonate. The sodium bicarbonate is necessary for keeping the saliva at an alkaline (basic) pH, which in turn is necessary for allowing nicotine to cross the lining of the mouth. The gum is available in strengths of 2 and 4 milligrams (mg), although the dose actually delivered to the chewer is 1 mg and 2 mg, respectively. Nicotine is absorbed from the gum gradually over 20 to 30 minutes, in the course of which nicotine levels similar to those seen after smoking a cigarette are produced in the blood. The gum is meant to be chewed intermittently, to allow time for the nicotine in the saliva to be absorbed. One should not chew the gum while drinking coffee, fruit juice, or cola drinks, because these beverages, by making the mouth more acidic, reduce the absorption of nicotine from the gum. Smokers are instructed to quit smoking and then to chew the gum regularly throughout the day, and also whenever they have the urge to smoke a cigarette. For maximum efficacy, nicotine gum should not be chewed within 10 minutes of drinking any beverage. Most people need to chew 8 to 10 pieces per day to obtain optimal benefits. Usually they chew the gum for 3 to 6 months but need to chew fewer pieces during the last couple of months. Side effects from chewing nicotine gum may include fatigue and soreness of the jaw, loosening of dental fillings, and occasionally nausea, indigestion, gas, or hiccups, particularly if one has chewed the gum so rapidly as to swallow nicotine-rich saliva.

Nicotine Patches. To make it easier to stop smoking, researchers developed patches that administer nicotine without the side effects of nicotine chewing gum. Patches deliver nicotine in its un-ionized (uncharged) chemical form, thereby allowing the drug to pass through the skin readily. Various patches deliver different doses and are applied to the skin once a day, for times that range from sixteen to twenty-four hours. Four patches were available as of 1994 in the United States: Habitrol (Ciba-Geigy), Nicoderm (Marion-Merrell Dow), Nicotrol (McNeil), and Prostep (Lederle). All of these are available as over-the-counter medications. The patches deliver nicotine doses that are equivalent to smoking fifteen to twenty cigarettes (one pack) per day. Higher-dose patches are used during the initial three months of quitting, and lower-dose patches are available for subsequent tapering. Smokers who want to quit are instructed to first stop smoking and then to apply the patch daily. The usually minor side effects from nicotine patches may include itching or burning over the patch site, which usually subsides within an hour, and local redness and mild swelling. Some people experience a sense of stimulation and, occasionally, insomnia; with sleep may come vivid dreams. These effects tend to occur during the first few days of patch use but not thereafter.

Nicotine Nasal Spray. The nicotine nasal spray was designed as a more rapid means of delivering nicotine to the smoker than the gum or the patch. The nasal spray consists of a small bottle containing a 10-mg/ml nicotine solution. A 50-milliliter spray containing 0.5 mg nicotine can be conveniently delivered using an accompanying manual pump. Each dose consists of two squirts, one to each nostril. This mechanism can deliver nicotine to the brain within 10 minutes, providing the most rapid nicotine delivery among the currently available nicotine replacement delivery systems. Patients are advised to use one or two doses per hour and may increase as needed. The minimum treatment is 8 doses per day, with a maximum limit of 40 doses per day (5 doses per hour). The side effects associated with the nasal spray are nasal irritation and throat irritation, sneezing, coughing, and teary eyes. These symptoms often occur during the first week of use but typically decline with continued use.

Nicotine Inhaler. The nicotine inhaler consists of a plastic tube-like mouthpiece into which is placed a cartridge containing a nicotine-impregnated plug. Nicotine vapor is produced when warm inhaled air passes through the plug and nicotine is delivered through the buccal mucosa. The inhaler produces a rate of nicotine delivery similar to the nicotine gum. Dose is related to temperature, consequently, low temperatures will inhibit the release of nicotine. Clinical trials of the nicotine inhaler have shown that it produces double quit rates compared with placebo, similar to the effects observed with the

three other nicotine replacement systems. Side effects from the inhaler include mild mouth and throat irritation, coughing, and runny nose. The frequency and severity of these symptoms decline with continued use of the inhaler.

Bupropion. Bupropion sustained release (SR) is a non-nicotine medication that ranks as a first-line form of treatment. It is available by prescription only. Bupropion was originally marketed as an antidepressant, Wellbutrin. On the strength of evidence from several placebo-controlled trials, the FDA approved the marketing of bupropion (SR), under the trade name Zyban, as a treatment aid for smoking cessation. The mechanism by which bupropion assists smokers is not clear but it is thought to be related to both noradrenergic and dopaminergic activity. Patients are advised to begin using bupropion with a dose of 150 mg per day for three days, then to increase to 150 mg twice a day for one to two weeks prior to a selected day, with continued treatment for up to seven to twelve weeks following the quit date. Bupropion has been shown to reduce withdrawal symptoms and to reduce the weight gain usually associated with stopping smoking. The most common side effects reported by bupropion users have been insomnia and dry mouth. Bupropion is contraindicated in persons with a history of seizures, or of eating disorders, and those who have used a monoamine oxidase inhibitor in the past 14 days.

Clonidine. Clonidine is an alpha2-noradrenergic agonist that was initially used for the treatment of hypertension, and subsequently found to diminish symptoms of both opiate and alcohol withdrawal. The efficacy of clonidine as a short-term smoking cessation aid was demonstrated in several studies in which clonidine was delivered either orally or in patch form. This drug has not received FDA approval as a smoking cessation aid, however, and should be considered a second-line treatment when first-line pharmacotherapies have not been successful. Clonidine use is associated with reductions in pulse rate and blood pressure, and abrupt discontinuation could result in a rapid rise in blood pressure and catecholamine levels. Side effects reported with clonidine use include dry mouth, drowsiness, dizziness, and sedation. Appropriate dose levels have not been established.

Nortriptyline. Nortriptyline is used primarily as an antidepressant (Pamelor) and has not been evaluated or approved by the FDA for the treatment of tobacco dependence. Increased abstinence rates with notriptyline use, compared with placebo, were observed in two controlled trials. In those smoking cessation trials, nortriptyline use was initiated at a dose of 25 mg/day, and increased gradually to 75 to 100 mg per day over 12 weeks. Sedation, dry mouth, blurred vision, urinary retention, lightheadedness, and shaky hands are the most commonly reported side effects of nortriptyline use. Nortriptyline may also cause cardiovascular changes. This side effect profile and the need for evidence from more controlled studies consigns nortriptyline to the status of a second-line smoking cessation aid at the present time.

Other treatments. A number of other treatments are available or have been used in the past to aid in smoking cessation. Although the effectiveness of these treatments has not been established by medical research, some individuals may benefit from them. None of these treatments, however, can magically cure smokers of their tobacco addiction without the commitment and effort that are usually required to quit.

Hypnosis has been widely used to increase a smoker's motivation or commitment to stop. While under hypnosis, the smoker receives suggestions, such as "smoking is a poison to your body," "you need your body to live," "you owe your body respect and protection." This treatment probably works best in combination with the previously discussed behavioral modification programs.

Acupuncture as a smoking-cessation technique involves the placement of needles or staples in various parts of the body, most commonly the ears. Although acupuncture may be helpful for some smokers, a meta-analysis did not support the efficacy of this form of treatment.

Lobeline and silver acetate medications have been available in pharmacies without a physician's prescription. Lobeline, a chemical similar to nicotine but with less psychoactivity, has been recently removed from the market by the Food and Drug Administration. Lobeline has been available in prescriptions such as CigArrest, Bantron, and Nikoban. Silver acetate, available in a chewing gum, mouthwash, mouth spray and lozenges, acts as a deterrent. Tobacco smoke combines with the silver in the mouth to precipitate silver sulfide, which has an unpleasant taste. The unpleasant taste presumably decreases the incidence of smoking.

TREATMENT OF SMOKELESS TOBACCO ADDICTION

Much evidence indicates that the use of smokeless tobacco produces addiction and leads to serious health consequences as does the use of smoked tobacco. However, little is known about effective treatment for smokeless tobacco (i.e., snuff or chewing tobacco) addiction. The general behavioral approach is similar to that for cigarette smoking, although the specific learned associations and cues are naturally somewhat different. Self-help materials are available from a variety of sources in the United States. Some strategies include the use of alternative activities, such as chewing gum, hard candy, sunflower seeds, nuts, toothpicks, or beef jerky. Formal treatment programs are also available in some parts of the country. At the present time, insufficient evidence exists to suggest that the use of established medications designed for helping cigarette smokers increases long-term cessation among users of smokeless tobacco.

(SEE ALSO: *Addiction: Concepts and Definitions; Relapse Prevention; Tobacco Smoking Cessation and Weight Gain; Treatment*)

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TOBACCO: HISTORY OF

Tobacco generally refers to the leaves and other parts of certain South American plants that were domesticated and used by Native Americans for the alkaloid NICOTINE. Tobacco plants are a species of the genus *Nicotiana*, belonging to the Solanaceae (nightshade) family; this also includes potatoes, tomatoes, eggplants, belladonna, and petunias. Including plants used for tobacco, there are sixty-four *Nicotiana* species. The two widely cultivated for use as tobacco are *Nicotiana tabacum* and *Nicotiana rustica*, the latter of which contains the higher levels of nicotine.

Three hundred tobacco farmhands pose during picking season in Granby, Connecticut, circa 1903. (© CORBIS)

Nicotiana tabacum is, however, the major source of commercial tobacco, although it has been hybridized with other *Nicotiana* species, with resultant alteration in chemical composition. *Nicotiana tabacum* is a broad-leaf plant that grows from 3 to 10 feet (1–3 m) tall and produces 10 to 20 leaves radiating from a central stalk. *Nicotiana rustica*, also known as Indian tobacco, was first cultivated by Native Americans and was probably the tobacco offered to Columbus. The word *tobacco* comes into English (c. 1565) from the Spanish word *tabaco*, probably from the Taino word for the roll of leaves containing the *N. rustica* that the American natives of the Antilles smoked.

HISTORY OF TOBACCO USE

Tobacco was introduced to Europeans by Native Americans at the time of Columbus's exploration of the New World (1492–1506). The first written records of tobacco use date from this time, but there is archaeological evidence for tobacco's wide use in the Americas as early as C.E. 600–900. Native Americans considered tobacco as sacred, a plant used in social, fertility, and spiritual ritual. For

example, tobacco was used for seasonal ceremonies, for sealing friendships, preparing for war, predicting good weather or good fishing, planting, courting, consulting spirits, and preparing magical cures. The desired effects of tobacco were a trance state, achieved by using the leaves in various ways, including smoking, chewing, snuffing, drinking (tobacco juice or tea), licking, and administering enemas.

Acute nicotine poisoning was a central aspect of the practice of shamanism in many parts of South America. South American shamans would smoke or ingest tobacco to the point of producing a nicotine-mediated trance or coma. The dose of nicotine could be titrated to produce a coma state resembling death, but from which the shaman would recover. Recovery from apparent death enhanced the perception of the shaman's magical powers.

In 1492 Columbus encountered natives in Hispaniola smoking tobacco in the form of large cigars. Enticed by the sacred and special regard in which they held tobacco, Columbus's crew experimented with tobacco smoking and soon became enthusiasts. Tobacco was brought back to Europe and, within a few decades, its use spread. People smoked it in the form of cigars and pipe and used it as snuff or chewing tobacco. Within forty years of Columbus's arrival, Spaniards were cultivating tobacco in the West Indies. Tobacco use then became widespread in Europe and in Spain and Portugal's American colonies by the late 1500s.

In 1570 the tobacco plant had been named *nicotiana* after Jean Nicot, the French ambassador to Portugal who introduced tobacco to France for medicinal use. Tobacco was said to be useful in the prevention of plague and as a cure for headache, asthma, gout, ulcers, scabies, labor pains, and even cancer. In the late 1500s, Sir Walter Raleigh popularized the smoking of tobacco for "pleasure" in the court of Queen Elizabeth (reigned 1558–1603); from there it spread to other parts of England.

James I of England (reigned 1603–1625), who succeeded Queen Elizabeth, was strongly opposed to tobacco use and wrote the first major antitobacco treatise, entitled "Counterblast to Tobacco," in 1604. King James described tobacco as "a custome loathsome to the eye, hateful to the nose, harmful to the brain, dangerous to the lungs, and in the black stinking fume thereof nearest resembling the horrible stygian smoke of the pit that is bottomless." Despite James's opposition, however, tobacco use flourished. Eventually, even James lessened his opposition to tobacco because of the lucrative income from its taxation.

During the 1600s, tobacco use had spread throughout Europe, Russia, China, Japan, and the west coast of Africa. Over the centuries, draconian penalties for tobacco use were occasionally promulgated. For example, Murad the Cruel of Turkey (1623–1640) ordered that tobacco users be beheaded, quartered, and/or hanged. Nevertheless, smoking persisted. In the American colonies, tobacco became the most important export crop and was instrumental in the economic survival of the colonies.

By the nineteenth century, tobacco production was a mainstay of American capitalism. Most tobacco was smoked as cigars or in pipes, or used as snuff. Cigarettes were hand rolled. A skillful worker could roll four cigarettes per minute. Cigarette smokers were primarily boys or women, and smoking was a behavior confined to the lower socioeconomic class. The invention of the cigarette rolling machine by James Bonsack in 1881 made tobacco use inexpensive and convenient. Bonsack went into business with W. B. Duke and Sons in Durham, North Carolina. Together they improved the machine; by April 30, 1884, the device could roll 120,000 cigarettes per day.

Just as cigarettes were becoming widely available and affordable, tobacco manufacturers strongly promoted their use. Massive advertising campaigns, government issue of cigarettes to soldiers during the world wars, glamorization of cigarettes in motion pictures, and the gradual incorporation of women into the smoking market increased the popularity of cigarette smoking in the United States and around the world. Smoking rates peaked in the United States for men in 1955, with 50 percent of men smoking, and in 1966 for women, with 32 percent of women smoking. As a result of clever marketing by the cigarette companies, smoking at that time was considered to be sophisticated, glamorous, individualistic, and even healthful.

While there had been occasional reports on the health hazards of cigarette smoking from the time of King James, the first large-scale studies documenting the link between cigarette smoking and cancer appeared in 1952 (Doll & Hill) and 1956 (Wynder et al.). Subsequently, hundreds of studies have shown that cigarette smoking accounts for 30

percent of cancers—including some cancers of the lung, mouth, throat, esophagus, bladder, and kidney, as well as some leukemia; and that it is the cause of some heart and vascular disease, stroke, emphysema, chronic obstructive lung disease, and other health problems. In 1962 the Royal College of Physicians in the United Kingdom, and in 1964 the U.S. surgeon general, issued reports on smoking and health, indicating that cigarette smoking most probably caused some lung cancers and other health problems. These reports mark the beginning of modern public-health efforts to control tobacco use.

Subsequent landmarks in tobacco control in the U.S. include the following:

- 1965—Federal Cigarette Labeling and Advertising Act (PL89-92) required health warnings on cigarette packages and an annual report to Congress on the health consequences of smoking.
- 1969—Public Health Cigarette Smoking Act (PL91-222) strengthened health warnings on cigarette packs and prohibited cigarette advertising on television and radio.
- 1973—Little Cigar Act (PL93-109) extended the broadcast ban on cigarette advertising to little cigars.
- 1984—Comprehensive Smoking Education Act (PL98-474) required rotation of four specific health warnings and mandated that the cigarette industry provide a list of cigarette additives.
- 1986—Comprehensive Smokeless Tobacco Health Education Act (PL99-252) required three rotating health warnings on SMOKELESS TOBACCO packages and advertisements, a list of additives and nicotine content in smokeless tobacco products, prohibited smokeless tobacco advertising on television and radio, and mandated reports to Congress on smokeless tobacco and a public information campaign on the health hazards of smokeless tobacco.

The four warnings currently rotated among cigarette packs are the following:

1. Surgeon General's Warning: Smoking Causes Lung Cancer, Heart Disease, Emphysema, and May Complicate Pregnancy
2. Surgeon General's Warning: Quitting Smoking Now Greatly Reduces Serious Risks to Your Health
3. Surgeon General's Warning: Smoking by Pregnant Women May Result in Fetal Injury, Premature Birth, and Low Birth-Weight
4. Surgeon General's Warning: Cigarette Smoke Contains Carbon Monoxide.

The three smokeless tobacco warnings that are rotated are these:

1. Warning: This Product May Cause Mouth Cancer
2. Warning: This Product May Cause Gum Disease and Tooth Loss
3. Warning: This Product Is Not a Safe Alternative to Cigarettes

As a consequence of education and other public health activities, tobacco use has declined in the United States. In the late 1900s, 25 percent of Americans, about 43 million people, smoke. About 45 million former smokers have quit. Unfortunately, adult smoking rates have been declining very slowly in recent years because adolescents are taking up smoking at undiminishing rates and grow up to become addicted adult smokers.

(SEE ALSO: *Advertising and Tobacco Use; Nicotine Delivery Systems for Smoking Cessation*)

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TOBACCO: INDUSTRY The tobacco industry is made up of the complex of primary suppliers, manufacturers, distributors (both wholesale and retail), advertising agencies, and media outlets that produce, promote, and sell tobacco products, as well as the law, public relations, and lobbying firms that work to protect these products from stringent public-health regulation and control. In due time, however, these precautions failed. The industry evolved in the late nineteenth and early twentieth century from many, relatively small enterprises that produced tobacco products for puffing, snuffing, and chewing. The products of these small firms delivered nicotine to the nasal and oral mucosa. With the evolution and refinement of the cigarette, the industry developed first into a

monopoly and then into an oligopoly in which a handful of major producers made this more sophisticated nicotine delivery system: a device that delivers nicotine by inhalation to the lungs and thence rapidly to the brain. Although its popularity is declining in the United States, cigarette use is increasing worldwide at over 2 percent per year, especially in much of Asia, Eastern Europe, and the former Soviet Union. An integrated system of suppliers, manufacturers, marketers, and sales outlets is constantly evolving to supply this vast and growing market. In the past, sophisticated legal and lobbying enterprises managed to protect this industry from the sort of regulation advocated by a number of public health groups—regulations that governments routinely impose on far less toxic products, but an admonition from an internal source as to the effects of tobacco led to a dramatic increase of public and regulatory pressure on the tobacco industry.

PRIVATE ENTERPRISE VERSUS STATE MONOPOLY

Tobacco (nicotiana) is a plant of the nightshade family (genus *Nicotiana*) and is native to the Americas; it was a major commodity of commerce in colonial times. Cigar tobaccos were key exports from the Spanish and Portuguese colonies of the Caribbean and South America, while tobaccos for snuff, pipe, and chew were the economic mainstays of the English colonies in Virginia, Maryland, and the Carolinas. Whereas most of Europe (and the rest of the world) established state-run monopolies for tobacco distribution, private enterprise was the vehicle of tobacco commerce in Great Britain (and eventually in the United States). The state monopolies provided both a popular product for the populace and revenue for the national treasury—but private enterprise, which always paid excise tax in Great Britain, was more resourceful in expanding the market. This phenomenon was exploited in the twentieth century and was especially apparent in the 1990s, with the remaining state monopolies becoming privatized and adopting the marketing techniques of the by-now enormous transnational tobacco companies, often actually merging with them.

FROM COTTAGE INDUSTRY TO MONOPOLY TO OLIGOPOLY

Relatively expensive, hand-rolled cigarettes became popular novelties in the United States and Europe in the mid-nineteenth century. The novelty came to dominate the industry over a period of forty years, from the mid-1880s to the mid-1920s, when, for the first time, more tobacco in the United States was used for cigarettes than for chewing tobacco.

A number of changes in the nineteenth century laid the groundwork for the cigarette's commercial success. The development of flue-cured tobacco and air-dried burley tobacco—easily processed into tobaccos for smoking (where the smoke might be inhaled) were major factors (Slade, 1993). Cigarette-making machines—first used commercially in 1883 by the American Tobacco Company—the development of safe matches, and an extensive railroad network to transport centrally manufactured cigarettes throughout the United States were among the other key factors responsible for this product's success.

Duke of Durham, North Carolina. These elements were successfully harnessed by Benjamin Newton (Buck) Duke, head of the American Tobacco Company. A working cigarette-making machine had been invented in 1881 by James Bonsack in response to a contest held by the cigarette maker Alan & Ginter of Richmond, Virginia (Smith, 1990). But the contest sponsors decided against using the invention since they did not know how to sell as many cigarettes as the machine was capable of making. Duke, however, realized that the low prices made possible by mass production, together with advertising to stimulate demand, would create a large enough market to absorb the vastly expanded production. He obtained favorable terms for using the machine in exchange for technical assistance in perfecting it. The machine Duke put on line in 1883 produced 120,000 cigarettes per day, the equivalent of 60 expert hand rollers. Duke's competitors had to pay more for Bonsack machines than he had, and Duke engaged in price wars to further weaken other manufacturers. Gradually, he bought out his competitors and monopolized the U.S. cigarette industry. By 1890, Duke controlled the cigarette market, and by 1910, just before his monopoly was broken, he controlled more than 80 percent of all tobacco products man-

ufactured in the United States, except for cigars (Robert, 1952).

Seeking further growth, Duke began to expand his cigarette business overseas (Robert, 1952). By 1900, a third of America's domestic production was being sent to Asia, and company factories were operating in Canada, Australia, Germany, and Japan. In 1901, Duke purchased a cigarette factory in Liverpool, England. Alarmed British manufacturers, seeking to avoid the fate of their U.S. compatriots, banded together as the Imperial Tobacco Company. The resulting trade war between American and Imperial ended in a truce. American was given exclusive trading rights in the United States and Cuba, and Great Britain became Imperial's exclusive territory. A new company, jointly controlled by both giants, was to sell cigarettes to the rest of the world. This modest sinecure was the birthright the parent companies gave the British-American Tobacco Company (BAT).

Antitrust Litigation. In 1907, the U.S. government filed an antitrust case against the American Tobacco Company. The result of this litigation was the dissolution of the trust four years later into a number of successor companies, some of which retain major roles in the U.S. cigarette market. These companies were the American Tobacco Company, the R.J. Reynolds Tobacco Company, Liggett & Myers, and P. Lorillard.

Once it had emerged from the confines of the trust, R.J. Reynolds, which had never before made cigarettes, developed and introduced Camel, a novel brand, in 1913 (Tilley, 1985). Camel was the first brand to combine air-dried burley, which had previously been important in chewing-tobacco products, with the then-conventional cigarette tobaccos—the flue-cured and Turkish (Oriental) varieties (Slade, 1993). Camel featured a coherent, national advertising campaign from N.W. Ayer that relied entirely on mass-media outlets in magazines and on billboards instead of on package-based promotions such as cigarette cards, coupons, and premiums. The legacy of this startling departure from the conventional cigarette-marketing techniques of the time is captured by the sly legend that still graces each pack of twenty unfiltered Camels sold in the United States: "Don't look for premiums or coupons, as the cost of the tobaccos blended in CAMEL Cigarettes prohibits the use of them."

The other thing that distinguished Camel from its competitors was its price. While the leading brands of the time, such as Fatima, sold for fifteen cents per pack of twenty, a pack of Camel sold for a dime. In short order, Camel overwhelmed the competition and ushered in a dramatic expansion of the domestic cigarette market. American Tobacco copied the Camel formula with Lucky Strike, and Liggett & Myers followed with its copycat product Chesterfield. Cigarette cards, premiums, and coupons were abandoned in favor of the mass media, and prices fell. Cigarette use, then only rising slowly, began an unprecedented increase. This growth continued virtually unabated for forty years or so, until finally slowed and eventually reversed by alarms that lung cancer and other major diseases could be caused by cigarettes (Fiore et al., 1993).

Only two firms that had no roots in the tobacco trust have played major roles in the U.S. cigarette market (Sobel, 1978). After Buck Duke's death in 1929, BAT purchased the Brown & Williamson Tobacco Company in Louisville, Kentucky. BAT gradually built this company into a major cigarette producer. For decades, its Kool brand dominated the menthol category, and during the 1930s and 1940s, its Wings brand gained market share by undercutting the prices of the majors. Brown & Williamson continues to offer a full range of cigarettes for the U.S. market. It also produces cigarettes for export to many of BAT's international markets.

The other upstart company was Philip Morris, which began its U.S. operations as a specialty cigarette maker in New York in the first quarter of the century. In addition to its standard brand called Philip Morris, it produced Marlboro—a cigarette for "ladies." The company expanded in the 1930s with a low-priced brand (Paul Jones) and a clever pricing scheme for Philip Morris English Blend (Robert, 1952; Sobel, 1978). It suggested a retail price for the latter slightly above that for the major brands, but it gave retailers a larger margin, thus encouraging prominent display of the brand in stores. In the mid-1950s, Philip Morris gave Marlboro a filter and had the Leo Burnett advertising agency remake its image entirely to one of rugged masculine outdoor daring on horseback. (The entire sweep of Marlboro advertising is included in the special advertising collection of the American Museum of National History in Washington, D.C.) By the mid-1970s, Marlboro was the leading U.S. cigarette and by the 1990s, thanks to the strength

of Marlboro's appeal to teens and young adults, Philip Morris overtook R.J. Reynolds to become the nation's largest tobacco-product manufacturer.

Smokeless Tobacco. Moist snuff and chewing tobacco enjoyed a 1980s and 1990s resurgence in popularity—this is based on the successful efforts of U.S. Tobacco (UST). It sells oral tobacco (e.g., Skoal Bandits, Skoal, Copenhagen) to adolescents and preadolescents (Denny, 1993). Oral tobacco is the only category of tobacco product whose consumption has increased in recent years in the United States. This increase is attributable to UST's innovative marketing of moist snuff to adolescent boys, and to imitation products from other manufacturers. Although UST envisions a global market for snuff, the World Health Organization has declared that countries in which oral tobacco is not a traditional product should ban it. A number of countries—including Australia, New Zealand, Hong Kong, and the European Community—have taken this step, often defying intense pressure from the U.S. government when doing so.

Table 1 lists the major tobacco-product manufacturers in the United States, the location of their corporate headquarters, and the major tobacco brands they market.

INNOVATION

The tobacco industry adapts to changing circumstances in many ways. Product innovation is a key strategy. Since the early 1950s, the major changes in cigarette design have come in response to public-health concerns that cigarettes constitute a leading cause of illness and death (McGinnis, 1993; Slade, 1993). Most of these innovations have been variations on filters and so-called low-tar designs. Ballyhooed with multibillion-dollar advertising budgets, these innovations propped up cigarette consumption over the years despite the complete absence of demonstrated benefit at the time they were introduced. Years of study (and as many years of unregulated sale) have only produced evidence for decidedly marginal benefits, yet the innovations have become firmly established. These supposed

TABLE 1
Leading U.S. Tobacco Companies, 1992

Company	*Home Office*	*Major Brands*
CIGARETTES:		
Philip Morris	New York	Marlboro Basic Benson & Hedges Merit Virginia Slims
RJR/Nabisco	New York	Winston Camel Salem
American Brands	New York	Carlton Lucky Strike
British American Tobacco (BAT)	London	Kool
Loews Corporation	New York	Newport Kent
Brooke Group	Miami	Generics
MOIST SNUFF:		
UST	Greenwich, CT	Copenhagen Skoal Bandits Skoal Classic

advances have been criticized by some as being nothing more than public relations gimmicks in the face of and in mocking response to profound public-health problems.

The cigarette companies continue to invent novel ways to deliver nicotine to the brain. Electronic devices, smokes with charcoal fuel elements, and tiny aerosol cans are but some of the gimmicks the companies have patented to facilitate the inhalation of nicotine. Despite these efforts, the industry remains dependent on smoking, with variations of the tobacco-filled cigarette the mainstay of its business for the foreseeable future.

INTERNATIONAL EXPANSION

Cigarette smoking has been declining in the United States, Canada, and Western Europe. Since the 1960s, however, the biggest cigarette manufacturers (BAT, Philip Morris, RJR/Nabisco, and, recently, Japan Tobacco Incorporated) have steadily increased their business in international markets (Taylor, 1984). This expansion has been accompanied by the weakening and dissolution of both national private and state-owned tobacco companies. The process got under way in Latin America in the 1960s, spread to eastern Asia in the late 1980s, and developed into a frenzy of deal making in Eastern Europe and the republics of the former Soviet Union in the early 1990s (Shepherd, 1985; Sesser, 1993).

Shepherd has described the process whereby a transnational corporation moves toward dominating a formerly self-contained market through product innovation, smuggling, aggressive advertising, and pricing policies. The result is a larger market for tobacco products than existed previously and a corporate management that is better able to oppose public-health efforts at regulation and control. Although cigarette consumption is down in the United States, Canada, and Western Europe, it is rapidly growing in most of the world—especially the so-called third world. The transnational companies have positioned themselves to both fuel and profit from this trend.

DIVERSIFICATION

The giant cigarette makers have invested their tobacco profits in other enterprises for more than twenty years, ranging from soft drinks and cookies to office products, insurance, and real estate. This process has resulted in the ownership by tobacco companies of some widely known consumer-product companies, including Kraft and Nabisco. Although the parent tobacco companies pretend that this phenomenon makes them somehow less involved in tobacco (none now have the word "tobacco" in their corporate name), a thoughtful examination of these businesses reveals the following:

> Tobacco products remain by far the most profitable sector of each of these conglomerates; and tobacco products are always responsible for most of the company profits (see Tables 2 and 3).

TABLE 2
1992 Overall Earnings for Six U.S.-Based Tobacco-Product Manufacturers (in Millions of U.S. Dollars)

Company	*Tobacco Revenues*	*Nontobacco Revenues*	*Tobacco as % of Revenue*	*Tobacco Income*	*Nontobacco Income (or Loss)*	*Tobacco as % of Income*
Philip Morris	25,677	33,454	43	7,203	3,757	66
RJR/Nabisco	9,027	6,707	57	2,687	947	74
American Brands	8,157	6,467	56	1,091	757	59
Loews Corp. (Lorillard)	2,185	11,506	16	915	(1,217)	233
Brooke Group (Liggett & Myers	606	114	84	53	(59)	211
UST	884	163	84	509	14	97
Totals:	46,536	58,411	44	12,458	4,199	75

SOURCES: Corporate annual reports.

TABLE 3
Profitability of Selling Tobacco Products Compared to Selling Other Goods and Services, 1992

Company	*Gross Profit Margin on Tobacco Product Sales*	*Gross Profit (or Loss) Margin on Sales Other than Tobacco*
Philip Morris	28%	11%
RJR/Nabisco	30	14
American Brands	13	12
Loews Corp (Lorillard)	42	(11)
Brooke Group (Liggett & Myers)	9	(52)
UST	58	9
Overall:	27	7

SOURCES: Corporate annual reports.

Not one of these companies has backed away from any available opportunity to sell tobacco products. Indeed, the strongest companies continue to invest in domestic and overseas ventures that have as their goal the expansion of tobacco consumption.

These companies make ready use of nontobacco subsidiaries to support their tobacco businesses. For example, RJR/Nabisco fired the ad agency that did their Oreo Cookie advertising after that agency also produced ads promoting an airline offering smoke-free flights. Philip Morris has used one of its Kraft-General Foods warehouses for its coupon-redemption program for the Marlboro Adventure Team.

Tobacco companies do not diversify to get out of the tobacco business. They diversify because tobacco has given them profits, the acquisitions seem sound investments, and the resulting product mix complements the core business in some manner.

PRICE WARS

Price competition has long been part of the tobacco industry strategy. It was the major tool for the achievement of monopoly power in the 1880s and was a key element in the early twentieth-century dominance of the market by Camel. In the 1930s, price competition, made possible by overly aggressive price increases by the majors, contributed to the emergence and growth of Brown & Williamson and Philip Morris (Sobel, 1978). From the end of World War II (1945) until 1980, however, price competition was virtually absent from the U.S. cigarette market.

In 1980, tiny Liggett & Myers, a firm that had become too small to enjoy oligopolistic profits, broke ranks with its fellows by introducing generic cigarettes. The strategy was made possible by the pattern of price increases in the industry—increases that had exceeded the rate of inflation for years. Brown & Williamson soon followed suit with its own generic brands, and within a few years every cigarette manufacturer had a multitiered pricing structure, with the heavily advertised, standard brands at the top. Prices for the major brands continued to rise steeply, far faster than inflation, through early 1993. Customers who might have stopped smoking because of high prices were kept in the market by the increasingly available lower priced offerings. By early 1993, however, investment analysts had become concerned because lower priced brands accounted for more than 25 percent of all cigarette purchases—with attendant threats to profits—and Philip Morris had become alarmed by the market share losses sustained by its

cash cow, Marlboro, to less than 25 percent of all cigarettes sold.

Philip Morris had a number of key strengths that gave it a flexibility not possessed by its competitors, including market leadership, an absence of corporate debt, and a strong youth market for Marlboro. Its principal competitor, RJR/Nabisco, had an enormous corporate debt—and although Camel had been making inroads into Marlboro's youth market, it was still far from the dominant cigarette. These factors led Philip Morris to cut prices substantially (while mounting the most elaborate promotional campaign ever seen in the industry). The competition was forced to follow suit with lower prices. Marlboro's brand share surged; the threat to profitability from lower priced brands subsided; and the competition was left somewhat weakened.

LOBBYING AND PUBLIC RELATIONS

In 1915, the U.S. tobacco industry formed the Tobacco Merchants Association (TMA) to lobby against the anticigarette laws that had become a problem for the industry in a number of states (Robert, 1952). These laws came about as a result of the efforts of antitobacco advocates, including Henry Ford and Thomas Edison. The TMA accomplished its objectives: By 1930, the state prohibitions on cigarettes had been diminished to easily ignored prohibitions that only barred the sale of cigarettes to minors.

In the 1950s, the industry faced a more substantial challenge—proof that cigarettes caused lung cancer. In addition to putting cosmetic filters on the product and making outrageous claims for their benefit (P. Lorillard trumpeted its asbestos-filtered Kent as "the greatest health protection in cigarette history"), the industry developed a sophisticated public relations and lobbying capability (Wagner, 1971). The public relations firm of Hill & Knowlton organized the Tobacco Institute to meet the industry's public relations and lobbying needs. The cigarette makers also formed the Tobacco Industry Research Committee (later reorganized and renamed the Council for Tobacco Research) to create the pretense that the industry was conscientiously involved in biomedical research to get to the bottom of the smoking and health question (Freedman & Cohen, 1993).

Although speculation existed as to how diligently the tobacco industry would pursue smoking research, they did in fact do so, but their conclusions, giving more light to the fact that tobacco is addictive and harmful, were not released. Routinely called the "tobacco cover-up" it resurfaced in later years with much of its strength coming from Bennett S. LeBow's agreeing, in 1997, to put warnings on cigarette packs stating that smoking is addictive. Leaked internal documents also served as evidence of the dangers. In 1998, however, other tobacco companies still contested that tobacco was not an addictive drug. Discovery, through LeBow, of the industry's nondisclosure and the understanding that the industry had evidence of the thereat of smoking, however, caused severe public attacks on the tobacco industry to be more common. Public campaigns have also been more potent with reducing youth smoking. Between 1998 and 2000 smoking had declined 54 percent in middle schools and 25.2 percent in high schools. Recently tobacco advertising legislation has weakened the strength of tobacco propaganda among youth populations, by banning all advertising that is determined to be too appealing to a minor. More legislation is in being proposed and being worked on to make nicotine a drug regulated by the FDA. Previously, the FDA has tried to apply regulations to tobacco and cigarettes as a nicotine delivery agent, but the courts had determined that Congress had not yet given the regulatory administration such authority, so new legislation must be passed for successful and lawful regulation. If such a bill is passed tighter control will be possible so that tobacco can be prohibited in public events where minors may be part of the targeted demographic, in response to public outcry. Furthermore, tobacco companies are prohibited from sponsoring public events and athletic competitions. In some states, legislation has also already been passed, and tried, winning large cash settlements to recover lost health costs suspected to be to tobacco use related. Included in some of these settlements have also been requirements for the tobacco companies to pay for more advertisements, but these advertisements are intended to reduce youth smoking. Despite the research, such as it was, the mounting costs to the tobacco companies because of law suits and penalties, and in the face of growing evidence of harm from a variety of other quarters, the smoking epidemic continues.

The Tobacco Institute, in alliance with the various branches of the industry, has stood as a bulwark against public-health activities for a gen-

eration. The Council for Tobacco Research has funded studies of marginal importance for public relations gain while operating a Special Projects branch for the benefit of tobacco-product liability defense. In these and other ways, the tobacco industry has attempted to insulate itself from significant regulation and from acceptance of any responsibility for the harm its products cause. Similar organizations exist to protect the interests of oral-tobacco manufacturers.

OWNERSHIP

The major tobacco-product manufacturers are publicly owned and traded corporations. As such, they are owned by their investors. Major institutions, including banks, insurance companies, and pension funds, hold the majority of shares in the tobacco industry.

SUMMARY AND CONCLUSION

The tobacco industry is a powerful oligopoly of product manufacturers in alliance with a network of suppliers and associated service organizations. Although its products form the leading cause of preventable death, it continues despite public sentiment and attempt to protect itself against appropriate regulation by extensive legal, public relations, and lobbying efforts. The industry is understandably driven by an interest in making money. It has never acted out of a primary concern for the health of its customers or the health of those around them. For a variety of reasons, including clever intervention by the industry, government has utterly failed to provide the sort of regulatory control expected when it comes to something as addicting and toxic as nicotine-containing tobacco products until a critical documentation leak occurred from within the companies of the tobacco industry.

(SEE ALSO: *Advertising and Tobacco Use; Nicotine*)

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TOBACCO: MEDICAL COMPLICATIONS

HISTORY

The notion that smoking tobacco is injurious to the body is not of recent origin. King James I of England, in his classic "*Counterblaste to Tobacco*," written in 1604, outlined a number of beliefs about tobacco's ill effects on health and urged his subjects to avoid it. He called smoking a "filthie noveltie . . . A custome lothsome to the eye, hatefull to the nose, harmefull to the braine, dangerous to the Lungs. . . ." Opinions on the possible benefits and health damage caused by use of tobacco varied over the next 300 years. Some nineteenth-century arguments that tobacco use injured health were linked to moral arguments against its use rather than to what today would be considered medical evidence.

In 1926 Sir Humphrey Rolleston of Cambridge University (the same ROLLESTON who headed the committee on the use of opioids) addressed the Harrogate Medical Society on the subject of medical aspects of tobacco and the possible toxic effects of nicotine. He drew few firm conclusions. Only a few health problems were clearly linked to tobacco. These included some irritation of the throat and upper air passages by furfural, pyridine derivatives, ammonia, and carbon monoxide, which he ascribed to combustion of vegetable material and "not, like NICOTINE, in any way special to tobacco." He did mention tobacco amblyopia, a disorder of the optic nerve leading to blindness, now thought to be a rare complication. Among the heart disorders Rolleston mentioned were extrasystoles (irregular heartbeats) and angina (pain caused by insufficient blood reaching the heart). He noted that nicotine constricted coronary arteries but suggested that people who suffered from extrasystoles might consider giving up coffee and tea before tobacco. He observed that cigarette smoking could cause arterial spasms, noting that it was linked to obliterative diseases of the large arteries among young Jews living in London's East End. Rolleston believed that cancers of the lip and oral cavity observed in smokers were probably caused by syphilis and therefore not firmly linked to smoking. He devoted only a few lines to smoking's adverse effects on the respiratory tract, observing that smoking was responsible for "causing cough, hoarseness, bronchial catarrh, and so emphysema of the lungs." In general, Rolleston observed that considering "the large number of heavy smokers, the comparative rarity of undoubted lesions due to smoking is remarkable." He concluded that "to regard tobacco as a drug of addiction may be all very well in a humorous sense, but it is hardly accurate."

But even as Rolleston was lecturing, researchers were looking at the evidence suggesting that smoking was responsible for the increasing number of lung cancer cases, a rare disease in the nineteenth century. Within thirty years there would be a growing consensus among the medical scientific community that tobacco smoking was the principal cause of lung cancer, causally related to other cancers, and a major contributor to cardiovascular diseases, peripheral artery disease, and chronic obstructive lung disease (emphysema and chronic bronchitis). Yet from the 1920s to the 1960s, cigarette smoking gained almost universal social acceptance. Using doctors and nurses and health-related slogans ("not a cough in a carload") in their advertisements, cigarette manufacturers implied that cigarette smoking was without health risk. By the 1960s the majority of adult males were smokers, with more than 70 percent in some age groups.

The turning point in the public's perception of the adverse consequences of tobacco smoking came with the publication of the *Report of the Royal College of Physicians* in England in 1962 and the *Report of the Surgeon General* in the United States in 1964. These two reports documented the experimental, epidemiological, and pathological evidence linking tobacco smoking to a variety, of diseases, the most notable of which were lung cancer, illness and death from heart disease, and chronic bronchitis and other lung disorders. Many more reports on the health consequences of smoking followed these two pivotal publications. Since 1969 the Office of Smoking and Health of the U.S. Public Health Service has coordinated the annual publication of a Surgeon General's Report on the health consequences of smoking, with several of the reports focusing on specific topics. In approaching such major reviews of specific health consequences of smoking, the Office of Smoking and Health assigns recognized experts to review and summarize all the existing scientific literature on the topic and then draw some conclusions from it. Some of the special topics that have been considered are health conse-

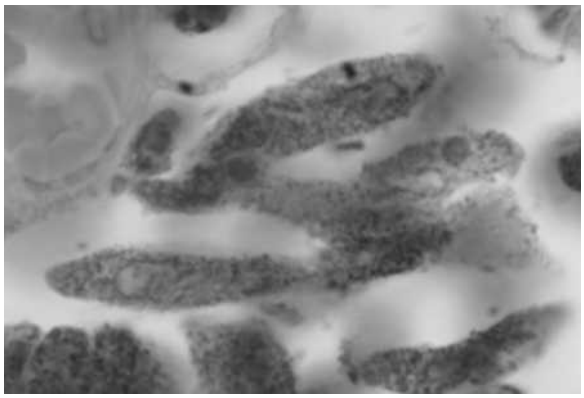
quences of smoking for women (1980), the changing cigarette (the implications for health of low tar/nicotine cigarettes and filters) (1981), chronic obstructive lung disease (1984), cancer and chronic lung disease in the workplace (1985), and nicotine addiction (1988). The 1972 report was the first to explore the health consequences of involuntary smoking (passive or secondhand smoking).

The 1979 and 1989 reports were overall reviews of the field, marking the fifteenth and twenty-fifth anniversaries of the landmark 1969 report produced when Dr. Luther Terry was Surgeon General. The 1979 report described tobacco smoking as "the largest preventable cause of death in America." It noted that statisticians were able to identify the following as deaths related to smoking: 80,000 each year from lung cancer; 22,000 from other cancers; up to 225,000 from cardiovascular disease; and more than 119,000 from chronic pulmonary disease. As of 2000, cigarette smoking remained the most important cause of preventable disease and premature death in the developed countries of the world. It is estimated that, depending on the age at which a person starts to smoke, 7 to 13 years of life are lost to smoking-related diseases. Nonetheless, nearly 47 million Americans continue to smoke.

TOBACCO-RELATED DISEASES

The Pharmacological Actions of Nicotine. Nicotine, the addictive component in tobacco, is a colorless liquid alkaloid that turns brown and begins to smell like tobacco when it is exposed to air. In addition to the psychological and social dimensions of tobacco dependence, nicotine by itself produces reinforcement. It has both stimulant and depressant effects on the body, and stimulates the release of endogenous opioids. Nicotine has negative as well as positive reinforcement effects. Negative reinforcement refers to the fact that smoking for some persons is related as much to avoidance of the discomfort of nicotine withdrawal as to seeking the pleasurable effects of nicotine.

Nicotine is quickly absorbed through the skin, mucous membranes, and lungs. Absorption through the lungs produces measurable effects on the central nervous system in as little as 7 seconds. This rapid rate of absorption means that each puff on a cigarette produces some reinforcement of the smoking habit.



Metastatic melanoma in the lung, magnified 450 times. (© Lester V. Bergman/CORBIS)

Pure nicotine is a poison that can kill within minutes by causing respiratory failure. Nicotine poisoning most commonly results from accidental ingestion of insecticides containing nicotine. A fatal dose of nicotine for an adult is 40 to 60 mg.

Cancer. Tobacco smoking has been shown to be the major cause of lung cancer in both men and women. The increased risk for lung cancer is directly related to the amount smoked. The risk of death from lung cancer is about twenty times greater for men who smoke two packs a day than for those who have never smoked. It is about ten times higher for those who smoke one-half to one pack a day. Depth of inhalation also influences risk of disease. Tobacco smoking is synergistic (produces a multiplier effect) with the effects of other carcinogenic risks, such as exposure to radon or asbestos. Smoking is also synergistic with alcohol in causing cancers of the oral cavity, larynx, pharynx, and esophagus.

Cardiovascular Disease. Smoking is one of three major causes of coronary heart disease (CHD); risk of death from CHD is 70 percent higher for men who smoke, with a similar effect for women. The risk due to smoking increases if there are risk factors present such as hypertension and elevated cholesterol levels. Smoking increases risk for stroke. For example, women who smoke twenty-five cigarettes or more per day have a risk for stroke almost four times higher than nonsmokers. Smoking also increases the risk of atherosclerosis (formation of plaques) in the peripheral arteries and the aorta. In peripheral arteries this condition can lead to insufficient oxygen reaching the mus-

cles; in the aorta it can lead to a rupture that is usually fatal.

Lung Disease. The link between tobacco smoking and chronic obstructive pulmonary disease (COPD) was noted in the 1964 Surgeon General's Report. COPD includes three related disorders: chronic mucous hypersecretion that causes cough and phlegm production; airway thickening and obstruction of expiratory airflow; and emphysema—abnormal dilation of air sacs and destruction of walls of the alveoli. Compared to nonsmokers, male smokers are three times more likely and female smokers are twice as likely to have a persistent cough.

Other Medical Disorders. These include peptic ulcers, upper respiratory infections, osteoporosis, and cancers of the pancreas, bladder, and esophagus.

The toxic properties and carcinogenic effects of tobacco smoke and its constituents have been studied in the laboratory using animals. The evidence linking tobacco use to death and disease in humans, however, relies heavily on epidemiological studies comparing the rates of various diseases as they occur in smokers versus nonsmokers, in light versus heavy smokers, and in continuing versus former smokers. The level of certainty that links tobacco use to a particular disease varies. Shopland and Burns (1993) have grouped diseases according to their established epidemiological association with cigarette smoking in five categories. These are outlined below.

Category A. Diseases for which a direct causal association has been firmly established and smoking is considered the major single contributor to excess mortality from the disease: cancers of the lung, larynx, pharynx (oral cavity), and esophagus; chronic obstructive pulmonary disease, including emphysema; peripheral vascular disease

Category B. Diseases for which a direct causal association has been firmly established but for which smoking is only one of several causes: stroke; coronary heart disease; cancers of the bladder and pancreas; aortic aneurysm; perinatal mortality

Category C. Diseases for which an increased risk (association) has been demonstrated but a risk whose exact nature has not been firmly established: cancers of the cervix, uterus, stomach, and liver; gastric and duodenal ulcers; pneumonia; sudden infant death syndrome

Category D. Diseases for which excess mortality in smokers has been observed but for which this observation is attributed to confounding variables (other factors that are commonly found among smokers): alcoholism; cirrhosis of the liver; poisoning; suicide

Category E. Diseases for which smokers have lower death rates than nonsmokers: endometrial cancer; Parkinson's disease; ulcerative colitis

The effects of tobacco use are not limited to specific diseases that lead to death. Tobacco use can stimulate enzymes in the liver, and this stimulation can result in alterations in the way various medications are metabolized. This alteration in metabolism can mean that the levels of medications in the body will not be high enough to be optimally therapeutic.

The overall increased mortality from smoking varies with the amount smoked. For those who smoke two or more packs of cigarettes per day, it is about double that of nonsmokers; for those who smoke less, it is about 1.7 times higher than for nonsmokers. The risk for various diseases can be powerfully affected by cessation, but not all risks decline at the same rate. Cardiovascular disease risk decreases markedly within a year of quitting smoking; risks of cancer decline more slowly, with some elevated risk still evident ten years after cessation. By ten to fifteen years after quitting, overall mortality of former smokers is not much higher than that of nonsmokers. Increased mortality rates are not as marked for pipe and cigar smokers, but they are still substantially elevated. The mortality risk for users of smokeless tobacco comes primarily from cancers of the oral cavity and throat.

The adverse effects of passive inhalation (second-hand smoke) are not considered here except in connection with the higher incidence of respiratory illness among the infants of mothers who smoke. But there is no question that there are differences in composition of mainstream smoke (the smoke inhaled by the smoker), sidestream smoke (produced by tobacco burning between puffs), and environmental smoke (the mixture of exhaled mainstream and sidestream smoke). Sidestream smoke is produced at lower combustion temperatures and has higher concentrations of carbon monoxide and organic constituents believed to be carcinogenic.

Psychiatric Disorders. Dependence on tobacco is associated with dysthymic disorder and other forms of depression. It is not yet known, how-

ever, whether depression prompts people to begin smoking or whether it develops in the course of dependence on tobacco. Mood disorders increase significantly during withdrawal from nicotine, and are common reasons for relapse.

WOMEN AND SMOKING

Women who smoke tobacco have the same risks for adverse effects as men. The early impression that women suffered fewer adverse effects from smoking was really due to lower levels of exposure (fewer women smokers and a tendency of women smokers to smoke less heavily.) As has been written more than once, women who smoke like men die like men. In 1986 deaths due to lung cancer among women exceeded deaths from breast cancer, becoming the leading cause of cancer death for women. Some women are at special risk. It has been documented that while the use of oral contraceptives alone does not constitute a serious health risk, the combination of oral contraceptives and cigarette smoking raises substantially the risk of cardiovascular disease, including subarachnoid hemorrhage (bleeding inside the skull).

Women who smoke have higher infertility rates than those who do not and are also more likely to have menstrual irregularities. Nicotine crosses the placenta, and because it constricts blood vessels, a decreased amount of oxygen is delivered to the fetus. In addition, smoking elevates the amount of carbon monoxide in the mother's blood so that it carries less oxygen to the fetus. Women who smoke during pregnancy have higher rates of premature detachment of the placenta (abruptio placentae), premature rupture of membranes, and preterm delivery. The greater the amount of tobacco smoked during the pregnancy, the higher the frequency of spontaneous abortion and fetal death. In the United States smoking has been associated with a 20 percent increase in preterm births among women who smoked a pack a day or more compared with those who did not smoke.

There is no consensus on whether smoking increases the probability of congenital malformations. However, it is well established that babies born to women who smoke during pregnancy weigh on average about seven ounces less than those born to nonsmokers. Apgar scores, a composite of measurements of the breathing, skin color, and reflexes of infants taken at one and five minutes after delivery, are lower for babies of women who smoked during pregnancy. Women who stop smoking early in pregnancy increase their likelihood of having normal deliveries and normal-birth-weight babies. Interestingly, epidemiological data suggest that passive smoke exposure during pregnancy (e.g., living with a smoker) can adversely affect birth weight of the baby. Infants born to mothers who smoke are far more likely to die before their first birthday, primarily as a result of respiratory complications and sudden infant death syndrome. Children of mothers who smoke seem in general more likely to suffer from colds, asthma, bronchitis, pneumonia, and other respiratory problems.

Efforts to educate the public about the health consequences of smoking, including smoking-prevention programs directed at young people and encouragement of smokers to quit, have led to a reduction in the prevalence of smoking in the United States and in several European countries since the mid-1960s. In general, white males in higher socioeconomic groups have lowered their smoking rate more than women and members of ethnic and racial minorities and lower socioeconomic groups. By the early 1960s lung cancer deaths among African-American men exceeded those among white men; by 1990 it was 30 percent higher. The lung cancer rate among both African-American and white women was virtually the same, reflecting similar smoking patterns. On the other hand, smoking rates are increasing in younger age groups in the United States. The rates of smoking have increased from 34.6 percent of the young adult population (aged 18–25) in 1994 to 40.6 percent in 1997 and 41.6 percent in 1998. An estimated 18.2 percent of young people in the 12–17 age bracket were smokers in 1998.

In contrast to the general decline of smoking in the West, the prevalence of smoking may actually be increasing in developing and newly industrialized countries where, even among medical students, cigarette smoking retains a cachet of sophistication and affluence.

(SEE ALSO: *Advertising and Tobacco Use; Complications; Nicotine; Treatment: Tobacco*)

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TOBACCO: SMOKELESS Since tobacco is a plant native to the New World, Native Americans were the first to use it. In addition to smoking it, they used it in smokeless forms—mainly chewing it, making teas and drinks from it, even using the ash in rituals that ranged from South America to Central America and the Caribbean to North America. It was used along with many other plants for both ritual and medicinal purposes.

The use of tobacco was brought to Europe by Columbus and other explorers, where it was taken up for recreation in both the smoked form (cigars and pipes) and the smokeless. Smokeless tobacco (ST) became popular in British society in the practice called sniffing, but British colonists in the Americas preferred to chew tobacco or use snuff. In the 1800s, chewing tobacco was widespread in the United States; its use decreased, however, when the spitting that resulted (into spittoons or cuspidors or wherever the spit fell) was linked to the spread of tuberculosis, one of the most dreaded and fatal of diseases. In addition, the mass production of machine-rolled cigarettes further decreased smokeless tobacco consumption. Around 1900, 52 percent of all tobacco used was smokeless; by 1952, that number had dropped to 6 percent (Lewis, Harrell, Deng, & Bradley, 1999). Indeed, the twentieth century saw declining sales of chewing tobacco until about 1970.

In the twentieth century, there have primarily been two types of ST: (1) snuff, the type one dips by placing it between the cheek and gum, or (2) chewing tobacco, the type one chews and places in the cheek area. Snuff is a cured, ground tobacco that comes in three forms: (1) fine-cut tobacco, (2) moist snuff, or (3) dry snuff (Glover et al., 1988; Christen et al., 1982; Christen & Glover, 1987). Fine-cut tobacco and moist snuff are used by placing a pinch between the cheek and gum or lower lip and gum. Dry snuff may be used by inhaling a pinch through each nostril or by placing a pinch between the cheek and the gum or the lower lip and the gum. Chewing tobacco is also produced in three forms: (1) looseleaf tobacco; (2) plug tobacco; or (3) twist chewing tobacco (Christen et al., 1982; Penn, 1902; Christen & Glover, 1987; Voges, 1984; U.S. Department of Agriculture, 1969; Smokeless Tobacco Council, 1984). All three forms are used by placing a "chaw" in the cheek and periodically chewing.

In the 1970s, the use of ST surged in the United States, with smokers showing a preference for moist snuff. It is increasingly evident that youngsters and adolescents are using ST products much more than they did in the recent past—of the six million users ST users in the U.S. in 1995, up to 25 percent were aged nineteen or younger (Lewis, Harrell, Deng, & Bradley, 1999). This resurgence of popularity over the last thirty years has been attributed to innovative advertising campaigns by tobacco companies

that used sports superstars, cowboy celebrities, and entertainers to promote their products. These campaigns represented an attempt to overcome or erase the old, unsanitary image of the habit, and replace it with a manly or "macho" image (Christen et al., 1982; Shelton, 1982; Glover, Christen, & Henderson, 1981, 1982).

NICOTINE, a dependence-producing drug found in ST, is the same drug that is found in smoking tobacco. Cigarette smokers inhale smoke containing nicotine into their lungs, and the nicotine is then transported into the bloodstream. ST users absorb nicotine directly through the lining of their mouths. Each time smokers smoke a cigarette, they absorb approximately 1 milligram of nicotine into their system. By comparison, people who use chewing tobacco receive approximately 4.5 milligrams of nicotine per chaw, and people who use snuff receive approximately 3.6 milligrams of nicotine per pinch (Benowitz, 1988).

ST is sometimes viewed as a safe alternative to cigarettes, but it is not. ST is directly related to a variety of health problems: bad breath, abrasion of teeth, gum recession, periodontal bone loss, tooth loss, leukoplakia, nicotine dependency, and various forms of oral cancer (Christen, 1985; Schroeder, Chen, & Kuthy, 1985). There are indications that smokeless tobacco also plays a role in cardiovascular alterations and neuromuscular toxicity (Schroeder & Chen, 1985; Squires et al., 1984).

Survey data as of the mid-1980s indicated that predominantly males use smokeless tobacco. In a large national survey of smokeless tobacco use in college, Glover and colleagues reported that about 22 percent of collegiate males were users of smokeless tobacco, whereas only 2 percent of collegiate females used it (Glover et al., 1986). In a study of 5,078 students from 67 high schools throughout the state of Massachusetts, 16 percent of males and 2 percent of females reported using it "once or twice." Eight and 4 percent of the males studied reported using it "several times" and "very often," respectively (McCarty & Krakow, 1985).

The increasing numbers of individuals who use ST demonstrated a need for education and cessation programs. In 1994, Oral Health America created the National Spit Tobacco Education Program (NSTEP) as part of its Oral Health 2000 initiative. NSTEP has received the endorsement of Major League Baseball and encourages players and users to quit–but the main goal is to reduce ST use among kids. NSTEP's chairman is Hall of Fame broadcaster Joe Garagiola, and baseball stars Frank Thomas and Jeff Bagwell, as well as all-time home run king Hank Aaron, endorse the program. County music superstar Garth Brooks did a public service announcement supporting the NSTEP cause, as did Philadelphia Phillies star Lenny Dykstra, who had all his teeth pulled because of overuse of ST. During spring training in 1997, NSTEP counseled sixteen major league teams on ST education, providing intervention and cessation programs (Walsh et al., 1998). Not only is it important to help the players quit, of course, but it is equally important to reduce the number of ST-using players whom kids idolize and watch every day on cable television.

NSTEP offers users several tips on quitting ST, among them: Be committed, and don't be discouraged by setbacks; quit with a friend or ask for support from non-chewing friends; put three dollars in a jar every day to see the financial benefits of quitting; if tobacco use is sports-related, chew seeds or gum instead; and when the quit date is set, visit the dentist for a teeth cleaning, which should help ease the initial nicotine craving.

Although survey data indicates that ST is used predominantly by men, it is enjoyed by a number of women, particularly Native American women, according to Dr. John D. Spangler, researcher at Wake Forest University Baptist Medical Center. A 2000 study among a group of Eastern Band Cherokee Indian women in North Carolina found that women who used ST were at an eight times greater risk of breast cancer than non-users.

(SEE ALSO: *Adolescents and Drug Use; Advertising and Tobacco Use*)

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TOBACCO: SMOKING CESSATION AND WEIGHT GAIN On the average, smokers weigh less than nonsmokers, and approximately 80 percent of smokers who quit will gain weight. The average weight gain for smokers who quit is 5 pounds compared to about 1 pound for continuing smokers over the same period, although some quitters (about 20 percent) will gain more than 10 pounds, and a smaller number (less than four percent) will gain more than 20 pounds. Women tend to gain more weight when they quit smoking than men, but the reasons for this are not known.

At least three major issues are important in the relationship between smoking cessation and weight gain. First, many smokers express fear of gaining weight as a reason for not quitting or weight gain as a reason for a relapse back to smoking. The data, however, are not clear that this is the case. Second, a number of hypotheses have been used to explain weight gain in quitters. Finally, because of smokers' stated concerns of weight gain accompa-

nying cessation, a number of strategies to reduce or delay weight gain have been tested.

FEAR OF WEIGHT GAIN

Fear of weight gain during smoking cessation is more common in women who smoke than in men who smoke. Among current smokers who have attempted to stop smoking, women also are more likely than men to report weight gain as a withdrawal symptom in smoking cessation. Despite this, there is not a relationship between weight gain concerns and serious smoking cessation attempts for either women or men.

Research on the effects of weight gain concerns on relapse to smoking has yielded mixed results. Although many unsuccessful quitters cite weight gain as the reason for relapse, the majority of studies indicate that weight concerns prior to attempting cessation have no relationship to successful quitting. A few other studies, however, have determined a relationship between the two.

SMOKING CESSATION AND WEIGHT GAIN

It is not clear whether weight gain during cessation is temporary or permanent, although the majority of studies indicate that some weight gain (about 5 pounds) is likely to be long–term. Although the mechanisms responsible for the weight gain are not clear, a number of hypotheses have been set forward. These include a metabolic effect for smokers; this is supported by research indicating that smokers and nonsmokers have few differences in the amount of calories consumed. Another hypothesis is that smoking lowers the body's "set point" for weight and smoking cessation raises that set point to be equivalent to that of nonsmokers. A third hypothesis is based on the observation that an increase in caloric intake occurs in those who stop smoking, and increased consumption may be responsible for the weight gain. Although weight gain is likely to accompany cessation, actual weight gain during smoking cessation does not appear to be related to cessation outcomes. Nevertheless, in reaction to smokers' stated concerns about weight gain, a number of strategies to prevent or reduce weight gain during cessation have been developed.

STRATEGIES OF WEIGHT CONTROL DURING CESSATION

The focus of weight control strategies during cessation has revolved around diet, exercise, and most recently, pharmacologic agents. Weight control programs through behavioral self–management of dietary intake have been largely ineffective. In two large randomized trials of behavioral weight management during cessation, the standard care (control) groups with no weight control intervention had better cessation outcomes than the groups that received the behavioral intervention. One of the studies, however, reported that the amount of weight gained was lower for individuals receiving the dietary weight control intervention than individuals not receiving it.

In recent years, a number of research studies examining the effect of physical exercise on weight control during cessation have been conducted. The majority of these studies have been conducted with women. The largest randomized study to date found that women who participated in exercise as well as a smoking cessation program were twice as likely to be abstinent from smoking 12 months after the program than those who participated in the smoking cessation program alone. In addition, the exercise group gained considerably less weight than the nonexercise group.

Pharmacologic agents are increasingly used to prevent or delay weight gain during smoking cessation. Nicotine itself has been the focus of much pharmacologic research. The effect of various nicotine replacement delivery systems, such as nicotine polacrilex gum, the transdermal nicotine patch, nicotine nasal spray, and the nicotine inhaler, on weight gain has been assessed. Nicotine polacrilex gum has been widely studied for its weight control effects during cessation. An early review of five existing studies showed that gum users gained less weight than those on a placebo; however, the effects were small. Recent randomized studies of the effects of nicotine gum on weight gain suggest that there are no long-term effects of gum use on weight gain, and with the discontinuation of gum, there are no significant differences in weight gain between gum users and nonusers. Overall, findings are mixed in terms of weight gain during use of the other nicotine replacement products. The studies that have been conducted on the nicotine transdermal patch indicate either no effect or a

delayed effect in controlling weight gain during cesssation. Similar findings have been reported for the nicotine nasal inhaler. Overall, it appears that any nicotine replacement effects on weight gain disappear after the nicotine replacement is discontinued.

Other pharmacologic agents have also been examined for their effects on weight gain during cessation. In a study of the effects of fluoxetine hydrochloride (Prozac) on weight gain during smoking, individuals on the drug gained significantly less weight than those on a placebo; however, the followup was very short (10 weeks). A study of the effects of *d*–fenfluramine, which is thought to suppress appetite by releasing serotonin, on weight gain during cessation suggested that *d*–fenfluramine did control weight over a placebo. Serious medical complications that accompany *d*–fenfluramine, at least when used in combination with phertermine, however, have diminished enthusiasm for this drug. A study using phenylpropanolamine, an over–the–counter weight control drug, indicated that phenylpropanolamine users gained less weight and had higher quit rates over a placebo group and a no treatment control group. A study of bupropion (Zyban) and weight gain indicated that weight gain was suppressed while on the drug, but the effect disappeared when the drug was discontinued.

SUMMARY

Smoking cessation is likely to result in some weight gain, with women gaining more weight than men. Both women and men express concern about gaining weight when quitting smoking; however, few studies have found a relationship between weight concerns and successful smoking cessation. Similarly, actual weight gain during cessation does not appear to predict relapse. Dietary programs seem to be ineffective in controlling weight gain during cessation, while exercise programs seem to have some benefit. Pharmacologic agents appear to be successful in delaying weight gain during cessation; however, after withdrawal from the drug, any significant effect on weight gain disappears.

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TOLERANCE *See* Addiction: Concepts and Definitions; Tolerance and Physical Dependence

TOLUENE *See* Inhalants

TOLERANCE AND PHYSICAL DEPENDENCE Tolerance and physical dependence are common *consequences* of drug self-administration. For those interested in understanding and modifying alcohol and drug abuse and the problems they cause, the greatest importance of tolerance and physical dependence is in the contribution they make as *determinants* of drug self-administration. Some alcoholics, for example, can appear normal at BLOOD ALCOHOL CONCENTRATIONS (BAC) that would prostrate most social drinkers. What role, if any, does tolerance play in paving the way to an escalation in drug use and in the medical and psychological problems caused by heavy drug use? In addition to being highly tolerant, alcoholics will also be physically dependent on alcohol. What evidence is there to support the common assumption that physical dependence is a critical factor in maintaining drug self-administration?

Such questions are best answered in the context of a general theory of how drug consumption is regulated. A useful starting point is the proposition that behavior is motivated by its consequences. Where tolerance is concerned, the important consequences of drugs are only those that depend on pharmacological effects. The pharmacological consequences that determine self-administration can be grouped according to whether they promote or restrain drug use. Rewarding consequences are those that increase the likelihood of drug use. Drugs may make a person feel alert, powerful, confident, relaxed, friendly, sexy, or talkative. They may alleviate ANXIETY, DEPRESSION, and physical PAIN. All these consequences and more have been hypothesized and evaluated as promoters of drug use.

People may initiate and maintain an episode of drug use in the pursuit of rewarding consequences, and they may end it because drugs also have aversive pharmacological consequences at higher doses. These effects should also be taken into account as restraints on self-administration. Many restraining consequences of drug use can be suggested, ranging from unwanted dysphoria (a state of unease) to frank physical illness.

In summary, a simple regulatory theory asserts that "reward" drives drug use and "aversion" restrains it. If there is tolerance to the rewarding or aversive effects of drugs, it is clear how tolerance might determine drug use. A reduction in the rewarding effectiveness of a given dose would require an increased dose to obtain the same degree of reward. Similarly, tolerance to aversive effects of a drug might mean a much larger dose could be taken before the restraining aversive effect occurred.

There is remarkably little scientific evidence for the common view that tolerance to the rewarding effects occurs. The common and plausible view that tolerance results in a loss of rewarding effectiveness is based mainly on anecdotal evidence. In contrast, there is ample scientific evidence of substantial tolerance to drug effects that could be viewed as restraints on the motivation to self-administer.

Physical dependence as a promoter of self-administration can be dealt with briefly. The earliest theories of dependence assumed that the avoidance of withdrawal was the most compelling motivation for persistent drug use. The experimental evidence for this view is strongest in the case of opiates, but weak to nonexistent for other drugs, including alcohol.

Tolerance can be characterized as a facilitator of consumption and its consequences, independent of the underlying reasons for drug use. If a person is able to drink a lot more before becoming sleepy or dizzy the capacity to drink is increased regardless of the reason for drinking. If the ability of tissue to resist damage does not increase with the body's capacity to resist the drug effects that regulate consumption, tolerance becomes an important determinant of medical and other problems.

As the twenty-first century begins, concepts of addictive disorders has focussed more on the compulsive and relapsing drug-taking behaviors than on tolerance and physical dependence. To that end, medications have been sought and used in the rehabilitative process. Specific medications have been demonstrated to be helpful for psychiatric disorders coexisting with addiction. Some medications showed promise in controlled studies in helping to rehabilitate patients dependent on nicotine, alcohol, or opiates.

(SEE ALSO: *Addiction: Concepts and Definitions; Causes of Substance Abuse; Research, Animal Model; Withdrawal*)

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TOPS *See* Treatment Outcome Prospective Study

TOUGHLOVE The generic term *toughlove* (or tough love) describes a style of caring applied in diverse interpersonal contexts whereby one person or group reasserts power over another for whom he or she is responsible. Claire Kowalski was the first person to use the term in published material, in 1976, to differentiate a respectful means of caring for elderly people that preserves self-mastery from a smothering style that promotes dependence. Since that first use, others have found the term useful. The Association of the Relatives and Friends of the Mentally Ill endorses the concept (Roberts, 1985). In its most common use today, the term describes the means by which parents of abusive, delinquent, or drug-abusing children can regain parental control. Toughlove is also the name of a SELF-HELP program for these parents and their children.

Toughlove, the self-help program, was developed by Phyllis and David York in 1980. They found that rescuing their daughter, who engaged in highly destructive behavior, did more harm than good. Instead, they permitted natural and logical consequences to correct their daughter's behavior while they sought emotional support from their friends. They wrote and published *Toughlove* (1980) and founded an organization called the Toughlove Support Network (which is described in their later book, 1984). The network's mission is to promote what they view as a mode of intervention for individuals, families, and communities.

According to the Toughlove philosophy, parents are the ones with the dominant power in a family. Children misbehave when parents fail to assert themselves or to take responsibility for their role as parents, but when parents' expectations are stated clearly, a child will no longer control the family. Parents are urged to describe the behavior they expect from their children. Speculation about the causes of child misbehavior is discouraged. Parents do not need to understand why their child misbehaves. Instead, they must act in coalition with other parents to assert control of themselves and their home environment.

Toughlove parents are taught not to feel guilty about their child's misbehavior, because children are responsible for their own actions. A Toughlove parent of a destructive child might say: "We have had enough. We are not rescuing you from the trouble you have caused. We love you enough to say no." Proponents of Toughlove believe that drug and alcohol abuse is the most important causative factor in the disruptive behavior among teens. Once parents suspect drug and alcohol abuse, it is important that they investigate by questioning their child's friends, school officials, other family members, and anyone else their child meets frequently. When parents find drug and alcohol abuse, they must require abstinence. Strict discipline and limit setting are seen as the only means of enabling children to behave and to have a chance of regaining control of their lives.

Parents must confront their child about the drug and alcohol abuse and stipulate the behavior they expect. Toughlove recommends that they require the child to stop using drugs and seek treatment if needed. If a child refuses to comply, he or she is to be ejected from the home. Many uncooperative children are sent to live with another Toughlove family until they are serious about meeting their own parents' stipulations. Children who refuse to

live with another Toughlove family are out on their own until they agree to their parents' rules.

To gain help in maintaining firmness and setting appropriate rules, parents attend a support group consisting of other parents who endorse the Toughlove principles. Toughlove support groups are organized by the parents without any professional leadership. Besides providing support for parents, Toughlove groups evaluate the effectiveness of treatment programs and the effectiveness of professionals who treat children for alcohol and drug abuse.

Hollihan and Riley (1987) used qualitative research methods to study a Toughlove parent group. They found that several themes characterized group sessions and defined the Toughlove program experience for parents. First, the lay-led group emphasized that old-fashioned values are superior to those inherent in today's method of raising children. Second, members regarded child-development professionals as advocates for modern child-raising methods that blame parents for child misbehavior. Third, they described the Toughlove group as their island of support within a pro-child social environment made up of the police, educators, social workers, and the courts. Last, the group provided successful models of rule setting by parents and enforcement of strict discipline—including as a final resort forcing a child to leave home. The group presented a persuasive and comforting rationale for the use of strict discipline that addressed the needs of parents who were experiencing great stress and feelings of failure (Hollihan & Riley, 1987).

Toughlove has been criticized as being simplistic and heavy-handed. According to Hollihan and Riley (1987), parents in the group they observed who did not believe their child was abusing drugs or alcohol were nevertheless instructed in how to document such abuse. Other possible causes of their child's misbehavior were ignored, because the Toughlove solution is supposed to apply in all situations. The tactic of throwing an unruly child out of the house is especially controversial. Although most children go to live with other Toughlove families, some are forced to leave with nowhere to go and can become homeless, a predator or a victim, or a threat to themselves and others. For example, John Hinckley, who attempted to kill President Ronald W. Reagan in 1982, had been cast out of his home by parents who endorsed Toughlove and who later warned other parents to be cautious in disciplining their children.

Neither the Toughlove program nor the style of caring identified with it has been evaluated. On the one hand, there is anecdotal evidence from parents to vouch for it. On the other, as illustrated by the Hinckley family, Toughlove solutions can make matters worse. At present, we do not know whether the positive or the negative is the more common outcome, or whether positive outcomes result from factors having nothing to do with Toughlove.

(SEE ALSO: *Adolescents and Drug Use; Parents Movement; Prevention Movement*)

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TOXICITY *See* Complications; Poison Control Centers, Appendix I, Volume 4

TRANQUILIZERS *See* Benzodiazepines

TRANSIT COUNTRIES FOR ILLICIT DRUGS Transit countries are those through which drug shipments travel to reach local dealers and users. Drugs that come to the United States from South America pass through a six million square-mile transit zone that is approximately the size of the continental United States. This zone includes the Gulf of Mexico, the Caribbean, and the eastern Pacific Ocean. U.S. strategy to deal with the cocaine problem, for example, might best be described as a series of concentric circles around the source and trafficking countries of the Andes, through (1) the surrounding countries in South America (2) the transit countries of MEXICO, Central America, and the Caribbean, to (3) the major consumer countries. Since the 1990s, the United States has similar objectives for dealing with both source and transit countries—namely, to strengthen their governments' political will and capability; to increase their effectiveness in terms of military and law-enforcement activities; and to help inflict significant damage on drug-trafficking organizations.

Since 1990, the U.S. government has developed detailed implementation plans for expanded drug-control activities on a regional and country-specific basis. The strategy emphasizes the major choke points at either end of the international chain: The three source countries of COLOMBIA, Peru, and BOLIVIA at one end and the primary transit countries of Mexico, the Bahamas, Jamaica, and Cuba at the other end. In addition to the source countries, only Ecuador, Venezuela, and Brazil in South America have the potential for profitable cultivation of COCA leaf, but the U.S. government believes that only small-scale cultivation and involvement in drug-transit activities exist in these countries. Consequently, only modest drug-control assistance has been made available to them—largely in the form of training, technical assistance, and commodities—to encourage them to take their own actions against high-value elements, such as money flows and essential and precursor chemicals. Brazil and Venezuela, for example, manufacture essential chemicals used in COCAINE production.

The success in the late 1990s of efforts by the governments of Bolivia and Peru to reduce coca cultivation led to increased coca cultivation and cocaine production in Colombia. In 2000, the U.S. Congress approved $1.3 billion of emergency aid to Colombia to help fight the increasingly powerful drug trafficking organizations. U.S. military assistance and equipment has also flowed into Colombia. As more success was achieved against cocaine source countries in the 1990s, and as pressure built against trafficking through Mexico and the Bahamas, drug traffickers dispersed their growing and processing operations and developed new smuggling routes, many in the Caribbean.

INTERMEDIATE COUNTRIES

The intermediate transit countries in the Caribbean and South America have played an increasing important part in drug trafficking, as opportunities for drug interdiction are more difficult. The small Caribbean states lack resources to perform adequate law enforcement; air drops of drugs to waiting boats have become common, because no Caribbean nation has a marine or security force capable of completely controlling territorial waters. However, operations by the U.S., Jamaica and the Bahamas in the late 1990s led to a decline in cocaine trafficking, while drug trafficking increased in Haiti, the Dominican Republic, and Puerto Rico.

Stopping the flow of drugs in these transit countries goes beyond intercepting drug shipments at sea or in the air. Countries must deny traffickers safe haven and prevent the corruption of political institutions. Moreover, the financial systems in these countries must not be used to launder drug profits. The U.S. government has helped Caribbean and Central American countries implement drug control policies that include the strengthening of law enforcement and judicial institutions, the modernization of laws, the strengthening of anti-corruption measures, and the operation of joint interdiction efforts.

The key to successful drug control in the surrounding and transit countries lies in U.S. ability to develop and use effective intelligence networks. The U.S. Department of Defense uses its intelligence resources, including powerful communications equipment, to assist in the interdiction effort.

STRATEGY SUCCESS

The success of the U.S. strategy for potential source and transit countries is predicated on building long-term institutions in these countries that work with the United States. However, the political destabilization of Colombia in the late 1990s is a potent reminder that policies can produce unintended consequences; the success of Bolivia and Peru in reducing coca cultivation triggered changes in Colombia that dwarf the problems of the previous decade.

To be successful, U.S. agencies must expand their efforts in the Pacific and the Caribbean to (1) collect and process intelligence; (2) help the transit countries develop their own intelligence collection, sharing, and dissemination capabilities; (3) help these countries take action on their own to apprehend traffickers and seize drug shipments; and (4) direct bilateral and multilateral efforts against drug trafficking MONEY LAUNDERING, asset forfeiture, chemical diversion, and drug shipments. However, critics point out that the drug supply can never be stopped and that interdiction efforts are largely a waste of money. They argue for demand-reduction programs in the U.S. However, U.S. policy remains firmly committed to reducing the passage of drugs through transit countries.

(SEE ALSO: *Crop Control Policies; Drug Interdiction; International Drug Supply Systems; U.S. Government*)

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TREASURY, U.S. DEPARTMENT OF *See* U.S. Government: Agencies in Drug Law Enforcement and Supply Control

TREATMENT ALTERNATIVES TO STREET CRIME (TASC) This is a program designed to divert drug-involved offenders into appropriate community-based treatment programs by linking the legal sanctions of the criminal-justice system to treatment for drug problems. The program now serves as a court diversion mechanism or as a supplement to probation or other justice-system sanctions and procedures. Created by President Richard M. Nixon's SPECIAL ACTION OFFICE FOR DRUG ABUSE PREVENTION (SAODAP) and funded by the Law Enforcement Assistance Administration (LEAA) and the National Institute of Mental Health (NIMH), TASC was an attempt to find a way to break the relationship between drug use and crimes committed to support the cost of obtaining illegal drugs. The idea for the initial TASC programs derived from an analysis of the criminal-justice system indicating that many drug-addicted arrestees were released on bail while awaiting trial and were likely to continue to commit crimes. Although there were provisions for supervision of drug-dependent offenders after conviction (on probation) or after release from prison (parole), no such mechanisms were in place to provide supervision of those awaiting trial. Yet, if arrestees could be directed to treatment, success in treatment could be taken into consideration at time of trial.

The first TASC programs, in Wilmington, Delaware, and Philadelphia, Pennsylvania, became operational in 1972. TASC currently operates in more than 100 jurisdictions in 28 of the U.S. states and territories. In the mid-1990s, TASC programs received support from the U.S. Department of Justice through the Bureau of Justice Assistance (BJA) Criminal Justice Block Grants to state and local governments. LEAA was discontinued in 1982. Many TASC programs have expanded their base of support so that state and federal funding is supplemented by private donations and grants or client fees.

TASC programs initially focused on pretrial diversion of first offenders. The original TASC model was structured around three goals: (1) eliminating or reducing the drug use and criminal behavior of drug-using offenders; (2) shifting offenders from a system based on deterrence and punishment to one that, in addition, fostered treatment and rehabilitation; and (3) diverting drug-involved offenders to community-based facilities so as to limit criminal labeling and also to avoid the learning of criminal

behavior that occurs in prisons. These goals were based on the assumption that treatment intervention had a better chance of success with first-offenders, since they had not yet been labeled as criminals. It also reflected community concerns that serious or dangerous offenders who might otherwise be incarcerated would instead be released. In practice, it turned out that most first-time *drug* arrests were not necessarily *first* arrests, so the program was quickly expanded to reach all drug-involved offenders that the courts were willing to divert into treatment.

TASC procedures determine a drug-dependent offender's eligibility for intervention, and they include assessment of the offender's risk to the community, severity of drug dependence, and appropriateness for treatment placement. After an individual is referred to a treatment program, TASC case-managment services monitor that individual's compliance with the conditions of the treatment and rehabilitation regime, including expectations for abstinence, employment, and improved personal and social functioning. Progress is reported to the referring justice-system agency. Clients who violate the conditions of their justice mandate—TASC "contract" (or treatment agreement)—are usually returned to the justice system, where the legal process interrupted by TASC diversion goes forward.

Specific "critical program elements" define the parameters of a well-described national TASC model. These have been carefully worked out by The National Consortium of TASC Programs (NCTP) (444 North Capitol Street, NW, Suite 642, Washington, DC 20001; Phone: 202/783-6868; FAX: 202/783-2704). These critical elements provide the structure for the linkages between the criminal-justice and treatment systems. This model makes it possible to easily replicate TASC programs anywhere in the United States, including urban, suburban or rural settings, and is easily adaptable to specific population needs. NCTP provides technical assistance for implementation of the model program, training for program development, systems coordination, program assessment, development and dissemination of materials (such as model policies, procedures, protocols, etc.), training in the use of the "critical elements," internships, and accreditation of TASC programs.

Many of the states have expanded the TASC model to provide a wide array of adjunct services to a wide variety of participants in TASC programs. Illinois TASC, for example, founded in 1976 by Melody Heaps, uses the name Treatment Alternatives for Special Clients (TASC, Inc.) in order to better describe the scope of its programs. The program provides case management and a comprehensive array of services throughout Illinois for men, women, and adolescents who have a variety of social, welfare, and health-related needs. Populations served include youth in the child-welfare system, AIDS-affected clients, DUI (drunk-driving) offenders, juvenile offenders, students, welfare recipients, offenders sentenced to home confinement, youth in community-based programs and those in the child-welfare system, Supplemental Security Income (SSI) recipients, pretrial arrestees, and Cook County Jail inmates. For each special population targeted and served, appropriate interventions and services have been devised, such as a school intervention program, a gang intervention program, and youth services for substance-abusing students and adolescents. Adult criminal-justice services include monitoring of offenders in home confinement using technologies such as electronic monitoring and drug testing; a jail project providing screening and assessment, orientation, intensive therapeutic-community counseling, transition counseling, and aftercare planning and management. Illinois TASC is the sole agency providing substance-abuse assessment and recommendations for the Illinois courts. As well as providing offender case-management services, it offers training for judges, state attorneys, public defenders, criminal-justice planners, and federal and state probation and parole staffs.

TASC programs play an important role in reducing the growing rates of drug-related street crime and alleviating court backlogs. They have been effective in identifying drug-involved offenders in need of treatment, assessing the nature and extent of their drug use and their specific treatment needs, and referring them to treatment. TASC clients have been found to remain in treatment longer and so have better posttreatment success. In addition, as an adjunct to parole and work release, the programs have the potential to help ease prison overcrowding. TASC also effectively fulfills its original purpose of linking the criminal-justice and treatment systems by providing client identification and monitoring services for the courts, probation

departments, and other segments of the criminal-justice system.

(SEE ALSO: *California Civil Commitment Program; Civil Commitment; Coerced Treatment for Substance Offenders; Crime and Drugs; Narcotic Addict Rehabilitation Act*)

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TREATMENT *See* Treatment; Treatment Types

TREATMENT CENTERS DIRECTORY *See* Appendix, Volume 4

TREATMENT FUNDING AND SERVICE DELIVERY No single accepted method or setting exists for the treatment of substance abuse—alcohol and other drug-abuse disorders. Treatment is offered in specialty units of general and psychiatric hospitals, residential facilities, halfway houses, outpatient clinics, mental-health centers, jails and prisons, and the offices of private practitioners.

In the United States during the 1970s and 1980s, drug abusers were commonly treated in programs distinct from those serving alcoholics. By the 1990s, the two treatment systems were merged; in 1991, of the estimated 11,000 substance-abuse treatment programs in the United States, 79 percent reported that they served both drug and alcohol abusers. Some 88 percent were enrolled in outpatient programs. Another 10 percent were in residential facilities. Only 2 percent were hospital inpatients.

The cost of treatment varied greatly depending on setting. In the early 1990s, hospital inpatient care was the most expensive on a daily basis ($300–600/day), but it was usually of short duration (30 days or less). Treatment in nonhospital residential programs was less expensive ($50–60/day), but it commonly lasted longer (a few months to 2 years). Programs that did not require the individual to live in a specialized facility were the least expensive, both on a daily basis ($5–15/day) and over a full course of treatment.

PRIVATE HEALTH INSURANCE

The availability of private health-insurance coverage for substance-abuse treatment grew in the 1980s. By 1990, better than 90 percent of health-insurance plans had explicit coverage for drug treatment. Individuals with such private insurance have a greater range of treatment providers from which to choose than those who are indigent and have only government-funded programs at their disposal. Programs that mainly rely on insurance reimbursement, however, tend to be more expensive than those that receive the bulk of their support from government sources.

U.S. GOVERNMENT FINANCING

In the U.S. general health-care system, 68 percent of the cost of services is borne by the individual, insurance company, or other private third-party payer. For substance-abuse or mental-health care, in contrast, the government supplies 63 percent of the funds for substance-abuse treatment. After the private sector, which provides 37 percent of the funds, the states traditionally have been the major source of treatment support (31%), followed by the federal government (24%), and then county and local agencies (8%). States often finance treatment by reimbursing providers through public-welfare programs or through grants or contracts. Some states transfer funds to county and local governments, which, in turn, purchase services from providers. Another financing mechanism is Medicaid, a combined state and federal program that pays medical bills for low-income persons. Under Medicaid, states can pay for substance-abuse care in inpatient general hospitals, clinics, outpatient

hospital and rehabilitation services, and in group homes with sixteen or fewer beds.

A federal program that pays the health-care costs of persons 65 years of age or older, or those who are disabled, is Medicare. This primarily covers inpatient hospital treatment of alcohol or drug abuse, as well as some medically necessary services in outpatient settings. The primary federal mechanism for paying for alcohol and drug treatment is the Substance Abuse Block Grant, administered by the Department of Health and Human Services. Funds from the block grant are distributed to the states (and territories) using a formula that takes the characteristics of the state's population into account. In fiscal year 1994, Congress appropriated approximately 1.3 billion dollars for the Substance Abuse Block Grant. The federal government also makes grants to individual treatment providers to support innovative treatment approaches, improve the quality of treatment, or to ensure services for underserved or special populations.

(SEE ALSO: *Treatment; U.S. Government Agencies*)

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TREATMENT, HISTORY OF, IN THE UNITED STATES The history of the treatment of alcohol and other drug problems is often assumed to be a straightforward story of progress—moralism, neglect, and brutality were displaced by scientific knowledge, medical activism, and professional civility; a view that the addict exercised free will in choosing to use drugs was succeeded by an understanding of how a "disease" or "disorder" could overrule the capacity to choose.

This assumption is historically incorrect. First, it neglects the coexistence and mutual influence of views emphasizing free will or social or biological determinism. While one view may have enjoyed greater influence at a given time, its competitors have never been vanquished. No generation has any more solved the puzzle of addiction than it has resolved the related enigmas of the relationship between mind and body, choice and compulsion. Second, it is equally incorrect to associate condemnation and neglect with the free-will position or kindness and activism with the determinist perspective. The truth is more complicated.

As various studies have demonstrated, there is a tenacious American folk wisdom about addiction. Simply put, it goes as follows: While addicts experience a compulsion to take a drug, this develops as the result of repeated bad choices that are socially influenced; further, addicts can rid themselves of compulsion only by developing self-discipline, perhaps with some skilled influence in the form of treatment. Thus, in our culture, and despite the modern message that "addiction is a disease like hypertension or diabetes," addicts are understood to be both sick and immoral, blameless and culpable, free and determined. In the popular mind, and among treatment professionals, addicts are ambiguous characters.

The history of treatment in the United States reflects this cultural dilemma. Cultures limit the range of possible responses to a problem, and because they tend to change very slowly in fundamental ways, to the extent that an important problem recurs or remains unsolved, the range of possible responses will be explored repeatedly as new generations search for fresh insights and effective methods of intervention. At various times, treatment has embraced exhortation and coercion, sermons and miracle drugs, democratic mutual aid, and autocratic professional prerogative—often simultaneously.

THE PREMODERN ERA

Modernity has different meanings with respect to the treatment of habitual drunkenness and drug addiction. In the case of habitual drunkenness, the modern era is traceable to the birth of ALCOHOLICS ANONYMOUS (AA) in 1935. In the case of drug

addiction, delineating historic periods is more difficult, but we will mark the modern era by the introduction of methadone maintenance (for heroin dependence) in 1965 and passage of the federal NARCOTIC ADDICT REHABILITATION ACT (NARA) in 1966.

We should also clarify our choices of terminology. The terms *alcoholism* and *alcoholic* date from the middle of the nineteenth century, but they did not come into common professional use until the early twentieth century and were not embedded in the American vernacular until after the rapid growth of AA during the 1940s. The more common professional terms in the premodern era were *inebriety* and *inebriate,* but as these often were used to refer to a heterogeneous group now called "substance abusers," we will use the durable term *drunkard* when writing about this era. Similarly, the term *drug addict* was not in common use until the early 1900s; before this time habitual users of drugs were known as "morphinists," "cocainists," or sometimes, "dope fiends." In order to speak generally and to avoid pejorative (if historically accurate) terminology, we will use drug addict, and we will use addict and addiction when speaking of both habitual drunkards and drug addicts.

THE TREATMENT OF HABITUAL DRUNKARDS

The Tradition of Mutual Aid. The organized, specialized effort to help habitual drunkards began with the Washington Total Abstinence Movement in 1842. This Washingtonian Movement stands at the head of a tradition of mutual aid that developed throughout the 1800s in close connection to American Protestantism, particularly its evangelical expressions. The Salvation Army, which traces its American incarnation to the mid-1870s, is also in this line, and so is AA and the many other "Anonymous" fellowships it inspired.

Washingtonian societies were dedicated to sobering up hard drinkers, usually (but not always) men. The societies intended to foster a solidarity based on shared experience with suffering that transcended profound social divisions. (They were neutral on the divisive question of prohibition.) Although some famous teetotalers like Abraham Lincoln were members, the societies included the disreputable, the unlettered, and sometimes nonwhites and women as equals. Their motives were couched in terms of Christian charity, economic self-improvement, and democratic principles.

The hallmark of mutual aid is the banding together of people in similar circumstances to help one another. (The popular term "self-help" is thus misleading.) The Washingtonians and their successors did not invent the methods by which they fostered solidarity and mutual support. However, in adapting the voluntary association to the reform of drunkards, the Washingtonians introduced new elements.

Owing its provenance to the revival meeting, the most striking and controversial (some found it distasteful) Washingtonian innovation was the confession of drunkards before their peers, and sometimes before a general audience. We are familiar with its contemporary form: "I am Jim B, and I am an [alcoholic, drug addict, etc.]"; but the practice dates from Washingtonian "experience lectures," forums for the telling of "drunkard's tales," stories of degradation, struggle, and redemption through sobriety. These introduced the drunkard's tortured inner life to the polite public. "You all know me and what I used to be," Salvation Army lecturers often began.

Some Washingtonian societies also established temporary homes, or refuges, for drunkards. These were places where drunkards could live for a short time while they sobered up and were introduced to the Washingtonian fellowship, whose members found them jobs and other necessities. A century later, AA would reinvent this institution (the recovery home) as part of its twelfth-step work—the commitment to help other drunks.

Although not continuous with these early refuges, beginning in Boston (1857), San Francisco (1859), and Chicago (1863), a number of formal inebriate homes were established to treat drunkards in the Washingtonian tradition. Typically, these were small institutions (fewer than 50 beds), operated as private charities, sometimes under religious or temperance auspices. They relied on the voluntary cooperation of their residents and used temperance fellowship as a form of what we now call aftercare. They were located in urban environments and did not isolate their residents from community life. Although they often were superintended by physicians, residence rarely exceeded three weeks and medical treatment was considered important only in managing withdrawal symptoms or DELIRIUM TREMENS (DTs). The terms

disease and "vice," *cure* and "reformation" were used interchangeably, and sober outcomes were attributed to the influences of family, friends, and the fellowship, not to medical intervention. Inebriate homes practiced a profoundly social (and sometimes spiritual) form of treatment based on the belief that the human capacity for transformation was never extinguished, no matter how "despotic" the "appetite" for alcohol.

For those in the Washingtonian line, the source of such optimism was their belief in the presence of an immortal God in the human mind. The mind, they believed, was distinct from the brain and other corruptible flesh and was formed in God's image. By the mid-1800s, the image of God was far more benign and rational than the often wrathful, finally inscrutable deity of even the early 1700s. This gradual change in the conception of God owed much to the spread of the market as arbiter of economic affairs and social relations. The rigorous logic of the market reordered economics from the academy to the workshop. In its train, a disciplined, optimistic rationalism—and the ideas of moral progress and human perfectibility—suffused popular culture and theology.

At the same time, another form of rationalism, that of natural science, was pervading popular discourse and causing tumult in seminary and pulpit. Science did not overthrow religion so much as assume a place alongside it. For believers, scientific order was a wonder of the divine plan. The natural "laws of health," as various rules of disciplined self-denial were known, were signals of divine intent, of God's ideas about right living. The drunkard was therefore both sinful and sick, having contracted the disease as the result of moral transgression. (A common analogy of the time was to syphilis; today, some religious leaders speak similarly of AIDS.) Thus, while Washingtonians and their successors spoke of addiction as a disease—by which they meant an organically based compulsion—they also employed clerical images, for they believed in the power of the divinely inspirited human mind to choose the rational good (total abstinence from alcohol) and to thus achieve health. In the Washingtonian tradition, the languages of morality and disease became assimilated, and remain so in the many contemporary Anonymous fellowships' claim that addiction is in part a "spiritual disease."

Although the Washingtonian Movement as such was defunct by 1850, Washingtonianism was extremely influential until about 1865. The tradition did not disappear, but in the decades following the Civil War (1861–1865), profound changes in American culture and society, and related changes in the temperance movement, blunted Washingtonian influence and gave new prominence to a competing philosophy of treatment and its attendant practices and institutional embodiment. The philosophy was that of biological determinism, or "somaticism," and its institutional expression was the "inebriate asylum."

The Asylum Tradition. In 1810, Benjamin Rush, a Philadelphia physician, signer of the Declaration of Independence, and first formulator of a disease theory of addiction (though not the inventor of the idea), proposed "sober houses" for drunkards. However, Samuel Woodward, a Massachusetts insane asylum superintendent and temperance orator was the father of institutional treatment based on a somatic explanation of habitual drunkenness. In a tract written in 1835, Woodward contributed two critical ideas to what would become the inebriate asylum movement of the nineteenth and early twentieth centuries. The first was that drunkards could not be treated successfully on a voluntary basis. The second, which flowed from the first, was that they needed legal restraint in a "well-conducted institution"—by which Woodward meant something like the insane asylum that he superintended.

The line of thinking staked out by Rush and Woodward had no institutional realization until an inebriate asylum subsidized by the State of New York opened in Binghamton in 1864. Another was opened in Kings County, New York, in 1869. In subsequent decades, pursuant to arduous promotion by the American Association for the Cure of Inebriates (AACI, founded in 1870), public inebriate asylums opened in Massachusetts (1893), Iowa (1904), and Minnesota (1908). Other jurisdictions chartered inebriate asylums but never built them (Texas and Washington, D.C.), and in California an inebriate asylum chartered in 1888 was converted to an insane asylum before the facility opened in 1893. Indeed, Binghamton was converted to an insane asylum in 1879. By the advent of Prohibition in the United States in 1920, all public inebriate asylums had been closed or converted to other use.

The inebriate-asylum movement spawned dozens of private sanitaria that treated well-to-do drunkards and, by the 1890s, drug addicts. However, judged by its manifestation in brick and mortar, the movement for public treatment was a failure. For two related reasons, the AACI was notably unsuccessful in converting legislatures to its cause. First, its physician members never could produce a strictly medical "cure" for addiction. Although its theorist-practitioners developed rigorously somatic explanations of addiction that dispensed with will power, spirituality, and the therapeutic necessity of fellowship, they relied on recuperation by bed rest, a healthy diet, and therapeutic baths (hydrotherapy), followed by the discipline of useful labor. This regime was highly structured (military analogies were popular) and medically supervised, and was set in a context of prolonged legal restraint (involuntary commitment). However, there was nothing particularly innovative or medical about this approach. Its methods already were the staples of lunatic asylums (called mental hospitals in most states after about 1900), almshouses, and county jails, institutions that managed huge numbers of habitual drunkards and, after the 1880s, drug addicts. Second, the inebriate asylum was an ambitious undertaking: like the insane asylum, it was to accommodate several hundred patients on a sequestered rural estate. Few legislatures could be persuaded that such costly new institutions were worth the price. In a word, the inebriate asylum was viewed as redundant.

The ideology of the inebriate-asylum movement—its adherents' view of the world—was shaped by two profound, contemporaneous developments in American culture and society: (1) the rising esteem and secularism of science and (2) the growing disorder and complexity of American society after the Civil War. The movement reflected the grand aspirations of Gilded Age science, whose practical applications were transforming American life: railroads and streetcars, the telephone, gas and electrical lighting—all attested to the power of science and human ingenuity. It was a time when "scientific" understanding became the basis for professional standing, not only for medicine, but for all manner of professional groups, from proto-social workers to plumbers. The metaphor of disease, and the optimistic message implicit in its use—that all defects could be cured—became popular among forward thinkers. In the most widely read book of its time, the utopian novel, *Looking Backward* (1888) by Edward Bellamy, the author characterized all sorts of misconduct as disease, and his near-perfect world of the year 2000 cured its rare wayward citizens in public hospitals.

If Washingtonians assimilated the languages of morality and disease, the rising generation of inebriate-asylum enthusiasts radically separated them, and often reduced human volition to a by-product of neurology. In the United States and Europe, they initiated research on the biology (and later, the genetics) of addiction. Primitive by today's standards, it nonetheless established a robust tradition of inquiry that remains lively.

The inebriate-asylum movement appealed to American aspirations to create a better world through science, but it also addressed growing fears of social disorder. The extent of such disorder should not be exaggerated, however; pre-industrial America was more disorderly than nostalgic chroniclers have made it seem, and urbanization and industrialization were less chaotic than critics sometimes contend. On the whole, though, life after the Civil War was more complex, more anonymous, and less certain.

Immigration from abroad was an important fuel for such change and promoted the (American) nativist fears that accompanied it. In the 1830s, free Americans were overwhelmingly Anglo-Saxon in origin and Protestant in belief. By the 1880s, this was changing dramatically. Burgeoning northern and western cities were becoming testing grounds for the promise and limits of diversity—indeed, for explanations of diversity. Amid glaring inequality of wealth and opportunity, cultural conflicts often were played out around practices of consciousness alteration. Protestant, native-born Americans (including African Americans) were remarkably abstemious (a notable success of the Protestant-driven temperance movement); the mostly Roman Catholic Italians and French were daily wine drinkers; Poles, Germans, and some Scandinavians drank large quantities of beer (some on Sunday—in public beer gardens).

Of Irish Catholics, who had a large temperance movement of their own but also a penchant for drunkenness (what is known as a "bi-modal distribution" of drinking habits), a California temperance editor wrote in 1883: "They are by far the worst and meanest material in which to store

whisky." Native Americans had been introduced to alcohol by traders and government agents from colonial times, so "firewater" became a factor in the westward movement and the ensuing Indian Wars. The "idolatrous" (non-Judeo-Christian) Chinese introduced opium smoking to America, a practice that crossed the color line during the 1870s and became popular among young white men and women during the 1880s. Then from 1900 to 1920, Mexicans became associated with *Cannabis* (MARIJUANA) use in the West and Southwest. In the South, African-American men frequently were accused of the riotous use of COCAINE, with subsequent designs on white women.

The increasingly diverse backgrounds of the U.S. population became a source of conflict and disorder; the rollercoaster ride of industrial capitalism was another. The United States experienced two prolonged economic depressions (then called "panics") between the Civil War and the turn of the century— from 1873 to 1878 and from 1893 to 1898. In between, a short but sharp slump during the mid-1880s took its toll on stability. During these years, the noun "tramp" entered the American language; the country experienced its first pronounced labor violence and political bombings (dynamite being an 1860s product of scientific ingenuity); in the spring of 1894, "armies of the unemployed" converged on Washington, D.C., from all over the country.

This era of mounting diversity and instability was marked by a failing faith in exhortation (verbal appeal) as a method to achieve social regulation and by a concomitant exaltation of coercive means (force). Although never abandoning altogether its sympathy for drunkards, the temperance movement made securing prohibitionist measures its primary objective. Although never withdrawing its support from surviving Washingtonian institutions, temperance adherents simultaneously supported the more stringent regime promoted by inebriate asylum enthusiasts, some of whom believed that an orderly, peaceful society required the lifetime detention of incurable addicts. Indeed, the temperance movement helped to popularize theories that purported to demonstrate a biological basis for the failure of certain racial and ethnic groups to live up to the abstemious standard of so-called native stock—or to benefit from treatment. In the name of "prevention," such views justified not only prohibition laws but also statutes that in a few states permitted the forced sterilization of addicts.

In sum, the legacy of the inebriate-asylum movement was the biologically based approach to understanding addiction, the corollary claim that addiction is the special province of medicine and physicians, the notion that successful treatment requires legal coercion, and the assertion that treatment is both a responsibility of government and a commodity to be sold on the market. These ideas endure as part of the complex intellectual, professional, and political fabric of treatment.

The Tradition of Mental Hygiene. The mental hygiene movement, customarily dated from the 1908 publication of Clifford Beers' *A Mind That Found Itself*, represented a departure from the somatic tradition of thought about mental disorder and addiction. At the same time, it did not appeal to spiritual explanations nor did it dwell on will power. Rather, mental hygienists employed a sociobiological determinism: Although addiction could be the result of hereditary biological defect, and could be incurable, its origins were mainly familial and social, and if the condition was addressed early on, could be arrested. Mental hygienists stressed the important roles of family, friends, and occupation in creating a salubrious environment for an addict's continuing sobriety. Mental hygiene did not speak the language of mutual aid, but it was similarly environmental in outlook. This was the beginning of what later would be called community mental health, and its point of view virtually defines what we understand to be "modern" about treatment and the biopsychosocial perspective.

The environmentalism of mental hygiene challenged the rationale of the asylum model of treatment. Mental hygienists criticized the asylum's lack of connection with community life and its reliance on involuntary treatment, claiming that only voluntary access to free or inexpensive care would attract patients in the early stages of drinking or drug-taking careers. The history of the Massachusetts Hospital for Dipsomaniacs and Inebriates (1893–1920) illustrates well the influence of mental hygiene philosophy and practice. Between 1893 and 1907, the hospital was run on the asylum model. After a complete reorganization in 1908, it followed a mental hygiene course: Most of its admissions were legally voluntary; the hospital established a statewide network of outpatient clinics; it worked closely with local charities, probation of-

fices, employers, and the families of patients. Known finally as Norfolk State Hospital, it was a preview of what treatment was to become, beginning in the 1940s.

Even so, Norfolk created on its campus a "farm" for the long-term detention of "incurables." The mental hygiene movement modified the emphasis of the asylum tradition but did not entirely abandon its practices. Indeed, under the banner of mental hygiene, between 1910 and 1925, many local governments across the United States established "farms" to segregate repeated public drunkenness offenders and drug addicts. Some of these persisted until the 1960s, and some have been reopened in recent years to accommodate homeless people with alcohol and drug problems. As discussed below, the asylum tradition remained particularly important in the treatment of drug addicts.

TOBACCO

Although tobacco use is now widely considered in the United States to be a problem akin to drug dependence, for most of the twentieth century it was not treated as such by either the medical or criminal-justice establishment. However, nineteenth-century temperance groups saw tobacco use as another form of inebriety. As far back as the 1890s, advertisements for patent medicines claimed to help people break the tobacco habit. In the great temperance upsurge of the early twentieth century, more than twenty states passed tobacco prohibition laws, but most of these were quickly repealed. Public concern with habitual tobacco use declined dramatically from the 1920s through the 1950s, and cigarette smoking (over smokeless tobacco, pipes, or cigars) became normative behavior among men and grew steadily among women. This situation changed abruptly with the publication of the 1964 *Report of the U.S. Surgeon General* that linked cigarette smoking to cancer. Since then, increasing attention has been paid to the tobacco habit, or TOBACCO DEPENDENCE, and to treatment for it. Treatment approaches are at least as varied as those described here for alcohol and other drugs. Pharmacological treatments, such as nicotine chewing gum and skin patches, have been used, as have acupuncture, hypnosis, mutual aid, aversive electric shock, and other techniques. While many people advocate that government or private insurance should pay for treatment of this addiction, to date there have been no suggestions that tobacco addicts should be treated on a compulsory basis, although the places where it is legal to smoke have been diminishing.

THE TREATMENT OF DRUG ADDICTS

Although the San Francisco Home for the Care of the Inebriate (1859–1898) treated a few opium addicts as early as 1862, Washingtonian institutions mainly treated drunkards. Similarly, although a few reborn drug addicts were among the legions of the Salvation Army and other urban missions by 1900, they were vastly outnumbered by reformed drunkards. Until the organization of what is today NARCOTICS ANONYMOUS (NA) in 1953, there was no large or well-defined group of addicts involved in the practices of mutual aid, and there were a variety of reasons for this.

Drug addiction was not a matter of widespread concern until after the Washingtonian philosophy had been eclipsed by the asylum model of treatment. Further, drug addicts were quickly perceived to be more exotic and ominous than habitual drunkards. Although there were many people addicted to morphine as a result of ill-advised medical treatment or attempts at self-treatment during the late 1800s, this more or less respectable population declined after the turn of the century as physicians and pharmacists reformed their dispensing practices and new laws required the disclosure of the content of patent medicines and nostrums. At the same time, a growing number of urban young people began to experiment with drugs, especially smoking opium, morphine, and cocaine. By 1910, drug addiction was popularly associated with petty thieves, dissipated actors, gamblers, prostitutes, and other nightlife aficionados, and with racial minorities and dissolute youth. Unlike habitual drunkards, drug addicts never were caricatured as boisterous and occasionally obstreperous nuisances or buffoons; especially after 1900, they usually were portrayed as dangerous predators and corrupters of society, alternating between drug-induced torpor (in the case of opiates) or hyperactivity and hallucination (in the case of cocaine) and a craving that propelled them on relentless and unscrupulous searches for drugs and the means to buy them.

The "criminal taint" of drug addiction, and the widespread view that most addicts were incurable

and would do anything to alleviate withdrawal symptoms, provided a powerful rationale for their prolonged confinement under strict conditions. Even the mental hygienists at Norfolk State Hospital had no expectation that addicts would remain sober and favored incarcerating them in the Massachusetts State Farm at Bridgewater, a correctional facility. Indeed, state hospitals were generally more opposed to admitting addicts than habitual drunkards, preferring to have them incarcerated in jails. Even more than drunkards, addicts disturbed the routine and good order of state hospitals, in no small part because they were, as a group, considerably younger and less conventional than other hospital patients. They pursued sexual liaisons in violation of institutional rules against fraternization; they smuggled drugs into the hospitals; and once through withdrawal, they escaped in droves.

Nor were jails and prisons anxious to take in addicts, mainly because of the problem of smuggling. By the late 1880s, opium was a customary (though illicit) medium of exchange at San Quentin Prison in California, and it was routinely available in the big county jails of the United States at the turn of the century. As state laws against the sale or possession of opiates and cocaine proliferated in the 1890s, and as they began to be more strictly worded and enforced after 1910, county jails and state prisons faced a major problem of internal order. This intensified with the implementation of the federal HARRISON NARCOTICS ACT (passed in 1914 to take effect in March 1915), particularly after a U.S. Supreme Court decision in 1919 made it illegal for physicians to prescribe opiates for the purpose of maintaining an addict's habit. The vast majority of drug offenders, even those arrested by federal agents, were prosecuted under state drug and vagrancy laws and sent to state and county lockups. The resulting crisis led jailers to support two related treatment strategies.

The first of these was the creation of special institutions for drug addicts. Thus the county farms mentioned earlier in this essay were created, or laws were passed to allow addicts to be committed to existing state or county hospitals with wards designated for this purpose. Mendocino State Hospital in California, Worcester State Hospital in Massachusetts, Norwich State Hospital in Connecticut, and Philadelphia General Hospital, to name a few, treated significant numbers of addicts in the 1910s and 1920s. Later, California (1928) and Washington (1935) opened state-sponsored variations on the jail farm, though under the auspices of their state hospital systems.

Nancy Reagan greets local youngsters who are members of the "Just Say No" club at the White House, June 22, 1986. (© Bettmann/CORBIS)

The growing number of addict-prisoners in the federal system also led to their segregation, first at Leavenworth, Kansas (mainly), and then at two narcotic hospitals opened at Lexington, Kentucky (1935) and Fort Worth, Texas (1938). Operated by the U.S. Public Health Service, these hospitals were in fact more like jails, although they were authorized to admit voluntary patients of "good character" whose applications were approved by the U.S. Surgeon General. Initially, these patients were kept involuntarily once they had been admitted, but a federal district court ruling in 1936 affirmed that voluntary patients could leave after giving notice. Before they were closed in the 1970s, the two facilities had admitted more than 60,000 individuals comprising over 100,000 admissions.

Jailers were also an important part of local political coalitions in support of a short-lived and controversial treatment strategy of the early 1920s—drug dispensaries for registered addicts. At least forty-four such clinics were established nationwide, most in late 1919 or early 1920, following the Supreme Court's antimaintenance ruling.

In principle, these were not to be maintenance clinics. Addicts initially were to receive their customary dosages of morphine (occasionally heroin, and very rarely, smoking opium), and were then to be "reduced" over a short time to whatever dosage prevented withdrawal. At this point, abstinence was to be achieved.

In practice, few of the clinics worked this way. Many clinic operators believed that their primary aim was to mitigate drug peddling by supplying addicts through medical channels. This implied a maintenance strategy at odds with the Supreme Court's interpretation of the Harrison Act and with some earlier state laws forbidding maintenance (in California and Massachusetts, e.g.). Further, most clinic operators agreed with the American Medical Association (AMA) that dispensaries could only work effectively within the law if prolonged institutional treatment was available once the addict's dosage had been reduced to the brink of withdrawal. In the absence of such institutional capacity, reduction was useless, and so clinic doctors rarely bothered. The Prohibition Unit of the U.S. Department of the Treasury (which enforced the Harrison Act), state boards of pharmacy (which typically enforced state drug laws), and local medical societies and law enforcement agencies regarded the clinics as stop-gaps, valuable only until adequate public hospitals could be opened.

In the midst of the inflation following World War I, localities looked to the states to finance such institutions and states looked to the federal government, particularly the U.S. Public Health Service, which had operated hospitals for merchant mariners since 1792. But legislation to create a federal treatment program failed to pass and the states were thrown on their own resources. The Prohibition Unit, convinced that the clinics were doing more harm than good, moved to close them, threatening dispensing physicians with prosecution. The clinics closed rapidly. The last one, at Shreveport, Louisiana, closed in 1923. Addicts were consigned to their customary ports of call in jails, prisons, or for the fortunate few, private sanitaria.

The controversy over maintenance did not disappear, however, particularly on the West Coast, where efforts to loosen its prohibition in the states of California and Washington continued until the United States entered World War II (1941). Further, both federal and state governments permitted the maintenance of a small number of addicts, usually of middle age or older, suffering from severe pain related to a terminal illness or an incurable condition. However, the period from 1923 through 1965 was generally characterized by the strict enforcement of increasingly severe laws against drug possession and sales, by relentless opposition to maintenance, and by treatment that was essentially in the asylum tradition, supplemented by the mental hygiene innovation of supervised probation. In 1961, California passed legislation permitting the compulsory treatment of drug addicts (including marijuana users) and established the California Civil Addict Program within the Department of Corrections. From 1962 to 1964, more than 1,000 people were committed to a 7-year period of supervision, which typically involved an initial year of residential treatment in a facility surrounded by barbed wire to discourage premature departure. In 1964, New York passed similar legislation but assigned its implementation to a special commission rather than to the Department of Corrections. As in California, New York's residential treatment facilities were "secure." As late as 1966, the federal NARCOTIC ADDICT REHABILITATION ACT (NARA), in most respects a piece of "modern" legislation, nonetheless provided for the compulsory treatment of addicts and made the hospitals at Lexington and Fort Worth into the institutional bases of the NARA program.

THE MODERN ERA

The modern history of alcohol and drug treatment has been shaped by the therapeutic pluralism descended from the mutual-aid, asylum, and mental hygiene traditions; the growing prestige of clinical and basic medical research; the coexistence of public and private sectors of treatment; and an increasingly complex field of interorganizational relationships involving several layers of government and substantial fragmentation within each layer.

ALCOHOLISM TREATMENT

The influence of ALCOHOLICS ANONYMOUS can hardly be exaggerated. Whatever its therapeutic success—a point of warm debate among scholars—AA has profoundly affected the treatment of people now regularly known as alcoholics. AA's impact has been both ideological and institutional; that is, its promotion of "disease theory" within the mutual-aid tradition has changed how recent generations think about excessive or problem-causing alcohol consumption and treatment methods, and the penetration of policymaking bodies and treatment institutions by people recovering from alcoholism has shaped the funding and practices of treatment.

AA's impact was facilitated by the growing influence of the mental hygiene movement during the 1920s and 1930s, for AA provided the critical therapeutic bridge between the segregating institution and the community at large. This was recognized quickly by men like Clinton Duffy, the great "reform" warden of San Quentin, who encouraged the establishment of AA groups in his prison in 1942. Much early twelve-step work was done in U.S. county jails. Harvard psychiatrist Robert Fleming opined in 1944 that the prolonged institutionalization of alcoholics was no longer necessary; a week's medical care in a general hospital followed by community-based psychotherapy and AA participation was his new prescription. The growth of AA permitted the first substantial stirrings of community care since the Washingtonian Movement.

During the early 1960s, some state hospitals, particularly in Minnesota, incorporated recovering alcoholics and the principles of AA into their treatment programs. What became known as the Minnesota model of short-term inpatient care (usually 28 days) and subsequent AA fellowship and recovery-home living spread slowly but discernibly among private treatment providers such as the HAZELDEN Foundation, also in Minnesota, and the Mary Lind Foundation in Los Angeles. Across the country, local councils on alcoholism, dominated by people recovering from alcoholism and encouraged by the NATIONAL COUNCIL ON ALCOHOLISM AND DRUG DEPENDENCE and the National Institute of Mental Health (NIMH, created in 1946, was an ardent promoter of community psychiatry), began to press states and localities for outpatient clinics, diversion of alcoholics from jail, and other methods consistent with the traditions of mutual aid and mental hygiene. Even so, treatment resources for alcoholics did not expand dramatically. A survey in 1967 found only 130 outpatient clinics and only 100 halfway houses and recovery homes dedicated to serving alcoholics. Alcoholics continued to be barred from most hospital emergency rooms.

All this advocacy and organizing activity were propelled by the concept of "alcoholism as a disease," a proposition given its most systematic modern exposition by E. M. Jellinek in *The Disease Concept of Alcoholism* (1960). Jellinek was more provisional in his use of the term than most of his readers appreciated, but he understood the important strategic value of such a claim. In the first instance, the language of disease challenged the legal and correctional system's jurisdiction over alcoholics; in addition, it provided a rationale for the increased availability of services for alcoholics within established medical facilities and under the aegis of public health. Jellinek was widely read in the literature of the earlier inebriate asylum movement, and although he disparaged its science he understood and sympathized with its aims. He fully understood that whatever its equivocal status as scientific truth, the assertion that alcoholism is a disease carries important implications for treatment policies.

Several important court decisions in the 1960s endorsed the view that alcoholism was a disease; in 1967, a presidential commission on law enforcement concluded that it was both ineffective and inhumane to handle public drunkenness offenders within the criminal-justice system and recommended creating a network of detoxification centers instead. In 1970, Congress passed the Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment and Rehabilitation Act (the "Hughes Act"). Senator Harold Hughes, a former governor of Iowa, was a recovering alcoholic. A persuasive speaker, Hughes became the conscience of the Congress in developing support for a more humane and decent response to people with alcoholism and related problems. He was supported in these efforts by Senator Harrison Williams, Congressman Paul Rogers, and several advocacy groups led by the National Council on Alcoholism and the North American Association on Alcohol Problems. While Hughes's early efforts had been supported by President Lyndon Johnson and Assistant to the President Joseph Califano, it was President Richard M. Nixon who signed the legislation establishing the NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM (NIAAA). This legislation made federal funds available for the first time specifically for alcoholism treatment programs.

The Hughes Act accomplished three goals of the modern alcoholism treatment movement. First, it effectively redefined alcoholism as a primary disorder, not a symptom of mental illness. Second, and based on this distinction, it created the federal agency— NIAAA—that would not be dominated by the mental-health establishment competing for the same resources. Finally, and of great practical importance, the Hughes Act established two major grant programs in support of treatment. One authorized NIAAA to make competitive awards

(grants and contracts) directly to public and nonprofit agencies; the other was a formula-grant program, which allocated money to states based on a formula accounting for per capita income, population, and demonstrated need.

NIAAA aggressively sought state adoption of the model Uniform Alcoholism and Intoxication Treatment Act, first drafted in 1971 by the National Conference of Commissioners on Uniform State Laws. Section 1 of the Uniform Act, as it was known, stated that "intoxicated persons may not be subject to criminal prosecution because of their consumption of alcoholic beverages but rather should be afforded a continuum of treatment." By 1980, thirty states had adopted some version of the Uniform Act, thereby decriminalizing public drunkenness.

The thrust of federal and state grant making was to create an effective system of community-based alcoholism treatment services. This occurred in tandem with the deinstitutionalization process that was rapidly depopulating state mental hospitals. Although we customarily think of deinstitutionalization as affecting only the mentally ill, in fact it had an important impact on alcoholics. In 1960, a decade before deinstitutionalization began in earnest, thirty-six states had provisions specifically for the involuntary hospitalization of "alcoholics," "habitual drunkards," and "inebriates." In addition, many states had voluntary-admission statutes. By the mid-70s, however, these laws were history. Prepared or not, local communities had to provide.

The alcoholism-treatment field was not static during the 1980s. The federal "block grant system," stringent drunk-driving laws, and the rise of EMPLOYEE ASSISTANCE PROGRAMS (EAPs) and insurance coverage for treatment, all important developments, will be discussed following a description of the modern era of drug treatment.

DRUG TREATMENT

Even by the late 1950s, the tough law, antimaintenance consensus of an earlier era of drug control and treatment was breaking down. A joint report of the American Bar Association and the American Medical Association in 1958, finally published in 1961, cautiously favored outpatient treatment and limited opioid maintenance as alternatives to "threats of jail or prison sentences." In 1962, appealing to disease theory, the U.S. Supreme Court struck down a California statute that made drug addiction *per se* a crime. Medical treatment, not the "cruel and unusual punishment" of incarceration, was the Court's *desideratum*. In 1963, the President's Advisory Commission on Narcotic Drug Abuse made substantially similar recommendations.

It was the experimental success of METHADONE MAINTENANCE that finally altered the discussion of opioid maintenance. Methadone, a synthesized drug with opioid properties, was invented by German pharmacologists during World War II and had been used at the U.S. PUBLIC HEALTH SERVICE HOSPITAL at Lexington to block addicts' withdrawal symptoms. In 1963 and 1964, with the support of the prestigious Rockefeller University, medical researchers Vincent Dole and Marie Nyswander began to study its wider use in the treatment of heroin addiction. Their research proceeded despite opposition by the federal Bureau of Narcotics, and was first published in 1965. The remarkable changes they observed in their patients soon were replicated by other scholars. Methadone maintenance attracted considerable notoriety and generated new enthusiasm for maintenance as a strategy of treatment.

Methadone maintenance did not become widespread overnight, however, and it has never been without controversy. The most fundamental criticism of maintenance has always been that it presumes "incurability," encourages users to continue to rely on a narcotic medication, and thereby undermines abstinence-based approaches. During the 1960s, and especially during the 1970s, when methadone maintenance programs expanded dramatically, this criticism came mainly from two sources: (1) abstinence-based programs run by recovering addicts more or less in the mutual-aid tradition and (2) minority poverty activists who saw in methadone a palliative strategy to treat what they saw as a symptom of economic deprivation without addressing its causes.

Opposition from those working in the mutual-aid tradition came chiefly from veterans of THERAPEUTIC COMMUNITIES inspired by Synanon (established in Southern California in 1958) and Daytop Village (opened in New York City in 1964). While most therapeutic communities saw addiction primarily as a result of characterological deficits and immaturity, some drew financial support from

the Office of Economic Opportunity (OEO), the short-lived, principal arm of the War on Poverty, and relied on an analysis of heroin addiction that located its social sources in adaptations to poverty. This was an important theme of much scholarship on addiction during and after the late 1950s. In this analysis, still vital today, no form of treatment is effective without job and community development to support aftercare and prevent relapse. Descending from the mental hygiene tradition, this view provided a rationale for great skepticism about any narrow medical approach that was proclaimed as a "solution" rather than as a first step. There was (and remains) no inherent contradiction between maintenance and antipoverty strategies, and many workers in antipoverty programs embraced methadone as a viable and useful treatment. But many did not, and the result was an uneasy pluralism in drug-treatment approaches. In 1966, when New York City launched a major expansion of treatment for drug addiction, it chose to make drug-free therapeutic communities the centerpieces of its effort.

The middle to late 1960s were marked by a modest expansion of publicly supported programs for drug addiction, characterized by competition among a variety of distinct and sometimes incompatible treatment philosophies: therapeutic communities; methadone maintenance programs; compulsory treatment with prolonged residential components; twelve-step programs; overtly religious programs; and a number of traditional mental-health approaches offering detoxification followed by supportive psychotherapies.

Despite the variety of approaches, accessibility to voluntary treatment remained limited throughout the 1960s. In 1968, NIMH undertook a survey to identify every private or public program focused on the treatment of drug addiction in the United States; it located only 183. Most of these were in New York, California, Illinois, Massachusetts, Connecticut, and New Jersey. Of these, 77 percent had been open for less than 5 years. Only the federal hospitals at Lexington and Fort Worth had been in operation for 20 years or more.

In addition to establishing the federal civil commitment program, the Narcotic Addict Rehabilitation Act of 1966 authorized NIMH to make grants to establish community-based treatment programs. The first of these were awarded in 1968; they provided federal support for therapeutic communities and methadone maintenance. This expansion of treatment capacity was also notable for its attention to problems associated with a variety of drugs. It came at a time of sharp increase in marijuana use among middle-class youth, an epidemic of amphetamine use, growing experimentation with LSD, and media preoccupation with the counterculture, or the "youth revolt." Thus, the political urge to provide treatment was fueled by two enduring concerns of Americans—unconventional and disorderly behavior by young people and minority group members; and the connection between drug use and crime. Anything that might work was tried.

The administration of President Richard M. Nixon took office in 1969 and made the connection between drugs and crime a priority, concentrating first on law enforcement, federal legislation (the CONTROLLED SUBSTANCES ACT of 1970), and a reorganization of federal enforcement agencies. In 1970, while the administration was beginning to consider the role of treatment in its overall strategy, heroin use among service personnel in Vietnam captured media attention. In response, on June 17, 1971, Nixon declared a War on Drugs and created, by executive order, the SPECIAL ACTION OFFICE FOR DRUG DBUSE PREVENTION (SAODAP) within the executive office of the president. He appointed as director Dr. Jerome H. Jaffe, a psychiatrist and pharmacologist from the University of Chicago and the director of the Illinois Drug Abuse Programs. SAODAP was the first in a two-decade series of differently named White House special offices concerned with the drug problem; Jaffe was the first in a series of so-called Drug Czars (though the title might most appropriately fit Harry ANSLINGER, autocratic boss of the Bureau of Narcotics for over 30 years.)

The creation of SAODAP marked the federal government's first commitment to make treatment widely available. Indeed, SAODAP's goal was to make treatment so available that addicts could not say they committed crimes to get drugs because they could not obtain treatment. Over the next several years, a variety of community-based programs were initiated and/or expanded. The major modalities were drug-free outpatient programs, methadone maintenance, and therapeutic communities. SAODAP deliberately deemphasized hospital-based programs, allowing the civil commitment program under NARA to wither away. Even so, the need to expand treatment for the Veterans Administration (VA) resulted in funding VA hospitals to

use their beds for both detoxification and rehabilitation. SAODAP fully supported methadone maintenance, regarded as experimental by NIMH and federal law-enforcement agencies, and became a focal point of controversy as it presided over the dramatic growth of methadone programs beginning in the early 1970s. Treatment within the military also was legitimized as an alternative to court martial.

SAODAP was given a legislative basis in 1972. The same legislation, the Drug Office and Treatment Act, also created a formula grant program for drug treatment comparable in intent to that for alcoholism treatment. The legislation required the production of a written National Strategy, and authorized establishment of the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA), analogous to NIAAA. Like NIAAA, NIDA was lodged within NIMH.

During its first two years, SAODAP directed an unprecedented expansion of treatment. In early 1971 there were 36 federally funded treatment programs in the United States. By January 1972 there were 235, and by January 1973, almost 400. For a brief, 3-year period, the federal resources allocated to treatment, prevention, and research exceeded those allocated to law enforcement, actually comprising two-thirds of the drug resources in the 1973 federal budget.

In 1973, Dr. Robert Dupont, also a psychiatrist, succeeded Jaffe at SAODAP. Dupont had established and directed a treatment program in Washington, D.C., and had extensive experience with methadone treatment. He extended the work of SAODAP and then provided for continuity of policy when he became the first director of NIDA.

During the administration of President Gerald R. Ford (August 1974–January 1977), the sense of urgency about drug problems declined. This was not due to indifference; it reflected a belief that the metaphor of war was not appropriate to a problem that might be controlled but was unlikely ever to be eliminated. The recent lesson of Vietnam—that wars must be quickly won to be popular—was not lost on Ford's advisors. Thus, Ford did not appoint a Drug Czar, leaving coordination of drug activities to a unit within the Office of Management and Budget. There were no sharp changes in policy, but the treatment budget was substantially reduced from the highwater mark of the Nixon era.

The administration of President Jimmy Carter heightened the expectations of those interested in expanded and improved treatment. One of Carter's close advisors, Dr. Peter Bourne, was a psychiatrist who had established treatment programs in Georgia and who had worked briefly in SAODAP during the Nixon administration. Bourne enjoyed more White House influence than any previous presidential advisor on drug issues. However, Bourne resigned in July 1978, and in the wake of his resignation, drug issues resumed their low profile. Resources for treatment from 1978 to 1980 were stagnant despite an unprecedented inflation rate.

Measured in 1976 dollars, the level of federal support for treatment was cut almost in half between 1976 and 1982. The Ford, Carter, and Reagan administrations all presided over this decline. At the same time, as the result of the impact of inflation on the cost of state and local government, these jurisdictions also curtailed their support, thus aggravating the impact of federal reductions.

However, the Reagan administration was ideologically different from its predecessors—it was characterized by considerable skepticism about federal activism in general and about the efficacy of drug treatment in particular. Although it increased resources for law enforcement and supply control and introduced a stringent policy of ZERO TOLERANCE that filled American prisons and newly popular (though hardly innovative) therapeutic boot camps with drug offenders, the Reagan administration downplayed treatment in favor of prevention—especially First Lady Nancy Reagan's "Just Say No" campaign and the president's public advocacy of widespread drug testing of employees in industry and government. The 1980 reorganization of the federal block grant program that supported both alcohol and drug treatment combined these funds into an Alcohol, Drug Abuse and Mental Health Services (ADMS) block grant and turned these funds over to the states. In the process, overall funding was reduced from 625 million to 428 million dollars and federal oversight was virtually abandoned. After 1984, federal regulation required that a certain percentage of these funds be spent on prevention rather than treatment. The Institute of Medicine estimated that the proportion of the ADMS block funds available to support drug treatment fell from 256 million dollars in 1980 to 93 million dollars in 1986—and this estimate did not account for inflation.

In spite of the Reagan administration's lack of interest in drug treatment, congressional interest

was rekindled. It was apparent by 1984 that HIV was being transmitted among drug injectors and by drug injectors to others, especially their female partners and their fetal young. Crack, an extremely potent and inexpensive form of smokable cocaine, was being aggressively marketed in areas of concentrated poverty, although it took the deaths of several prominent athletes, particularly Len Bias, a first-round draft choice of the Boston Celtics, to pique concern with the growing use of cocaine. Prodded by Congress, the second Reagan administration, in its closing years, did increase funding for both research and treatment. However, according to the Institute of Medicine, these increases did not compensate for the effects of previous budget cuts and inflationary erosion. Adjusted for inflation, public funding for drug treatment in 1989 (the last Reagan budget) was substantially below the level of 1972 through 1974, the opening years of Nixon's War on Drugs.

Even so, the Reagan administration retained its emphasis on law enforcement and prevention. To better focus on prevention, in 1987 it created the Office for Substance Abuse Prevention (OSAP), placing it within the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA). Most prevention activities carried out by the National Institute on Drug Abuse (NIDA) were transferred to OSAP, the first director of which was Dr. Elaine Johnson.

In 1989, President George H. Bush reinvigorated the position of drug czar when he appointed Dr. William Bennett, former secretary of education in the Reagan administration, to head his new White House drug policy office, the Office of National Drug Control Policy (ONDCP). ONDCP was charged with coordinating demand-side (prevention and treatment) and supply-side (law enforcement) matters relating to drugs. There were increases in resources for treatment—and even more substantial increases in law-enforcement efforts. Although Bennett had recruited a noted drug-abuse scholar, Dr. Herbert Kleber, as his deputy for demand-side activity, the ONDCP chief and his staff remained skeptical about the value of treatment, continuing the decade-long policy of emphasizing prevention and law enforcement.

Later in 1989, much of the authority and funding for drug treatment was transferred from NIDA to another new agency created within ADAMHA, the Office for Treatment Improvement (OTI). Dr. Beny J. Primm, a major figure in drug treatment, was recruited to organize OTI and to be its first director. OTI was given responsibility for oversight of the block (formula) grant for drug and alcohol treatment and prevention and was given new authority and budget resources to make grants for treatment-demonstration projects.

In 1992, Congress decided that the placement of OTI and OSAP within ADAMHA, which also housed NIDA, NIAAA, and NIMH, was leading to conflicts between the missions of research and those of treatment and prevention. In still another reorganization, the three research institutes—NIDA, NIAAA, and NIMH—were transferred to the National Institutes of Health (NIH), and the remaining service functions were incorporated into a new agency, the SUBSTANCE ABUSE AND MENTAL HEALTH SERVICES ADMINISTRATION (SAMHSA). SAMHSA was composed of three centers: the Center for Substance Abuse Prevention (CSAP), consisting primarily of the former OSAP; the Center for Substance Abuse Treatment (CSAT), consisting primarily of the former OTI; and the Center for Mental Health Services (CMHS), consisting of the service-demonstration grant projects that were formerly within NIMH.

Succeeding President Bush in 1992, President Bill Clinton appointed Dr. Lee Brown as his Drug Czar. Brown, a criminologist by academic training, had been a police chief in New York and Texas. Although there were some signs within the administration that drug treatment was understood to be an important part of attacking persistent joblessness and welfare dependency, the early Clinton budgets made only slight shifts in resource allocation. Further, as Clinton's health-care reform, welfare reform, and crime and employment strategies became hostage to management of the national budget deficit and partisan politics, no major initiatives specifically on drug treatment were introduced during the first two years he was in office. Some provisions for more treatment within the criminal-justice system were part of the original crime bill. As a result of the recession of the early 1990s, and faced with the necessity of accommodating in their jails and prisons huge numbers of drug offenders incarcerated on mandatory sentences, states and counties also failed to restore the support an earlier era provided for treatment. In some cases, they retrenched considerably. In 1996, Brown was succeeded as Drug Czar by General

Barry McCaffrey. Although McCaffrey signaled an early intent to shift federal resources toward the treatment of America's "three million hard-core users" (as he put it during his confirmation hearing), his performance in office took quite a different turn. By 1998, it was clear that McCaffrey's principal concern was interdiction, especially in Mexico, and his budgets reflected this continuing emphasis. Although the 1999 federal drug budget included a $143 million increase in the federal block grant for drug treatment, two-thirds of the funds remained committed to supply reduction.

A TWO-TIERED SYSTEM

Beginning in the 1970s and promoted by NIAAA, NIDA, and a few insurance industry leaders like The Travelers, health insurance policies began to provide coverage for the treatment of alcohol and drug dependence. Sometimes this was the result of labor negotiations; sometimes it was the result of state insurance commission mandates for its inclusion. In response to the availability of support, private hospitals (both nonprofit and for-profit) expanded their treatment capacities dramatically. There had been no such growth in the private-treatment sector since the boom of the inebriate asylum era.

Commonly, treatment programs within the private sector were based on the Minnesota model, emphasizing twelve step principles and employing recovering people. Such programs typically consisted of a brief period of inpatient detoxification followed by several weeks of inpatient rehabilitation. Twenty-eight days was such a common duration of inpatient care that the programs often were referred to as 28-day programs. The posthospital phase of treatment usually consisted of participation in AA, Narcotics Anonymous, or Cocaine Anonymous.

Such programs—often called chemical-dependency programs because they admitted people with drug *and* alcohol problems—catered almost exclusively to those with health insurance. (In many instances, they represented important profit centers for medical institutions needing to subsidize financial losses from other services, like emergency rooms.) Those without insurance either had no access to treatment or made use of the network of publicly supported programs—a network that became increasingly thin during the 1980s and increasingly under pressure to find sources of funds other than public grants and contracts and payments from medical programs for the indigent (such as Medicaid). Sliding fee scales became more commonly used, and in some places scarce public treatment slots were absorbed by fee-paying drinking drivers mandated to treatment by stricter penalties for drunk driving and more systematic enforcement of such laws.

The growth of the private sector was spurred as well by EMPLOYEE ASSISTANCE PROGRAMS (EAPs), efforts to intervene in alcohol and/or drug problems at places of employment. This strategy goes back at least to the Washingtonian movement, but formal EAPs date from the 1940s. Their ranks swelled during the 1970s and 80s. Generally, EAPs referred people with more serious alcohol and drug problems to formal—usually private—treatment programs, which were paid primarily by fees derived from third-party payers, such as insurance companies, who in turn derived their funds from policies paid for or subsidized by employers. The sharply rising cost to employers of providing alcohol and drug treatment was a major factor in the rise of managed care, which was aimed initially at controlling the cost of mental health and alcohol and drug treatment. The major mechanism by which the managed-care industry addressed the cost of treatment was to challenge the practice of using several weeks of inpatient care as the initial phase of treatment for alcohol and drug dependence. In practice, treatment providers were told that inpatient treatment beyond a few days could not be justified and would not be paid for under the insurance policy.

The success of managed care in reducing costs by constraining the use of inpatient treatment resulted in a dramatic growth of managed-care organizations and an equally significant contraction and restructuring of the private alcohol and drug treatment system. By the early 1990s, a number of states had obtained federal permission to use managed-care approaches to contain the costs of treatment for individuals covered by federal programs like Medicaid. The future of funding for treatment, the various public grant and contract programs notwithstanding, is inseparable from the broader national debate on the financing of health care.

In 1990, the Institute of Medicine described U.S. treatment arrangements as a two-tiered system, comprised of public and private sectors, in which

the private sector served 40 percent of the patients but garnered 60 percent of total treatment expenditures. Although the ratio of patients to revenues cannot be known for earlier eras, this two-tiered structure is a creature of the nineteenth century, when treatment was established both as a public good and a commodity. Barring some revolution in the organization of U.S. health care, this is unlikely to change soon. What remains to be seen is what the balance of public and private treatment will be, what innovations or reinventions will be born of financial necessity, or as the result of homeless addicts and a groaning correctional system. History allows us to predict the likely questions, but it is not a very reliable guide to specific answers.

(SEE ALSO: *Disease Concept of Alcoholism and Drug Abuse; Temperance Movement; Treatment Types; U.S. Government: Drug Policy Offices in the Executive Office of the President; U.S. Government: The Organization of U.S. Drug Policy*)

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JIM BAUMOHL
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TREATMENT IN THE FEDERAL PRISON SYSTEM The federal prison system of the United States has made repeated efforts to treat drug-abusing prisoners. The issue was first raised in 1928 by the chairman of the Judiciary Committee of the U.S. House of Representatives. He reported that the three then-existing federal penitentiaries—Atlanta, Leavenworth, and McNeil Island— held 7,598 prisoners, 1,559 of whom were

"drug addicts." To deal with these prisoners he called for a "broad and constructive program in combatting the drug evil," and he recommended the establishment of special federal "narcotics farms" for the "individualized treatment" of drug-abusing prisoners. He hoped that there would become institutions that "will reduce and also prevent crime . . . and greatly alleviate the suffering of those who have become addicted."

In 1930, the U.S. Bureau of Prisons (BOP) was established to handle the burgeoning population of federal prisoners, caused mainly by the enforcement of PROHIBITION. The BOP's first directorate was eager to launch special programs for drug-abusing prisoners, but many in Congress and elsewhere believed that prisons should have little or no direct role in treating drug-abusing offenders. A compromise was struck. The U.S. Public Health Service (USPHS) was authorized to establish and administer two hospitals that would offer state-of-the-art drug-abuse treatment, and the BOP was permitted to freely assign addict prisoners to the facilities. The first USPHS HOSPITAL opened in 1935 at Lexington, Kentucky; the second was opened in 1938 at Fort Worth, Texas.

REHABILITATION EFFORTS

In the 1960s, a broad consensus emerged that prisons should do whatever possible to rehabilitate drug-abusing inmates. In 1966, Congress passed the NARCOTIC ADDICT REHABILITATION ACT (NARA), which, among other initiatives, ordered in-prison and aftercare treatment for narcotic addicts who had been convicted of violating federal laws. Between 1968 and 1970, the BOP established NARA-mandated drug-treatment units within five of its prisons. In the 1970s, the BOP assumed direct control over both USPHS hospitals and began to develop an extensive network of programs for the treatment of drug-abusing prisoners throughout the system. In 1979, the BOP required the development of NARA-standard drug-treatment programs in all its prisons, publishing it *Drug Abuse Incare Manual*. In 1985, the BOP established a task force to evaluate the state of drug-abuse treatment programs within federal prisons. The review found that administrative problems had hampered the BOP's drug-treatment efforts. In response, in 1986, the position of chemical-abuse coordinator was established within each prison, and in 1988, the position of national drug-abuse coordinator was created to oversee drug-abuse treatment efforts throughout the federal prison system.

At the end of 1990, the BOP held some 59,000 prisoners. About 54 percent of federal prisoners were serving sentences for drug-related crimes. At the time of their admission, 47 percent of federal prisoners were classified as having moderate to serious drug-abuse problems. Under the BOP's classification scheme, a moderate problem designation indicates that the inmate's use of drugs or alcohol had negatively affected at least one "major life area"—school, health, family, financial, or legal status—in the two-year period prior to arrest.

In 1991, the BOP's drug-education program was required for all inmates with any history of drug abuse or drug-related crime. By the end of 1992, an estimated 12,000 to 15,000 federal inmates completed drug-education programs. Counseling services—ALCOHOLICS ANONYMOUS (AA), NARCOTICS ANONYMOUS (NA), group therapy, stress management, prerelease planning—were available on an ongoing basis at most federal prisons, and the BOP planned to make them available to inmate volunteers at all institutions at any time during their incarceration.

Transitional drug-abuse treatment services were being developed throughout the BOP. The administration of these services were divided into two six-month components, each of which included individual and family counseling, assistance in identifying and obtaining employment, and random urine testing. The first component was provided in the BOP's community corrections centers; the second component was provided as post-release aftercare, in conjunction with the Probation Division of the Administrative Office of the U.S. Courts.

To assess the effectiveness of its current multidimensional drug-abuse treatment efforts, the BOP has begun a major evaluation of these programs that will analyze data on both in-prison adjustment and postrelease behavior for up to five years after release.

(SEE ALSO: *Coerced Treatment for Substance Offenders; Prisons and Jails, Drug Treatment in*)

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JOHN J. DIIULIO, JR.

TREATMENT OUTCOME PROSPECTIVE STUDY (TOPS) This is a prospective clinical, epidemiological study of clients who entered drug-abuse treatment programs from 1979 to 1981. During the course of TOPS, 11,182 clients were interviewed at admission to drug-abuse treatment by program researchers hired to work in assigned clinics and professionally trained and supervised by Research Triangle Institute (RTI) field staff. The interviews at admission covered demographics, history of drug use, treatment, arrest and employment behavior in the year prior to treatment, and status upon admission to treatment. The study was sponsored by the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) and by the RTI. The study population included 4,184 clients from 12 outpatient methadone programs, 2,891 clients from 14 residential programs, and 2,914 clients from 11 outpatient drug-free programs in 10 cities. Interviews with questions on behavior, services received, and satisfaction were collected by the program researchers every three months while clients remained in treatment. The self-report data were supplemented with data abstracted from the clinical and medical records of all clients selected for the follow-up, and questionnaires describing the treatment philosophy, structure, practice, and process were completed by counselors and program directors.

The follow-up data included interviews 1 and 2 years after treatment with 1,130 clients who were admitted in 1979; follow-ups 90 days and 1 year after treatment of 2,300 clients who entered treatment in 1980; and follow-ups 3 to 5 years after treatment of 1,000 clients who entered programs in 1981. Professional field interviewers hired, trained, and supervised by RTI field staff were able to locate and interview between 70 and 80 percent of the clients selected for these interviews.

TOPS has resulted in a substantial body of important knowledge about drug-abuse treatment and treatment effectiveness. The client populations of outpatient METHADONE PROGRAMS, long-term residential programs, and outpatient drug-free programs who participated in TOPS differed on many sociodemographic and background characteristics. The residential clients were significantly more likely to report multiple use of drugs, more drug-related problems, suicidal thoughts and attempts, heavy drinking, predatory crimes, and less full-time employment compared to the methadone clients. Outpatient drug-free clients were more likely than methadone clients to report drug-related problems, suicidal thoughts or attempts, predatory crimes, and heavy drinking, but they were less likely than residential clients to use multiple drugs. These results demonstrated that each type of program served very different, important segments of the drug-abusing population. The high rates of self-referrals to methadone (48%) and criminal-justice referrals to residential and outpatient drug-free treatment (31%) suggest differences in clients' motivations for seeking treatment and, consequently, differences in retention, services received, and outcomes.

The drug-abuse patterns reveal the differential concentration of types of drug abusers across the major categories. Clients on methadone were primarily (52%) traditional heroin users who used only cocaine, marijuana, and alcohol, in addition to heroin. One in five of these clients, however, used heroin and other narcotics, as well as a variety of non-narcotic drugs. The remaining quarter of clients on methadone were classified as former daily users who had histories of regular use but did not use heroin on a weekly or daily basis in the year before treatment. Residential clients had diverse patterns of use, and the majority of outpatient drug-free clients were users of alcohol and marijuana (36%) or single non-narcotics users (22%).

Symptoms of depression are very commonly reported by clients entering drug-abuse treatment programs. Overall, about 60 percent of TOPS clients reported at least one of three symptoms of depression at intake: nearly 75 percent of the women under 21 years of age reported one or more symptoms of depression. Other results suggest that the duration of regular drug use and the number of prior treatment episodes are important indicators of the effectiveness of any single treatment episode; clients with lengthy drug-abuse or drug-treatment histories have poorer prognoses.

Clients who have come into treatment by way of the criminal justice system do as well or better than other clients in drug-abuse treatment. Formal or informal mechanisms of the criminal justice system appear to refer individuals who had not previously been treated and many who were not yet heavily involved in drug use. Involvement with the criminal justice system also helps retain clients in treatment up to an estimated six to seven additional weeks. Drug abuse treatment programs vary in the nature and intensity of the treatment services provided, the types of therapists and therapies provided, the average length of stay, and the inclusion or exclusion of aftercare.

The study of the treatment process in TOPS programs focused on many important aspects of the structure, nature, duration, and intensity of drug-abuse treatment. Descriptions of aspects of the treatment process were developed from clients' self-reports of needs for treatment services, services received, and satisfaction, combined with abstractions of clinical and medical records and descriptions of programs by counselors and directors. The outpatient methadone and outpatient drug-free treatment programs had budgets per slot of approximately 2,000 dollars per year. Therapeutic communities had an average expenditure of 6,135 dollars per bed.

The number of available services (medical, psychological, family, legal, educational, vocational, and financial services) varied during the years 1979 to 1981. Fewer services appeared to be available in the later years of the study. The proportion of clients in residential treatment programs who received family, educational, and vocational services decreased noticeably during the three-year period. During this same period, the clients' demands for services increased. Programs in TOPS appeared to focus on the client's primary drug of abuse rather than addressing the client's multiple drug use, drug-related problems, and social and economic functioning. Low-dose methadone (69% of the clients admitted were initially treated with less than 30 mg of oral methadone daily) was the most common pattern of methadone treatment in the programs participating in TOPS.

In TOPS, multiple measures of treatment outcome were necessary to describe changes in the client's ability to function in society after treatment. In general, clients who remained in treatment at least three months had more positive post-treatment outcomes, but the major changes in behavior were seen only in those who remained in treatment for more than twelve months. Analyses of the TOPS data show that the post-treatment rate of daily heroin, cocaine, and psychotherapeutic-agent use among clients who spent at least three months in treatment was half that of the pretreatment rate. The post-treatment rates of weekly or more frequent use for clients who stayed in treatment at least three months were 10 to 15 percent lower than the rates for shorter-term clients. The results showed that time spent in treatment was among the most important predictors of most treatment outcomes. Stays of one year or more in residential or methadone treatment, or continuing maintenance with methadone, produced significant decreases in the odds of a client using heroin in the follow-up period. Clients in TOPS also reported a substantial decrease in depression symptoms during the years after treatment.

Analyses of the effects of treatment on behavior have focused on reductions in predatory crime and the costs associated with crime. The assessment of the benefit/cost ratio indicates that substantial benefits are obtained in reductions of crime-related costs regardless of the measures used within the year after treatment. Reducing transmission of the AIDS virus would increase the benefit portion of benefit/cost ratio even more.

(SEE ALSO: *Drug Abuse Treatment Outcome Study; Treatment Alternatives to Street Crime; Treatment Types*)

ROBERT HUBBARD

TREATMENT PROGRAMS, CENTERS, AND ORGANIZATIONS: AN HISTORICAL PERSPECTIVE The development of treatment programs for the age-old problem of drug and alcohol abuse has been a fairly recent phenomenon. Most formal treatment programs were founded in the latter half of the twentieth century; the mid-1960s were a period of significant focus on U.S. social programs. Growing out of President Lyndon B. Johnson's Great-Society strategy was a new way of viewing the community's capacity to take ownership of its social problems, develop collaborative strategies, and heal its own wounds. Toward that end, a new lexicon emerged—*community-based*, *storefront*, and *streetworker*—to identify but a few terms. The programs that evolved from this movement employ a variety of treatment philosophies; some treatment centers target a specific gender, ethnic, or age group. This article presents an overview of some significant drug and alcohol abuse treatment programs, centers, and organizations.

HAZELDEN FOUNDATION

Hazelden (PO Box 11, CO3, Center City, MN 55012-0011; 800-257-7810) was established in 1949; it was one of the pioneering programs that developed the approach to treatment that is now widely known as the MINNESOTA MODEL. Today, the private, nonprofit Hazelden Foundation operates residential rehabilitation programs (main headquarters in Center City, Minnesota, with additional facilities in Illinois, Minnesota, New York, and Florida) providing Minnesota Model treatment for thousands of adult alcoholic, drug-dependent men and women each year. Hazelden offers accredited distance learning programs for addiction studies, and in 2000, granted its first master of arts degrees in Addiction Counseling.

Residential treatment consists of an open-ended stay lasting an average of twenty-eight days. Primary rehabilitation is done by a staff of trained counselors who are also working their own programs of recovery. During the first week of primary rehabilitation, the staff concentrates on problem identification, guided by assessments of psychological, spiritual, health, social activities, and chemical-use profiles. After the client's problem is identified, an individual treatment plan is formulated both for and with the client. Goals, objectives, and methods are identified in the treatment plan and progress in meeting these expectations is monitored. Treatment at Hazelden is integrated with the principles of ALCOHOLICS ANONYMOUS.

SYNANON

Founded in 1958 by Charles E. Dederich, Synanon pioneered a breakthrough approach to the treatment of drug dependence. Using some of the approaches he had personally experienced in ALCOHOLICS ANONYMOUS, a mixture of self-reliance and Buddhist philosophies, and his own bombastic interpersonal style, Dederich shaped a self-help organization that grew from a small storefront in Santa Monica, California, to over 2,000 members in multiple residential settings across the United States by the early 1970s. The organization amassed considerable wealth, and as it became more self-sufficient, Synanon members began to consider their process a religion. By the mid-1970s, the organization was engaging in controlling and even violent practices against its members, including forced vasectomies and abortions. The whole system also began to have increasingly violent interactions with outsiders—including intimidation and actual physical assaults. The organization, so lauded in the press during its early years, became an object of national criticism. Then Dederich reversed his earlier position of shunning chemicals and began to drink. In 1978, he was indicted for conspiracy to commit murder, and the court instructed him to vacate leadership. A small cadre of members still venerated him until his death in 1997. Synanon ceased its drug-treatment programs in the 1980s and is no longer involved in any human-service business.

Controversies aside, the methodologies developed and refined by Synanon became the precursor for the drug-free THERAPEUTIC COMMUNITY approach. This strategy has proven significantly effective for both ADOLESCENTS and adults, regardless of the types of drug they use.

The salient ingredients pioneered at Synanon remain fundamentally intact in drug-free therapeutic communities in the United States and elsewhere. These fundamental ingredients fall into four major categories: (1) behavior management and behavior shaping, (2) emotional and psychological life, (3) ethical and intellectual development, and (4) work and vocational life. Within each of these categories, elaborate sets of techniques use deliber-

ate but artful dissonance and confrontation as major tools for changing behavior.

DAYTOP VILLAGE (ALSO DAYTOP FOR A DRUG FREE WORLD)

Daytop Village, Inc. (54 West 40th Street, New York, NY 10018; 212-354-6000), which began in 1964, had its roots in a research project conducted by Alex Bassin and Joseph Shelly of the Probation Department of the Second Judicial District of the Supreme Court of New York. They were awarded a grant from the National Institute of Mental Health to initiate a new approach for treating drug-addicted convicted felons. This new approach would offer an alternative to incarceration, in the form of a residential treatment center modeled roughly after Synanon. The founders of Daytop Village included Dr. Daniel Casriel, David Deitch, a former Synanon director, and Monsignor William B. O'Brien, a Roman Catholic priest.

Daytop's primary effort was long-term residential treatment, but by the mid-1970s, day-care models had been implemented, as well as discrete adult and adolescent programs. During the mid-1980s, Daytop expanded its program to include working adults—both after work and during special employer-contracted daytime hours. In the late 1980s Daytop instituted special programs for pregnant women.

The basic assumption underlying the Daytop treatment system is that drug dependence is a mix of educational, biomedical, emotional, spiritual, and psychosocial factors—and the treatment environment must attend to all of these. This philosophy serves as the basis for many successful treatment programs.

MARATHON HOUSE

In 1966, streetworkers for Progress for Providence (Rhode Island) began to acknowledge a growing community presence of HEROIN, heroin dealers, and addicts. Representatives from this organization pursued training with Daytop Village, seeking technical assistance to establish a Providence-based initiative. Marathon House, the first New England-based THERAPEUTIC COMMUNITY, was established in Coventry, Rhode Island, in October 1967.

In successive years, additional facilities were opened in Massachusetts and Connecticut. A facility for ADOLESCENTS in Middletown, Rhode Island, began operating in 1970. While relatively short lived, it laid the groundwork for those modified therapeutic communities Marathon currently operates throughout New England. In February 1971, Marathon acquired a historically significant property in Dublin, New Hampshire, the Dublin Inn. In the 1990s, this facility became the center for three distinct Marathon programs: the original New Hampshire adult therapeutic community, the Lodge at Dublin, a facility for male adolescents, and the Alcohol Crisis Intervention Unit, a small social-setting detoxification facility. In 1999, Marathon became an affiliate of Phoenix House.

PHOENIX HOUSE

Founded in 1967, Phoenix House (164 W. 74th Street, New York, NY 10023; 212-595-5810) was a second-generation THERAPEUTIC COMMUNITY (TC) program that developed from the treatment approach originated at SYNANON. Phoenix House provides drug-free residential and outpatient treatment for adults and adolescents, plus intervention and prevention services. Phoenix House operates programs in correctional facilities and homeless shelters. It is one of the largest nongovernmental, nonprofit drug-abuse service agencies and has a 1-800-COCAINE substance-abuse information and referral service.

HAIGHT-ASHBURY FREE CLINIC

The Haight-Ashbury Free Clinic (558 Clayton Street, San Francisco, CA 94117; 415-487-5632) was founded in June 1967 by David E. Smith, M.D., with the help of other physicians from the University of California Medical School at San Francisco and community volunteers to provide medical services for the waves of young people, known as hippies, who came to San Francisco during the "Summer of Love." These young people often lived in crowded, unhygienic conditions and were vulnerable to respiratory, skin, and sexually transmitted diseases. The Free Clinic offered an alternative to an established medical care system that members of the Counterculture saw as difficult to access, dehumanizing, unresponsive, and often judgmental about their nontraditional lives. The

clinic's philosophy included beliefs that health care is a right, not a privilege, and that it should be free and nonjudgmental.

The free clinic became a source of innovative drug-abuse treatment, where many health professionals received their early field training, and treatment approaches were developed for the DETOXIFICATION of OPIOID, SEDATIVE-HYPNOTIC, stimulant, and PSYCHOACTIVE drug abusers. Today, Haight-Ashbury Free Clinics, Inc., provides a full spectrum of community medical services to an ethnically mixed population of the working poor, the unemployed, and the HOMELESS.

GATEWAY FOUNDATION

In 1968, Gateway Houses Foundation was incorporated as a not-for-profit corporation and became the first THERAPEUTIC COMMUNITY in Illinois. Modeled on DAYTOP VILLAGE, it was established as a residential setting in which former drug addicts could help other drug abusers find a way to live drug-free, useful lives in the community.

The early years of treatment experience demonstrated that not all of those entering Gateway needed long-term residential treatment. Programs were devised or modified to fit the specific needs of the individuals served. The agency adopted the name Gateway Foundation in 1983 to better symbolize the services offered. To extended care (residential, long-term treatment), Gateway added outpatient (both intensive and basic), detoxification, and short-term treatment, as well as community-based EDUCATION and PREVENTION PROGRAMS.

The therapeutic community remains the core of Gateway's programs. Participation in TWELVE-STEP support groups are the client's mainstay during and after treatment. Gateway Foundation's successful treatment center within the Correctional Center of Cook County (the largest U.S. county jail) resulted in treatment programs for inmates in other Illinois and Texas correctional programs. Treatment for all Gateway clients includes work and social-skills development, continuing education, and employment counseling.

OXFORD HOUSE

The autonomous halfway-house movement of the 1990s, Oxford House, Inc., owes its momentum to J. Paul Molloy, who in 1975 established the first Oxford House in Silver Spring, Maryland. The stimulus for this first house was a decision by the state of Maryland to save money by closing a publicly-supported halfway house. The men living in it decided to rent and operate the facility themselves. Operated democratically, residents of the house determined how much each would have to pay to cover expenses, developed a manual of operations, and agreed to evict anyone who returned to substance use. When the first Oxford House found itself with a surplus of funds, the residents decided to use the money to rent another house and expand the concept. Each subsequent house followed suit. There are now separate houses for men and women. In 2000, there were approximately 350 houses in North America.

Volunteer medics sort through medicine donations to the Haight Ashbury Free Clinic, a clinic specializing in the treatment of young drug users. San Francisco, July 1967. (© Ted Streshinsky/CORBIS)

While not affiliated in any way with AA or NARCOTICS ANONYMOUS (NA), the principles of

these groups are integral to the operation of each Oxford House. Individuals can remain in residence as long as needed to become stably sober. The average length of stay is thirteen months.

Although a recovery house can be self-run and self-supported without being an Oxford House, if it wishes to affiliate, it must file an application for a charter with Oxford House, Inc. (9314 Colesville Road, Silver Spring, Maryland 20907). Oxford House, Inc., a nonprofit corporation, does not own property, but helps groups wanting to start a new house.

SECOND GENESIS, INC.

Second Genesis, Inc. (7910 Woodmont Avenue, Suite 500, Bethesda, MD 20814; 301-656-1545), is a long-term, residential and outpatient rehabilitation program for adults and teenagers with substance abuse problems. Founded in Virginia in 1969, under the direction of Dr. Sidney Shankman, Second Genesis is a nonprofit organization operating residential THERAPEUTIC COMMUNITIES and outpatient services that serve Maryland, Virginia, and Washington, DC. Second Genesis admits adults, women and their young children, and teenagers.

The Second Genesis residential program has been described as a school that educates people who have never learned how to feel worthy without hurting themselves and others. Through highly structured treatment, Second Genesis combines the basic values of love, honesty, and responsibility with work, education, and intense group pressure to help correct the problems that prevent people from living by these values. Discovering self-respect in a family-like setting, residents are taught to replace behavioral deficits and substance abuse with positive alternatives. The Mellwood House facility in Upper Marlboro, MD, provides residential treatment for women and their young children, offering children's services, vocational counseling, parenting classes, and anger management workshops. In 1998, Second Genesis opened adult and adolescent outpatient programs, providing group and individual therapy, educational services, and DWI/DUI counseling.

WALDEN HOUSE

Walden House (520 Townsend St., San Francisco, CA 94103; 415-554-1100) is a comprehensive THERAPEUTIC COMMUNITY (TC), which began in San Francisco, CA. It consists of residential facilities for adults and adolescents, a day treatment program, outpatient services, and a nonpublic school and training institute. Walden House is a highly structured program designed to treat the behavioral, emotional, and family issues of substance abusers.

The heart of the Walden House TC is a long-term residential treatment program, consisting of a series of phases from orientation to aftercare. Within the TC, all the household tasks, groups, and seminars promote responsibility and emotional growth. The activities are part of an integrated array of therapeutic experiences, in which residents continuously see themselves in a context of mutual support. The philosophy of Walden House emphasizes self-help and peer support.

Founded in 1969 by Walter Littrell as a response to the drug epidemic of the 1960s, Walden House has grown into one of the largest substance-abuse programs in California. The program pioneered the use of alternative treatments with substance abusers, for example, herbs, diet, and physical exercise. Walden House has designed many special programs to treat particular populations, including clients with AIDS, homeless people, minorities, pregnant women, mothers, and clients referred from the criminal-justice system as an alternative to incarceration.

OPERATION PAR

Operation PAR, Inc. (Parental Awareness & Responsibility) was founded in 1970 by Florida State Attorney James T. Russell, former Pinellas County Sheriff Don Genung, County Commissioner Charles Rainey, and Shirley Coletti, a concerned parent. In the years since its founding, PAR has developed one of the largest nonprofit systems of substance-abuse EDUCATION, PREVENTION, TREATMENT, and RESEARCH in the United States. At present, PAR operates more than twenty-five substance-abuse programs in nineteen locations in Florida. Operation PAR's THERAPEUTIC COMMUNITY (TC) has been in continuous operation since 1974. The program targets individuals who are severely dysfunctional and who exhibit antisocial behaviors as a result of substance abuse. The facility is an important alternative to incarceration for criminal courts throughout central Florida. Approximately 70 percent of clients

have histories of significant involvement with the criminal-justice system.

Overall services provided by PAR TC include individual and group counseling, counseling groups for special populations, AA and NA support groups, on-site educational services, vocational training and a job placement program, work experience, recreational therapy, and parenting therapy and classes. In April 1990, services were expanded to include residential living, called PAR Village, for the children of maternal substance abusers.

PROJECT RETURN FOUNDATION, INC.

Project Return Foundation, Inc. (10 Astor Place, 7th Floor, New York, NY 10003; 212-979-8800), a nonprofit, nonsectarian, multipurpose human-services agency, operates several New York City residential drug-free (RDF) THERAPEUTIC-COMMUNITY (TC) programs. The agency was founded in 1970 as a self-help and community center for substance abusers by two recovering addicts, Carlos Pagan and Julio Martinez. Project Return also operates a women's and children's treatment center, allowing children to remain with addicted mothers during treatment.

Under the leadership of president Jane Velez the agency diversified significantly. Project Return also operates an outreach, anti-AIDS education/prevention program, a medically supervised, drug-free outpatient program, and a modified TC-oriented health-related, facility for substance abusers who are HIV+ and symptomatic. The latter service is administered jointly by Project Return Foundation, Inc., Samaritan Village, and H.E.L.P., Inc. In total, nearly 1,000 men and women receive daily treatment and rehabilitative services through programs administered by Project Return Foundation, Inc.

All of Project Return's RDF TC programs are run according to the same clinical principles—they provide comprehensive, holistic, individualized treatment and rehabilitative services to the residents through interdisciplinary treatment teams. Interdisciplinary teamwork spans the entire length of stay in the TC programs, from admissions to discharge.

ABRAXAS

The Abraxas Foundation was started in Pennsylvania in 1973, in response to Requests for Proposals (RFP) from the Governor's Council on Drug and Alcohol Abuse. Abraxas's founder, Arlene Lissner, had been the deputy clinical director for the State of Illinois drug-abuse treatment system. There were two mandates to the RFP: (1) that a drug-treatment program be devised to directly serve the juvenile and adult justice system, and (2) that the program would utilize a then-abandoned U.S. forest-service camp, Camp Blue Jay, within the Allegheny National Forest. The original proposal stressed the development of a comprehensive program incorporating intensive treatment, education, and, of particular importance, a continuum of care to assist residents to reenter through regional reentry facilities. After an initial attempt to use only a behavioral approach, a THERAPEUTIC COMMUNITY (TC) model was implemented.

By 1988, all Abraxas facilities had focused their target populations solely on adolescents and had become gender specific. For example, Abraxas V in Pittsburgh was developed as an all-female residential facility. In 1990, an intensive project known as Non-Residential Care was developed to provide community-based transitional services to youngsters returning to Philadelphia after placement in state institutions. The success of this project led to its expansion to Pittsburgh. Inspired by the Non-Residential Care model, Supervised Home Services was developed later that year as a nonresidential reentry service for youngsters returning to Philadelphia from Abraxas's residential programs.

Education has been an integral part of the philosophy of treatment since Abraxas's inception. The Abraxas School, a private high school on the Abraxas I treatment campus, offers a full curriculum of courses and special educational services for the resident population. Alternative schools have been developed in Erie and Pittsburgh in recognition of the tremendous difficulty troubled adolescents have returning to public high schools. Abraxas has also extended its programming to include families of origin: The Abraxas Family Association meets in chapters throughout Pennsylvania and West Virginia to offer education, group counseling, intervention, and referral work to the families of clients.

INSTITUTE ON BLACK CHEMICAL ABUSE (IBCA)

Founded in 1975, the Institute on Black Chemical Abuse (2616 Nicollet Avenue S, Minneapolis,

MN 55408; 612-871-7878) is an open-membership organization that provides culturally specific programs and client services for the African-American community. IBCA defines cultural specificity as the creation of an environment that encourages and supports the exploration, recognition, and acceptance of African-American identity and experience, including the unique history associated with being African American in the United States and the role that racial identity plays in drug dependence. Programs are designed to address the devastating effects of the drug-abuse problem on this community. Services are provided in assessment and intervention for outpatient treatment and aftercare, black co-dependency issues, home-based support, and for pregnant women and young children.

IBCA's efforts in the community provide training and prevention resources to educate those who face the problems of substance abuse. The Technical Assistance Center (TAC) offers training workshops, program consultation, and resource materials on African Americans and substance abuse. TAC also educates and trains clergy members working with these issues in the community. The IBCA prevention programs have involved school and business leaders in social-policy programs aimed at establishing community awareness of substance-abuse issues; the Drug Free Zones program, in particular, has received national recognition.

JEWISH ALCOHOLICS, CHEMICALLY DEPENDENT PERSONS AND SIGNIFICANT OTHERS FOUNDATION, INC. (JACS)

JACS is a nonprofit, tax-exempt, volunteer membership organization located at 850 Seventh Avenue, New York, NY 10019; 212-397-4197. JACS was established as a result of work done by the Task Force on Alcoholism and Substance Abuse of the Federation of Jewish Philanthropies of New York (UJA-Federation).

JACS provides support programs and conducts retreats enabling recovering Jewish substance abusers and their families to enhance family communication, and reconnect with Jewish traditions and spirituality. The programs are designed to help participants find ways in which Judaism can assist their continuing recovery. Participants and rabbis explore the relationship between Jewish spiritual concepts and TWELVE-STEP PROGRAMS.

In addition to conducting retreats and support programs, JACS provides community outreach programs. These programs disseminate information to educate and sensitize Jewish spiritual leaders, health professionals, and the Jewish community about alcoholism and substance abuse, and about the effects of ALCOHOLISM and drug dependence on Jewish family life.

SOCIETY OF AMERICANS FOR RECOVERY (SOAR)

Society of Americans for Recovery (600 E. 14th Street, Des Moines, IA 50316; 515-265-7413) was founded by Harold E. Hughes, a former governor and senator from Iowa. It is a national grass-roots organization of concerned people whose aim is to prevent and treat dependence on alcohol and other drugs, and to educate the public about substance abuse and about its successful treatment. The organization sponsors regional conferences throughout the country and publishes a newsletter.

The organization lobbies to fight the stigma that society places on alcoholics and addicts, and it advocates and lobbies for more and better treatment. It also encourages people to learn more about addictions and recovery and to meet others who are active in communities on behalf of substance-abuse issues.

BETTY FORD CENTER

This eighty-bed hospital for recovery from chemical dependency was named in honor of President Gerald Ford's wife, who was treated successfully and who promotes such therapy. The center is located southeast of Palm Springs, California, on the campus of the Eisenhower Medical Center.

The staff at the center views ALCOHOLISM and other drug dependencies as chronic progressive diseases that will be fatal if they are not treated. The program at Betty Ford is designed so that patients learn to become responsible for their own actions and recovery. Because chemical dependency affects the family unit, the center has created the family-treatment program, a five-day intensive process that includes education and individual and group therapy. The center's staff also addresses the fact that women have traditionally been hidden chemically dependent people, so their treatments for women differ from those for men.

(SEE ALSO: *Alcohol- and Drug-Free Housing; Amphetamine Epidemics; Appendix III, Volume 4: State-by-State Treatment and Prevention Programs; Association for Medical Education and Research in Substance Abuse; Civil Commitment; Coerced Treatment; Ethnic and Cultural Relevance in Treatment; Ethnicity and Drugs; Halfway Houses; Jews, Drugs, and Alcohol; Lysergic Acid Diethylamide; Pregnancy and Drug Dependence; Prevention Movement; Prisons and Jails: Drug Treatment in; Sobriety; Substance Abuse and AIDS; Treatment/Treatment Types; Treatment Alternatives to Street Crime; Treatment, History of; Treatment In the Federal Prison System; Vulnerability as Cause of Substance Abuse: Race; Vulnerability as Cause of Substance Abuse: Sexual and Physical Abuse*)

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TREATMENT The following series of articles provides the reader with brief descriptions of some of the diverse ways that people with sub-

stance-related problems can be helped. It is organized into two subsections. *Treatment* consists of summaries of the common ways that problems relating to specific substances are currently treated. Different approaches are described for alcohol, cocaine, heroin, polydrug abuse, and tobacco. *Treatment Types* presents descriptions of distinct interventions that are applicable to dependence on a variety of drugs.

In practice, many treatment programs are hybrids, incorporating features from several distinct treatment modalities and adapting them to specific needs having to do with age, gender, ethnic, racial, and socioeconomic factors, provider preference, and the economic realities that govern delivery of treatment.

Neither of the sections is exhaustive. A variety of substance dependence interventions employed in other countries and by certain ethnic groups in the United States (such as sweat lodges among some Native American tribes) are not covered. Nevertheless, the entries included here should allow the reader to become reasonably familiar with what is considered mainstream treatment in the United States at the turn of the millennium.

TREATMENT

This section contains summaries of the common ways that problems relating to specific substances are currently treated. It is organized first by drug and then by treatment approach. Different approaches are described for *Alcohol*, *Cocaine*, *Heroin*, *Polydrug Abuse*, and *Tobacco*. The reader should also see the entries for each of these topics and the entries for *Barbiturates*, *Inhalants*, and *Nicotine* under their individual headings, and the section below entitled *Treatment Types*.

This section contains the following articles:

Alcohol Abuse: 2000 and Beyond;
Alcohol, An Overview;
Alcohol, Behavioral Approaches;
Alcohol, Pharmacotherapy;
Cocaine, An Overview;
Cocaine, Behavioral Approaches;
Cocaine, Pharmacotherapy;
Drug Abuse: 2000 and Beyond;
Heroin, Behavioral Approaches;
Heroin, Pharmacotherapy;
Marijuana, An Overview;
Polydrug Abuse, An Overview;
Polydrug Abuse, Pharmacotherapy;
Tobacco, An Overview;
Tobacco, Pharmacotherapy;
Tobacco, Psychological Approaches;
Twelve Step Facilitation (TSF).

Alcohol Abuse: 2000 and Beyond Every day, more than 700,000 people in the United States receive treatment for problems with alcohol use. Treatment can be behavioral therapy, or behavioral therapy in combination with medication. New therapies will likely take advantage of findings from neuroscience about alcohol's effects in the brain and include medications targeted at specific sites in the brain involved in the development of alcohol use problems.

BEHAVIORAL THERAPY AND ALCOHOLISIM TREATMENT

A broad range of psychological therapies currently are used to treat alcoholism. Many of these therapies have been in use for some thirty years. Others are more recent developments. Many older treatments for alcoholism were developed before modern standards of evaluating treatment outcomes were accepted in the alcohol field. Thus, the various approaches to treating alcoholism have different levels of scientific support for the effectiveness. Treatments that have been evaluated include client-treatment matching and professional treatments modeled on the twelve steps of Alcoholics Anonymous. Newer treatments that have been developed and evaluated include brief or minimal intervention, motivation enhancement therapy, and cognitive-behavioral therapy.

Brief or Minimal Intervention. One in five men and one in ten women who visit their primary care providers are at-risk drinkers or alcohol-dependent. Brief intervention, which is designed to be conducted by health professionals who do not specialize in addictions treatment, can help at-risk drinkers to decrease their risk and to motivate alcohol-dependent patients to enter formal alcoholism treatment. The main elements of brief intervention can be summarized by the acronym FRAMES: feedback, responsibility, advice, menu of strategies, empathy, and self-efficacy. Although

research has shown that brief interventions can be effective it has not yet been widely implemented.

Patient-Treatment Matching. Patient-treatment matching is using a patient's individual characteristics (such as gender, anger level, social functioning, and severity of alcohol dependence) to select an appropriate treatment therapy. A commonly held view in alcoholism treatment is that matching patients to treatments will improve treatment outcome. This view was supported by thirty small-scale research studies conducted during the 1980s that found a variety of matching effects. A large multi-site clinical trial, Matching Alcoholism Treatments to Client Heterogeneity (Project MATCH), was initiated in 1989 to rigorously test the most promising hypothetical matches. Patients were randomly assigned to one of the following three different types of behavioral therapy:

Motivational Enhancement Therapy (MET), a brief intervention using techniques of motivational psychology to encourage individuals to consider their situation and the effect of alcohol on their life, to develop a plan to stop drinking, and to implement the plan.

Cognitive -Behavioral Skills Therapy (CBST) in which alcoholism is viewed as a type of maladaptive, learned, behavioral response to stressful triggers. In CBST, the patient is taught ways to respond to drinking-provoking situations with non-drinking actions. Patients practiced drink-refusal skills, learned to manage negative moods, and learned to cope with urges to drink.

Twelve-step Facilitation Therapy (TSF), which encouraged patients to become involved in Alcoholics Anonymous (AA). In TSF, trained therapists helped patients to find AA sponsors, arranged for regular AA attendance, introduced patients to AA literature and other materials, and helped patients to work the first five of AA's twelve steps. (TSF was designed specifically for Project MATCH. Although grounded in the twelve-Step principles, it was a professionally delivered, individual therapy different from the usual peer-organized AA meetings and was not intended to duplicate or substitute for traditional AA.)

No decisive matches between patients and treatments were found; the three treatments were approximately equal in their efficacy for all patients. Further, treatment in all three approaches resulted in substantial, long-term reductions in drinking and related problems.

Twelve-step Programs. Professional Treatment based on the twelve steps of AA is the dominant approach to alcoholism treatment in the United States. Higher levels of AA attendance during and following professional treatment are consistently associated with better outcomes, but AA affiliation without professional treatment has not routinely resulted in improvement. Twelve-step approaches also have been found to be more effective than motivational enhancement therapy for individuals whose social networks support drinking.

Medications for Alcoholism Treatment. One of the major changes in alcoholism treatment is the current and future availability of medications that can improve treatment outcome. Medications that interfere with craving can reduce the likelihood that a recovering alcoholic will suffer a relapse. Two such medications are currently available: naltrexone in the United States and acamprosate in Europe. A third medication, nalmefene, is currently under study.

Naltrexone. Naltrexone is the first medication approved to help maintain sobriety after detoxification from alcohol since the approval of disulfiram (Antabuse ®) in 1949. Originally developed for use in treating heroin addicts by reducing their cravings for this drug, naltrexone was observed to reduce alcohol use by heroin addicts. Further research confirmed this observation: naltrexone used in combination with verbal therapy prevented relapse more than standard verbal therapy alone.

Acamprosate. Acamprosate was developed in Europe. Clinical trials are now underway in the United States to gain approval by the FDA to market acamprosate in the United States. The results of the European clinical trials of acamprosate were very similar to those found in the U.S. with naltrexone; about twice as many people did well with acamprosate as they did with placebo. They also found, as with naltrexone, that the medication is

effective only in combination with behavioral therapy.

Nalmefene. A new opiate antagonist—nalmefene—has recently been tested for use in alcoholism treatment. This medication significantly reduced relapse to heavy drinking among recovering alcoholics, decreased the risk of relapse, and produced no significant side effects. In studies in which naltrexone and nalmefene were compared, nalmefene entered the bloodstream more quickly and had a somewhat lower risk of liver toxicity than did naltrexone.

Combined Therapeutic Approaches. Combining behavioral therapies with pharmacotherapies is likely to be the next important advance in alcoholism treatment. There are several ways in which behavioral and pharmacological therapies could work together: One therapy might continue to function if the other failed; each therapy might increase the effectiveness of the other; or each might act on the same neural circuits. Naltrexone, used in combination with behavioral therapy, has been shown to prevent relapse more than behavioral therapy alone. The effectiveness of combined therapeutic approaches, including approaches which combine both acamprosate and naltrexone, are currently being examined.

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Enoch Gordis

Alcohol, An Overview Alcohol abuse and Alcoholism are serious problems. *Alcohol abuse* refers to heavy, problematic drinking by nondependent persons, while *alcoholism* suggests Tolerance, Physical Dependence, and impaired control of drinking. There are an estimated 9 million alcohol-dependent persons and 6 million alcohol abusers in the United States (Williams et al., 1989).

Problems that arise from misuse of alcohol vary widely, but they often include the following areas: financial, legal, family, employment, social, and medical. Medical complications include alcoholic liver disease, gastritis, pancreatitis, organic brain syndrome, and the Fetal Alcohol Syndrome (FAS). It is estimated that more than 100,000 alcohol-related deaths occurred in the United States in 1987 (Centers for Disease Control, 1990). The most common alcohol-related death is a motor vehicle fatality.

Despite the complex nature of alcohol abuse and dependence, research has burgeoned over the past decade and has deepened our understanding of the causes, prevention, and remediation of alcohol abuse and alcoholism. Here, we briefly review assessment of alcohol problems, detoxification, and treatment.

ALCOHOLISM ASSESSMENT

To appropriately assign an individual to treatment, his or her condition must be accurately evaluated. Management of alcoholism may be seen as involving a five-stage sequential process: screening, diagnosis, triage, treatment planning, and treatment-outcome monitoring. Specific procedures exist to help inform clinical decisions at each of these stages (Allen, 1991). Screening tests help determine whether a drinking problem might exist. If this seems likely, formal and more lengthy diagnostic procedures are performed to specify the nature of the problem. If the diagnosis of alcoholism is established, determination of the type of treatment setting and intensity of care needed for detoxifying and treating the patient must be made next. Treatment planning can then be initiated to establish rehabilitation goals and strategies appropriate to the patient. Finally, outcome is monitored to determine if further treatment is needed or if a different treatment approach is advisable.

DETOXIFICATION

When an alcohol-dependent person abruptly stops drinking, physiological symptoms may occur. This cluster of symptoms is termed *alcohol withdrawal*, and symptoms can range from relatively mild discomfort to life-threatening problems. Mild symptoms include sweating, tachycardia (rapid heartbeat), hypertension, tremors, anorexia, sleeplessness, agitation, and anxiety. More serious consequences involve seizures and, rarely, DELIRIUM TREMENS (DTs), characterized by agitation, hyperactivity of the autonomic nervous system, disorientation, confusion, and auditory or visual hallucinations. It has been postulated that as the number of untreated withdrawal episodes increases, the potential for more serious symptoms in subsequent withdrawals may also escalate. This phenomenon is known as *kindling* (Brown, Anton, Malcolm & Ballenger, 1988).

Treatment of alcohol withdrawal includes both pharmacological and nonpharmacological interventions. It is generally believed that if the withdrawal symptoms are mild to moderate, no medications are needed. Instruments such as the Clinical Institute Withdrawal Assessment Scale (Foy, March & Drinkwater, 1988) have recently been developed to gauge severity of withdrawal symptoms. Nonpharmacological techniques used to treat milder forms of alcohol withdrawal include efforts to reduce anxiety and to provide emotional reassurance. Patients in withdrawal should receive the B vitamin thiamine so as to prevent the occurrence of the WERNICKE—Korsakoff syndrome, a serious neurological complication of alcoholism.

If the symptoms are more severe, however, drugs should be prescribed. The most commonly used medications to treat withdrawal have been BENZODIAZEPINES. The benzodiazepines have been demonstrated in randomized clinical trials to reduce the occurrence of seizures and other serious withdrawal symptoms. They have a wide margin of safety. Side effects, however, include transient memory impairment, drowsiness, lethargy, and motor impairment. Benzodiazepines must be tapered down and then stopped after the patient is no longer suffering from withdrawal because patients can develop dependence on them. In addition, the physiological effects of benzodiazepines are synergistic or additive with those of alcohol—hence, it is important that patients not drink while taking them. Other medications to treat withdrawal include beta-adrenergic blockers, alpha-2 adrenergic agonists, calcium channel blockers, and anticonvulsant agents such as carbamazepine; however, the first two categories of drugs do not prevent seizures and, therefore, are less useful than benzodiazepines. Recent research suggests that carbamazepine may be an effective alternative to benzodiazepines, while calcium channel blockers are still in early stages of research.

TREATMENT OPTIONS

After screening, diagnosing, and detoxifying a patient, the clinical staff has numerous options for short- and long-term treatment. While a more detailed review of these interventions can be found in Hester and Miller (1989), the techniques can be categorized as follows:

Alcoholics Anonymous. Since the 1940s, ALCOHOLICS ANONYMOUS (AA) has been an important component of alcoholism rehabilitation, and many recovered alcoholics are convinced that AA was essential for their recovery. As a means of achieving and maintaining SOBRIETY, AA consists of regular meetings utilizing fellowship, mutual support for sobriety, open discussions, and a program known as the TWELVE STEPS. The effectiveness of AA has not been established by randomized clinical trials, largely because the organization was developed outside the scientific mainstream. A well-designed study by Walsh, Hingson, and their colleagues (1991) was, however, done in the setting of an EMPLOYEE ASSISTANCE PROGRAM (EAP). Employees seeking or referred for treatment were randomly assigned to inpatient treatment with AA as a component, AA alone, or self-choice of treatment. All three treatment conditions resulted in equal improvement in job performance; however, inpatient treatment did better than AA or self-choice in terms of several aspects of drinking behavior. Inpatient treatment was particularly valuable for those employees who were abusing both alcohol and COCAINE. Other self-help groups that do not use the twelve-step program (e.g., RATIONAL RECOVERY) also exist.

Minnesota Model. The MINNESOTA MODEL is so named because it originated in several alcoholism programs in Minnesota and is the most common type of inpatient treatment for alcoholism in the United States. It stresses complete abstinence

and employs methods such as group and individual therapy, alcohol education, family counseling, and required attendance at AA meetings. The staff in these programs are usually a mixture of professional individuals and recovering alcoholics. The evidence for its effectiveness is limited. The study by Walsh et al. (1991) supports the idea that these programs are effective. Studies on health-care utilization costs before and after treatment for alcoholism also add evidence that these programs are effective. When this general program is used to treat drug problems other than alcoholism, it is often referred to as a chemical-dependency program.

Group Psychotherapy. Group psychotherapy is widely used in the treatment of alcoholics. The many types of group psychotherapy employ supportive, cognitive, psychoanalytic, or confrontational techniques. Also, group psychotherapy is often used in conjunction with other approaches, such as AA and pharmacologic adjuncts to treatments.

Individual Psychotherapy. Individual psychotherapy attempts to probe possible underlying reasons for problem drinking and subsequently strives to guide the patient in working through emotional difficulties. Some of the cognitive and behavioral approaches described below can also be considered forms of psychotherapy. Similar to group psychotherapy, individual psychotherapy is often combined with other treatment activities. Despite the widespread use of group and individual psychotherapy, the scientific evidence supporting their efficacy as isolated treatments is limited.

Family and Marital Therapy. This type of therapy involves the problem drinker, spouse, and sometimes other family members. Over the past several years, research interest has heightened in determining the contribution of family and marital factors in aiding the patient to sustain recovery. Generally, family and marital therapy seeks to enhance communication, problem-solving, and positive reinforcement skills.

Social-Skills Training. Social-skills training includes techniques for improving communication skills, forming and maintaining interpersonal relationships, resisting peer pressure for drinking, and becoming more assertive. Research on its effectiveness has been encouraging.

Relapse Prevention. RELAPSE PREVENTION is a behavioral approach that deals with teaching the patient to successfully cope with environmental situations that may serve as high-risk drinking stimuli. Relapse prevention is important in alcoholism treatment, since many patients who are successfully detoxified and stabilized tend to revert to drinking. While relapse prevention is widely used, the evidence of its effectiveness is again limited, albeit promising.

Stress Management. Stress-management techniques may be employed to reduce emotional discomfort, which may contribute to drinking behavior. Specific techniques include deep-muscle relaxation, biofeedback, systematic desensitization, and cognitive and behavioral strategies to cope with stress-inducing stimuli.

Pharmacotherapy. Since the 1950s, DISULFIRAM (Antabuse) has been the most widely used medication in the treatment of alcoholism. Patients on disulfiram are deterred from drinking because to do so would cause physical discomfort, including headaches, flushing, and rapid heartbeat. A major problem in using disulfiram is lack of patient compliance. Several techniques have been developed to enhance compliance, including establishing a contract with the client or significant other on disulfiram administration, offering positive and negative incentives for taking the medication, and using implants.

In addition to disulfiram, recent advances have been made in the development of medications that directly curb desire to drink. The most promising include serotonergic agents and opioid antagonists—these agents act on brain mechanisms that are believed to be related directly to drinking.

Aversive Therapy. This type of therapy attempts to establish a conditioned avoidance response to alcohol. Drinking is paired with unpleasant experiences, such as electric shock, nausea, vomiting, or imagined unpleasant consequences. The underlying rationale of AVERSION conditioning is that patients will be less likely to drink if they associated alcohol consumption with immediate negative consequences. Good evidence that this approach is effective is lacking, because of the absence of randomized clinical trials evaluating aversive therapy. Some programs using it report very high levels of abstinence, however, in the months following inhospital treatment.

Patient-Treatment Matching. A newer strategy in alcoholism treatment attempts to match particular types of treatments to relevant patient characteristics, rather than assigning all patients to

similar treatments. Common patient-matching variables include the patient's collateral psychopathology, degree of alcohol involvement, and personality and motivational characteristics. Approximately forty studies, although based on small numbers of patients, have supported the concept that patient-treatment matching improves treatment outcome.

Community-Reinforcement Approach. The community-reinforcement approach (CRA) is a broad-spectrum treatment approach that focuses on positive reinforcers for abstinence in the patient's natural environment. Specific techniques include adding improvements to the patient's employment conditions, marital relationships, problem-solving skills, social skills, and stress management—and different components of the program are chosen for the individual, depending on his or her life problems. The initial studies of CRA are encouraging.

CONCLUSIONS

Advances in treatment research have led to a variety of treatment interventions. The alcoholism-treatment community must become better able to assist the recovery of alcoholics and alcohol abusers. Advances in assessment technology have helped identify patient needs more clearly; this subsequently enables the clinician to provide a treatment regime tailored to the needs of the patient. An important future direction for alcoholism-treatment research is to discover how to more precisely match patients with specific types of treatment interventions. Also, development of new medications to directly reduce drinking behavior will have a major impact. Future treatments will likely combine pharmacologic interventions with behavioral and psychosocial therapies to further improve treatment outcome.

(SEE ALSO: *Accidents and Injuries from Alcohol; Complications; Treatment, History of; Treatment Types*)

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Alcohol, Behavioral Approaches The use of behavioral and other psychological treatments for alcohol abuse has a long history. In the nineteenth century, Benjamin Rush, often regarded as the founder of American psychiatry, described a variety of social and psychological cures for chronic drunkenness. Treatment procedures derived from principles of learning and conditioning were being tested in the 1920s, prior to the development of modern pharmacologic approaches. Currently, there is a large scientific literature documenting the effectiveness of various behavioral treatments for alcohol problems.

The most obvious argument for the use of behavioral approaches in treating alcohol abuse is that the drinking of alcohol or ethyl alcohol is a *behavior*. Regardless of the therapeutic approach used, the criterion for success or failure in treatment studies is typically behavioral—whether and how much a person continues to drink. Research amply demonstrates that drinking behavior is substantially influenced by a wide variety of psychological processes, including beliefs and EXPECTAN-

CIES, the examples of friends and family, the customs and norms for drinking within one's society or subgroup, emotional states, family processes, and the positive and negative consequences of drinking. Treatments that address these factors directly, then, might be expected to be helpful in overcoming alcohol problems.

In fact, dozens of well-controlled studies since the 1960s do support the effectiveness of behavioral treatments. The benefits of such treatment have typically been larger than those reported for pharmacologic approaches and have been shown in some studies to endure over follow-up periods of several years. This research in itself provides a convincing reason to use behavioral methods in treating alcohol abuse.

Still another reason is the finding that psychosocial processes strongly influence whether or not a person will relapse after treatment. The likelihood of relapse is decreased by factors such as marital stability, social support, personal coping skills, employment, and confidence in one's abilities to deal with problems. Factors like these in a person's life *after* treatment are important determinants of outcome. Treatment methods that anticipate and address these post-treatment adjustment challenges are thus important.

There is, however, little reason to argue for behavioral versus pharmacological treatment approaches, since these two approaches can be used together with good result. Behavioral methods play a key role in addressing psychosocial aspects of drinking problems and are compatible with the use of medications, where they are appropriate.

ALTERNATIVE BEHAVIORAL METHODS

A behavioral approach to treating alcohol abuse does not involve just one method. Rather, a variety of strategies can be used to accomplish the central goal—to change drinking behavior—and several methods are typically employed in a treatment program.

Treatment methods should not be confused with treatment goals. The general behavioral methods described below can be applied in pursuit of different goals. Sometimes the goal of treatment is the complete elimination of alcohol drinking for the rest of a person's lifetime (total and permanent abstinence). For others, the goal may be to reduce alcohol use to a level that will no longer threaten a person's physical or psychological health. The goals of treatment may also include other important dimensions besides drinking—to get and hold a job, to have a happier marriage and family life, to learn how to deal with anger, and to find new ways of having fun that do not involve drinking. Finally, it is worth noting that clients may have treatment goals that differ from those of the therapist. Behavioral treatment methods do not inherently dictate outcome goals, but they can be used to achieve goals once chosen.

Teaching New Skills. Alcohol is often used in an attempt to cope with life problems. People may drink to relax or loosen up, to get to sleep, to feel better, to enhance sexuality, to build courage, or to forget. In truth, alcohol rarely works as an effective coping strategy for dealing with emotional and relationship problems. In the long run, it often makes such problems worse. Yet the seeming immediate relief can make alcohol appealing when a person is faced with bad feelings or social problems. To the extent that a person comes to rely upon drinking to cope, that person is termed *psychologically dependent* on alcohol.

One behavioral approach, sometimes called *broad-spectrum treatment*, directly addresses this problem by teaching the person new coping skills. Ten controlled studies, for example, have found that the addition of *social-skills training* increases the effectiveness of treatment for alcohol abuse. People are taught skills for expressing their feelings appropriately, making requests, refusing drinks, and carrying on rewarding conversations. *Stress-management training* has also been shown to help prevent relapse to drinking. People learn how to relax and deal with stressful life situations without using drugs.

Self-Control Training. Another well-documented behavioral approach is *self-control training*, which teaches methods for managing one's own behavior. Some common elements in self-control training include: (1) setting clear goals for behavior change; (2) keeping records of drinking behavior and urges to drink; (3) rewarding oneself for progress toward goals; (4) making changes in the way one drinks, or in the environment, to support new patterns; (5) discovering high-risk situations where extra caution is required; and (6) learning strategies for coping with high-risk situations. Although often used to help people reduce their

drinking to a moderate and nonproblematic level, self-control training can also be used when total abstinence is the goal. This method has been found to be particularly helpful for less severe problem drinkers. It has also been found to be more effective than educational lectures for drunk-driving offenders.

Marital Therapy. There are several reasons to consider treating not only the excessive drinker, but also the spouse. First, problem drinking commonly affects the drinker's partner in adverse ways. Secondly, the spouse may be quite helpful during treatment in clarifying the problem and in developing effective strategies for change. Thirdly, the spouse can provide continuing support for change after treatment. Finally, marital distress may be a significant factor in problem drinking, and direct treatment of marital problems can help to prevent relapse.

Research indicates that problem drinkers treated together with a spouse fare better than those treated individually. Behavioral marital therapy in particular is well supported by current outcome research.

Aversion Therapies. Another set of treatment strategies applies the learning principle of aversive counterconditioning (called AVERSION THERAPY). The idea here is that if drinking is paired with unpleasant images and experiences, the desire for alcohol is diminished, and drinking decreases. There is sound evidence that it is possible to produce a conditioned aversion to alcohol in both animals and humans. The taste and even thought of alcohol become unpleasant. There is also evidence that aversion therapy is successful to the extent that this kind of conditioned aversion is established during treatment. Some forms of aversion therapy pair the taste of alcohol with unpleasant sensations such as nausea, foul odors, or electric shock. A newer form, termed *covert sensitization*, uses no physical aversion of this kind but instead pairs alcohol with unpleasant experiences in imagination. These approaches may be particularly useful for those who continue to experience craving or a strong positive attachment to alcohol.

Psychotherapy. Many kinds of psychotherapy have been tried with alcohol abusers. In general, studies suggest that individual psychotherapies with a goal of insight into unconscious causes of drinking have been largely unsuccessful. Likewise, group psychodynamic psychotherapies have had a poor track record in treatment-outcome studies. As a distinct element, confrontational group therapy, a common element of U.S. treatment programs, is also unsupported by current research. More recently, *cognitive therapies* have gained popularity, and some controlled trials supporting their efficacy.

Changing the Environment. Yet another behavioral approach is behavior modification by changing the consequences of drinking. The goal here is to eliminate positive reinforcement for drinking, and to make alternatives to drinking more rewarding. Studies have reported success in working unilaterally with a drinker's spouse to make changes that discourage drinking and reinforce alternatives. A complex treatment known as the *community-reinforcement approach* (CRA) has fared well in comparisons with traditional methods. The CRA systematically encourages rewarding alternatives to drinking, teaching skills needed for living without alcohol. The CRA incorporates a number of treatment elements, including marital therapy, social-skills training, the taking of disulfiram (Antabuse—a medication that causes aversive effects when alcohol is ingested), and job-finding training. The use of *behavioral contracting*—drawing up a specific agreement about future drinking and its consequences—has been found to be an effective component of treatment in several studies.

Brief Motivational Counseling. An interesting and unexpected finding in more than a dozen well-controlled studies is the effectiveness of relatively brief motivational counseling. Certain treatments, consisting of one to three sessions, have been found to be significantly more effective than no treatment and often as effective as more extensive treatment regimens. These motivational approaches, now studied in several nations, typically include a thorough assessment, feedback of findings, clear advice to change, and an emphasis on personal responsibility and optimism. The key seems to be to trigger a decision and commitment to change. Once this motivational hurdle has been crossed, people frequently proceed to change their drinking on their own without further professional assistance. In fact, treatment approaches that proceed directly into strategies for changing drinking may fail because they do not address this motivational prerequisite for change.

Therapist Style. Other recent research indicates that the skills and style of the therapist have

important effects on treatment outcome. With impressive consistency, therapist success has been linked to an empathic and supportive style, rather than an aggressive and confrontational approach. Directive and confrontational tactics tend to elicit resistance and defensiveness from clients, which in turn are predictive of a lack of therapeutic change. It is clear that the same treatment approach can have dramatically different outcomes when administered by different therapists.

HOW IS SUCCESS JUDGED?

In one sense, judging the outcome of treatment would seem simple: Either the person is or is not still drinking in a problematic manner. A closer examination of treatment-outcome research quickly reveals a number of complexities.

First is the question of the standard against which a treatment is to be judged. Is a "success" rate of 60 percent spectacularly good or shameful? This is decided relative to the expected outcome without the same treatment. This is why the usual standard for judging effectiveness in medical research is the *controlled trial* in which clients are randomly assigned to different treatment methods. In the absence of proper controls, one cannot judge adequately whether the outcome of a treatment is better or worse than it would have been without the special treatment. Evidence from properly controlled trials is more consistent than the results of uncontrolled trials, presenting a clearer picture of effectiveness.

A second complexity is: What constitutes success? When success is defined very conservatively, as total abstinence from alcohol (not even one drink) since the end of treatment, low success rates can be expected. Yet if some drinking is permitted among "successes," it is necessary to define the acceptable limits for how much, how often, and with what consequences. Some studies have reported only a category of "improved" cases without adequate definition.

Once successful outcome is clearly defined, there is the problem of how to measure it. Should a researcher accept the client's self-report? Should friends and family members be interviewed? Should blood, breath, or urine samples be required? If multiple outcome measures are used, how does one decide which is the truth?

Still another example is the issue of length of follow-up. Success rates are typically highest within a few weeks or months from the time of treatment. A large percentage of relapses occur between three and twelve months after treatment. Short follow-up periods, then, overestimate success rates. Longer follow-ups raise the additional problem of how to deal with lost cases. If one studies only those who can be easily found two years later, success rates may be inflated.

For these reasons, the effectiveness of treatment approaches is best judged by accumulating evidence from several properly controlled studies. Conclusions presented above, regarding the efficacy of different psychological treatment approaches, were drawn on this basis.

MATCHING PEOPLE TO TREATMENTS

It is unlikely that research will ever identify a single superior treatment for alcohol abuse. Drinking and alcohol-related problems are far too complex. The cause for real optimism is found in the number of different approaches with reasonable evidence of effectiveness. For a given person, then, the chances of eventually finding an effective approach are good.

Recent research indicates that these various treatment approaches work best for different kinds of people. As such evidence accumulates, it will be increasingly possible to choose optimal treatment strategies for people based on their individual characteristics. Treatment systems, therefore, should work toward providing a range of different approaches, rather than offering the same basic treatment to everyone with alcohol problems.

(SEE ALSO: *Causes of Substance Abuse; Disease Concept of Alcoholism and Drug Abuse; Treatment Types*)

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Alcohol, Pharmacotherapy Research on pharmacotherapy for ALCOHOLISM continues to expand, as there are still many questions unanswered at the turn of the millennium. Currently, the most widely used medication for the treatment of alcoholism is DISULFIRAM, which has been in use for half a century. Disulfiram (Antabuse ®) does not act to reduce the CRAVING for ALCOHOL or ameliorate the euphorigenic (feeling of well-being) effect of alcohol. A variety of newer drugs were tested in the late 1990s but have not fulfilled early expectations. It was hoped that "anticraving" medications and medications that reduce the "high" from drinking alcohol would be particularly useful in recovering alcoholics who are prone to relapse. Medications originally developed to treat DEPRESSION and ANXIETY were also thought to have potential for managing drinking behavior in specific subgroups of alcoholics. These also do not appear to be helpful except among some alcoholics with comorbid psychiatric disorders.

This article focuses on four categories of medications that are either currently available or are still being tested for the treatment of alcoholism and alcohol abuse. These include the following: alcohol-sensitizing agents; agents that directly attenuate drinking behavior; agents to improve cognition in patients with alcohol-induced impairments; and agents to treat psychiatric problems concurrent with alcoholism. The most promising medications within each of the above categories are examined, addressing their stage of development, clinical efficacy, potential side effects, and future research. The first section of this article will describe briefly the methodology used to conduct clinical pharmacotherapy studies.

CONTROLLED CLINICAL TRIALS

The method used to determine medication efficacy is called the controlled clinical trial. The key components of clinical trials include the following: control groups; random assignments of eligible subjects to medication or to control groups; use of placebos (identically appearing but inactive medications) for the control group—unless a standard effective medication is available to serve as the comparison; assurance that neither the patients receiving the drug nor the physicians administering/prescribing know whether they are getting the active medication or the placebo (called double-blind); methods that validly and reproducibly measure the response to the medication; methods to monitor whether subjects take the medication; and procedures to follow all the patients who entered the study for the duration of the clinical trial. After the data are collected, they must be analyzed by using the appropriate statistical tests.

Randomizing. It is important to randomize eligible patients to the treatment and placebo groups, because this assures that the two groups are comparable except for the medications being prescribed. If some method other than randomization is used to assign patients to treatments, it is likely that the groups will differ in important characteristics such as severity of illness. If one of the groups is in general more severely ill than the other, the sicker group is less likely to do well regardless of the treatment. If the more severely ill group receives the active medication, the difference between the medication group and the placebo group after treatment does not appear as great because the placebo (control) group was less ill at the beginning. Thus, it may appear that the medication was not effective.

Double-blinding. "Blinding" of both the patients and the physicians is necessary because of their expectations and beliefs. Patients usually seek treatment in the expectation that the physician will prescribe or recommend something that will cure or improve their condition. Hence, patients who receive placebos often feel better. Therefore, if a placebo control is not used, one might conclude that a new treatment works when one is only observing the placebo kind of response. (Conversely,

patients often report side effects when they take placebos. So not all side effects are necessarily due to an active medication.) Physicians often believe very strongly that the new drug will be the effective treatment they are searching for, and their objectivity is diminished by this bias. To remove this influence on their perception of the outcome of treatment, the physicians treating the patients are "blinded" as well as the patients, hence a double-blinding is effected.

Accurate Assessment. If the methods used to assess the response to treatment do not accurately measure the response to the medication, erroneous conclusions may be drawn (Fuller, Lee, & Gordis, 1988). Patients' self-reports about their response to treatment should not be used without corroborating data in controlled clinical trials unless no other means for obtaining information is available. Such reports may be inaccurate for a variety of reasons, including inaccurate memory and the tendency to give socially desirable answers.

It is also important to know whether the patients actually took the medications. Often, patients do not take their medications or take them erratically, particularly if they are being treated for an asymptomatic condition for a long period of time and/or if the medication has a high incidence of unacceptable side effects (Haynes, Taylor & Sackett, 1979).

Patients who drop out of treatment frequently are atypical of all patients in treatment. In alcoholism treatment, the dropouts are usually drinking and having problems because of their drinking. So, if a study bases its conclusions only on those who stay in treatment, the results of the therapy are likely to be exaggerated. Therefore, it is important to locate and assess treatment response in all or almost all who initially began treatment. For an excellent description of clinical trials, their methods and issues, see Byar et al. (1976).

If control groups were used, methods other than randomization were used to assign patients to the disulfiram group or the control group. Hence, the groups were not comparable, and placebo groups were rarely used. "Blinding" was not done. No attempts were made in most of the studies to determine whether patients took the medication. The alcoholic's report on abstention from alcohol was the only information obtained to judge whether disulfiram was effective. In some studies, only about half the patients were available for follow-up.

Multi-Site Trial. During the past decade, more rigorously designed clinical trials of disulfiram have been done, and these give more precise information about the efficacy of disulfiram. The largest of these was a multi-site clinical trial done in nine Veterans Administration clinics (Fuller et al., 1986). In this study, 605 men were randomly assigned to three groups:

1) a 250-milligram disulfiram group (the usual dose);
2) a 1-milligram disulfiram group; and
3) a no-disulfiram group.

The 1-milligram group was equivalent to a placebo, because this dose is not sufficient to cause a disulfiram—ethanol reaction (DER) but controls for the expectation that one will get sick if one drinks alcohol while taking disulfiram. The no-disulfiram group was told they were not receiving Antabuse®; it was a control for the standard counseling that alcoholics receive in treatment. The patients in the two disulfiram conditions were "blinded" as to whether they were receiving the 250-milligram or the 1-milligram dose. The data to judge the effect of treatment were collected by research personnel who had no involvement in the treatment of the patients and were "blinded" to group assignment. The research staff members interviewed the patients, cohabiting relatives, and friends (*collaterals*) every two months during the year of follow-up. Urine specimens were collected every time the patients returned to the clinic and were analyzed for the presence of alcohol. A vitamin, riboflavin, was incorporated into the 250-milligram and 1-milligram tablets. The nodisulfiram patients received a tablet identical in appearance to the disulfiram tablets but containing only riboflavin. The urine specimens were also analyzed for riboflavin. This allowed the investigators to tell whether the patients were taking their medications regularly.

In contrast to most of the previous studies, this tightly designed study did not find that more of the patients who received disulfiram stayed sober for the year than those who received the placebo or counseling only. Nor was disulfiram associated with better employment or social stability; however, in about 50 percent of the men who relapsed, drinking frequency was significantly less for those who received disulfiram than for those who received either the placebo or no disulfiram. This subset of

men who relapsed by drinking less frequently if assigned to disulfiram were slightly older and had more social stability (as indicated by longer residence at their current address) than the other men who relapsed. These results indicate that disulfiram is not more effective than routine treatment for most male alcoholics—female alcoholics were not included in the study—but may have some benefit for socially stable male alcoholics.

In the multi-site study, only 20 percent of the patients took the medication regularly; however, abstinence for the year was highly associated with compliance with the disulfiram regimen. This suggests that if ways were found to get patients to take disulfiram regularly, the effectiveness of the drug would be greatly improved. This conclusion has to be tempered by the finding that those who regularly took the 1-milligram placebo or the vitamin without disulfiram, as well as those who took disulfiram, were much more likely to remain sober than those who were less adherent to their regimens. Nevertheless, alcoholism treatment researchers have studied various methods for improving compliance with disulfiram, and preliminary results suggest that these may be beneficial. These treatment strategies have included having the spouse or a treatment facility staff member observe the patient ingesting the medication, establishing a contract with the patient about taking it, and/or building in positive (rewards) or negative (loss of privileges) incentives to take it. A recent controlled study of disulfiram taken in the presence of a relative, friend, or member of the clinic staff found that this method of administration resulted in significantly less alcohol being consumed during a six-month period (Chick et al., 1992). More well-designed studies of these measures to improve compliance with the disulfiram regimen are needed before it is known if they will improve the effectiveness of disulfiram as a treatment for alcohol dependence.

On the basis of the large well-designed studies done to date, it seems prudent to recommend that disulfiram should not be used initially in the treatment for alcoholism. However, if the patient relapses and has indicators of social stability, a discussion with the patient about the possible benefits and the possible risks of disulfiram is warranted, and if the patient is willing to take disulfiram, a trial course is warranted. During the first six months of treatment, it is important that liver tests be monitored closely. The effectiveness of the drug may be enhanced if the patient agrees to take it under supervision.

ALCOHOL-SENSITIZING AGENTS

The most commonly used alcohol-sensitizing agent is disulfiram, which has been used in clinical practice since the 1950s to deter alcoholics from drinking. It is not an aversive drug in the strict sense of the word, since it is not used, as apomorphine is used, to condition individuals to have an aversive response at the sight or smell of alcohol. Rather, its objective is to deter drinking by the threat of having a very unpleasant reaction if one does drink alcoholic beverages. Its severity depends on the amount of alcohol and disulfiram in the blood. The symptoms of the reaction include facial flushing, tachycardia (rapid heart beat), palpitations, dyspnea (indigestion), hypotension (lowered blood pressure), headaches, nausea, and vomiting. Deaths have occurred with severe disulfiram—ethanol reactions (DERs).

A DER results when alcohol is ingested because disulfiram inhibits the functioning of an enzyme, aldehyde dehydrogenase. This enzyme is needed to convert the acetaldehyde—the first metabolic product in the catabolism of ethanol—to acetic acid. If aldehyde dehydrogenase is inhibited, an elevation in blood acetaldehyde results. The increased circulating acetaldehyde is believed to cause most of the symptoms and signs of the DER.

Disulfiram is given orally. The usual dose is 250 milligrams, although larger doses have been used. Doses of less than 250 milligrams may fail to cause a DER, while doses of more than 250 milligrams have a greater risk of producing serious side effects. Adverse effects of disulfiram range from mild symptoms such as sedation, lethargy, and a garlic-like or metallic taste in the mouth to more serious side effects such as major depression, psychotic reaction, or idiosyncratic toxic hepatitis—which may be fatal. A dose between 250 milligrams and 500 milligrams is usually adequate to cause a DER if alcohol is ingested but not so high as to cause major side effects. The dose should be individualized for each patient.

Alcohol-sensitizing agents other than disulfiram also exist. CALCIUM CARBIMIDE, which is available in Canada under the brand name Temposil, has been used clinically, although it is currently not ap-

proved by the FDA for use in the United States. Calcium carbimide produces physiological reactions with alcohol similar to those produced by disulfiram, but the onset of action is quick—within one hour after administration—compared to twelve with disulfiram. Also, the duration of action is short—approximately twenty-four hours—versus up to six days with disulfiram. Calcium carbimide, with its faster onset of action, might be especially helpful with impulsive drinkers. A possible side effect of calcium carbimide is reduced thyroid function, however, thus making its use problematic in patients with thyroid problems. It has some additional side effects that include dizziness, slight depression, skin rashes, and impotence. One puzzling side effect of calcium carbimide is a mild elevation in the patient's white blood cell count. As of 2000, there is a paucity of randomized clinical trials comparing calcium carbamide to placebo—so, its efficacy is uncertain.

AGENTS THAT ATTENUATE DRINKING BEHAVIOR

The development of medications to curb drinking behavior is one of the important and exciting areas of alcohol research. In developing such medications, researchers have relied on new information about the biological bases of drinking behavior and alcohol craving. This process is complex and involves the interactions among several neurochemical mechanisms, including NEUROTRANSMITTERS, hormones, neuropeptides, RECEPTORS, second messenger systems, and various ion channels in multiple regions of the brain.

Recent research has focused on medications that alter the functional activity of several neurotransmitter systems. In this section, we discuss medications that directly attenuate drinking by acting on the following neurotransmitter systems: SEROTONIN, OPIOIDS, DOPAMINE, and GAMMA-AMINOBUTYRIC ACID (GABA).

Agents That Affect the Serotonin System. Several lines on animal and human research suggest that brain serotonin is associated with alcoholism. Serotonin levels are lower in several regions of the brain in rats selectively bred to drink alcohol than in rats that do not prefer alcohol. In humans, measurements of cerebral spinal fluid levels of 5-hydroxyindoleacetic acid (5-HIAA), a metabolite of serotonin, revealed lower levels of 5-HIAA in alcoholics who were abstinent for four weeks than in nonalcoholics. Also, the availability of the serotonin precursor, tryptophan, appears to be lower in alcoholics, particularly those in early onset of alcoholism (drinking before twenty years of age).

SEROTONIN-UPTAKE INHIBITORS, commonly used to treat depression, seemed to be effective in reducing alcohol consumption in both animal models and humans. Serotonin-uptake inhibitors act by preventing the uptake of serotonin during synaptic transmission, resulting in a prolonged action. They are easily administered (orally) and require only a single daily dose.

The serotonin-uptake inhibitors available for clinical testing include fluoxetine (Prozac), fluvoxamine, citalopram, and viqualine. Several double-blind, placebo-controlled studies of these agents in various types of subjects—ranging from social drinkers to chronic alcoholics— showed an increase in the number of abstinent days and a decrease in the number of drinks on drinking days (Gorelick, 1989). The effect of the serotonin-uptake inhibitors studied has, however, been modest (a 25% decrease in alcohol intake).

The precise mechanism of action of the serotonin-uptake inhibitors on drinking behavior is unknown. One of the most plausible explanations offered is their ability to suppress appetitive behaviors in general. However, consummatory behaviors are quite complex, and even this hypothesis may be an oversimplification.

In addition to the serotonin-uptake inhibitors, agents that selectively block (antagonists) or activate (agonists) the subtypes of serotonin receptors were considered promising. At least four major types of serotonin (5-hydroxytryptamine, or 5-HT) receptors exist: 5-HT_1, 5-HT_2, 5-HT_3, and 5-HT_4. In turn, 5-HT_1 has several subdivisions, including 5-HT_{1A} receptor. Research in the early 1990s appeared to indicate that a 5-HT_3 antagonist, ondansetron, reduced alcohol consumption in alcohol abusers (Toneatto et al., 1991). Also, 5-HT_{1A} and 5-HT_2 receptors were believed to influence alcohol intake. For example, buspirone, a 5-HT_{1A} agonist and an antianxiety agent, was shown in some studies to reduce alcohol consumption in humans.

Finally serotonergic agents (e.g., fenfluramine) that cause a release of serotonin from presynaptic neurons were tested for clinical efficacy in reducing alcohol intake. In addition, the administration of serotonin precursors was thought to alter drinking

behavior. Several animal studies showed that tryptophan (precursor to serotonin) and 5-hydroxtryptophan (hydroxylated form of tryptophan) reduce the amount of alcohol consumed.

As of 2000, however, serotonergic agents have not fulfilled their initial promise. A 1999 review of forty-one major clinical studies of anti-alcohol medications and eleven follow-up studies reported that the data from studies of serotonergic agents were confounded by the high rates of comorbid mood disorders in the subject populations. These medications appear to be useful primarily in the treatment of alcoholics with concurrent psychiatric diagnoses.

Agents That Affect the Dopamine System. DOPAMINE is another neurotransmitter identified as influencing drinking behavior. Dopamine is thought to play a major role in the stimulant and reinforcing properties of alcohol as well as other drugs. Decreased levels of dopamine are observed in the NUCLEUS ACCUMBENS of alcohol-seeking rats (as compared with nonalcohol-seeking rats). The nucleus accumbens is the region of the brain believed to be involved with alcohol craving. Studies in the early 1990s demonstrated that the application of alcohol to the nucleus accumbens and striatum of a rat brain causes a release of dopamine (Wozniak et al., 1991; Yoshimoto et al., 1991).

The administration of medications that increase brain dopamine levels (bromocriptine, GBR 12909, and amphetamine) results in a reduction of alcohol intake in alcohol-preferring rats. Several studies have been conducted in humans using the dopamine type 2 agonist (D_2) bromocriptine. One study (Borg, 1983) indicated that bromocriptine reduced alcohol craving and consumption in severe alcoholics, while another (Dongier et al., 1991) found a reduction in alcohol consumption and an improvement in psychological problems in both bromocriptine-treated and placebo alcoholics, although no significant differences were observed between the two groups.

The efficacy of the dopaminergic medications in the long-term management of alcoholism is currently unclear. Further research needs to be conducted on the two major subtypes of dopamine receptors, D_1 and D_2. In addition, their interaction with other neurotransmitter systems needs to be investigated. An illustration that neurotransmitter systems do not work in isolation and that a medication affecting one may also alter another is present in several studies, which have shown that blocking the serotonin 5-HT_3 receptor with the antagonist ICS 205–930 results in an attenuation of alcohol-induced release of dopamine in the nucleus accumbens and corpus striatum of the rat brain (Wozniak et al., 1990; Yoshimoto et al., 1991).

Agents That Affect the Opioid System. Studies have shown that the opioid system also plays a role in modifying drinking behavior. Many researchers believe that alcohol craving and increased drinking behavior are related to low brain levels of endogenous opioids (compounds with opium or morphine-like properties, e.g., ENDORPHINS and ENKEPHALINS). Subsequently, increasing the opioid levels causes a decrease in drinking. This is supported by several studies. For example, administration of the opioid agonist [D-Ala^2, $MePhe^4$, $Met(O)^5$-ol]-enkephalin decreases alcohol consumption in alcohol-preferring mice. Large doses of morphine (a classic opioid agonist) also result in a significant reduction in alcohol intake. In addition, increasing the availability of endogenous enkephalins by injecting mice with the enkephalinase inhibitor kelatorphan (which prevents breakdown of endogenous enkephalins) results in decreased alcohol consumption. Finally, one study demonstrated that high-risk individuals (those who have a family history of alcoholism) have lower plasma levels of beta-endorphin than do low-risk individuals (no family history of alcoholism for at least the three preceding generations).

Some researchers have challenged the hypothesis that excessive drinking is related to decreases in endogenous opioid levels. Experimental evidence includes the observation that low doses of morphine cause an increase in alcohol intake in rats.

Regardless of the mechanism of action, the opioid ANTAGONISTS NALTREXONE and NALOXONE—currently used to treat opiate abuse—have been shown to influence alcohol consumption. Both agents reduce voluntary alcohol intake in rats and monkeys. In humans, studies have shown that alcoholics treated with naltrexone have fewer drinking days, fewer relapses, and less subjective craving for alcohol (Volpicelli et al., 1992; O'Malley et al., 1992). In addition, naltrexone (Trexan) appears to cause few side effects. Interestingly, naltrexone-treated alcoholics who did have one or two drinks were less likely to continue drinking. This is impor-

tant, since some alcoholics appear to lose control of drinking after one or two drinks.

Naltrexone was the subject of a number of clinical trials in the United States; as of August 2000, ten out of thirty NIH-sponsored clinical trials were studies of naltrexone. However, a review of pharmacotherapeutic agents presented to the National Institute on Alcohol and Alcohol Abuse (NIAAA) in November 1999 concluded that the effectiveness of naltrexone in the treatment of alcoholism appears to be limited. Another review of pharmacotherapy in the treatment of alcoholism published in the *Journal of the American Medical Association* (1999) noted that naltrexone reduces the relapse rate and the frequency of drinking in alcoholics, but does not substantially enhance the abstinence rate. Studies of a similar compound, nalmefene, yielded the same results.

A secondary drawback to the use of naltrexone in treating alcoholism is the apparent reluctance of many physicians to prescribe it. An NIH study of physicians in three representative states found that very few used it with their patients. The reasons given were the physicians' lack of familiarity with the drug, and its relatively high cost to the patients.

Agents That Affect the GABA System. Several studies have now investigated the GABA system as a modulator of drinking behavior. The number of GABAergic receptors appears to be greater in the nucleus accumbens region of the brain of alcohol-preferring rats than in those of the alcohol-nonpreferring rats. An anti-craving drug that is presently approved for use in the European Community, acamprosate (calcium acetylhomotaurinate), is thought to inhibit presynaptic GABA (B) receptors in the nucleus accumbens (Berton et al., 1998). A German researcher has noted that this new anti-craving medication has no psychotropic side effects nor any potential for abuse or dependence. Acamprosate lacks hypnotic, anxiolytic, antidepressant, and muscle-relaxant properties (Zieglgaensberger, 1998). Although acamprosate is being used in clinical trials in the United States as of 2000, however, its effects are unclear. It appears to reduce the frequency of drinking, but its effects on enhancing abstinence are no greater than those of naltrexone.

AGENTS TO IMPROVE COGNITIVE FUNCTION

Chronic heavy drinking can lead to impairment of most cognitive functions, including abstract thinking, problem solving, concept shifting, psychomotor performance, and memory. The two most common diseases of cognitive impairment in alcoholism are alcoholic amnestic disorder (WERNICKE-Korsakoff syndrome) and alcoholic dementia. Alcoholic amnestic disorder is associated with prolonged and heavy use of alcohol and is characterized by severe memory problems. Though the exact cause is unknown, this disease is thought to be preventable by proper diet, including vitamins, particularly the B vitamin thiamine. The other impairment, alcoholic dementia, has a gradual onset and thus displays various degrees of cognitive impairment, including difficulties in short-term and long-term memory, abstract thinking, intellectual abilities, judgment, and other higher cortical functions.

Most studies indicate that alcoholics with impaired cognitive function will have poorer treatment outcome. This, of course, depends on the severity of impairment. Little research has been conducted with medications to improve cognitive function. Serotonin-uptake inhibitors have shown some promise in improving learning and memory. One study with the serotonin-uptake inhibitor fluvoxamine demonstrated improvement in memory in patients suffering from alcohol amnestic disorder, but not in patients with alcoholic dementia.

AGENTS TO TREAT PSYCHIATRIC DISORDERS CONCOMITANT TO ALCOHOLISM

Alcoholism may be accompanied with various psychiatric problems including anxiety, depression, antisocial behavior, panic disorders, and phobias. Part of the problem in treatment is to determine if the psychiatric disorder developed before alcoholism (primary), or after (as a result of) alcoholism (secondary). Nevertheless, several studies have been conducted predominately with medications used to treat depression and anxiety.

Agents to Treat Alcoholics with Depression. Depression has been associated with alcoholism, especially with relapse to drinking. A frequent pharmacologic treatment of depression is with a

group of medications called tricyclic ANTIDEPRESSANTS (desipramine, imipramine, amitriptyline, and doxepin). Their efficacy in treating alcoholics with depression is, however, largely unknown. This is in part because of poor methodological studies. A recent study of desipramine was conducted on alcoholics with and without secondary depression (Mason & Kocsis, 1991). Preliminary findings showed that desipramine is effective in reducing depression in the depressed group and may also prolong the period of abstinence from alcohol in both depressed and nondepressed patients. Preliminary results of another study suggested that imipramine both improves mood and reduces drinking in alcoholics suffering from major (primary) depression.

In addition to the tricyclic antidepressants, the serotonin-uptake inhibitors are used to treat depression. One of these inhibitors, fluoxetine (Prozac), is widely used as an antidepressant. As discussed earlier, fluoxetine has been studied to see whether it attenuates drinking behavior in nondepressed alcoholics, but findings as of 1999 indicate that its usefulness is limited to alcoholics in the dual-diagnosis population.

Lithium, an effective medication for the treatment of manic-depressive disease, has also been studied as a pharmacologic agent in the treatment of alcoholic patients. In one multi-site clinical study of lithium in depressed and nondepressed alcoholics, lithium therapy was not effective in reducing the number of drinking days, improving abstinence, decreasing the number of alcohol-related hospitalizations, or reducing alcoholism dependence (Dorus et al., 1989). This investigation as well as other studies did not address the effectiveness of lithium in other types of psychiatric disorders that may respond—including hypomania (a mild degree of mania), bipolar manic-depressive illness, and other mood disorders. Studies of lithium in the 1990s concluded that it lacks efficacy in the treatment of alcoholism.

Agents to Treat Alcoholics with Anxiety Disorders. Recent studies have indicated that a sizeable proportion of individuals who abuse alcohol also suffer from anxiety disorders. Buspirone, an agent commonly used to treat anxiety, has shown potential in reducing alcohol consumption. As discussed earlier, buspirone acts as an agonist on the serotonin 5-HT_{1A} receptors and also alters the dopamine and norepinephrine systems.

An attractive feature of buspirone is that its use does not lead to physical dependence on the drug, as with antianxiety drugs, particularly with BENZODIAZEPINES. Furthermore, buspirone lacks side effects often found with anxiolytic medications. For example, buspirone lacks sedative, anticonvulsant, and muscle-relaxant properties, does not impair psychomotor, cognitive, or driving skills, and does not potentiate the depressant effects of alcohol.

Administration of buspirone to rats and monkeys has resulted in a decrease in alcohol intake (Litten & Allen, 1991). In humans, one study reported that buspirone diminished alcohol craving and reduced anxiety. Another study found buspirone to be more effective with alcoholics suffering from high anxiety than those with low levels of anxiety. A third study on more severe alcoholic patients found no effect. Thus, further research is needed before this drug's efficacy can be accurately evaluated.

In summary, the evidence indicates that effective treatment of a psychiatric disease may also be beneficial to the treatment of alcoholism, particularly in alcoholics with coexisting psychiatric disorders, but that psychoactive medications are not "magic bullets" for most alcoholics.

CONCLUSIONS

Development of new medications to decrease drinking, prevent relapse, and restore cognition may have a role in alcoholism treatment in the future—but as a part of treatment regimens—given with other nonpharmacological therapies. Advances in understanding the mechanisms responsible for alcohol craving, drinking behavior, cognition, and even some of the psychiatric disorders such as depression and anxiety disorders have not yet produced a medication that substantially improves abstinence rates. Some researchers have recommended a careful matching of subgroups of alcoholics to the medications that are presently available as a possible pharmacological treatment strategy.

Moreover, as of 2000, there is much that is still not known about the pharmacological treatment of alcoholism. The 1999 NIAAA report outlined three major areas of inquiry that need further research:

The optimal dosing strategy for anti-alcohol medications and the optimal duration of treatment.

The possible utility of combination therapies, either combinations of different medications or combinations of medication and psychotherapy.

The usefulness of specific pharmacotherapies for women; different ethnic and racial groups; adolescent and geriatric patients; and polydrug abusers.

(SEE ALSO: *Complications; Disease Concept of Alcoholism and Drug Abuse; Drug Interactions and Alcohol; Drug Metabolism; Treatment, History of*)

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Cocaine, An Overview COCAINE abuse and dependence should be approached as chronic disorders that require long-term treatment. The clinical course of cocaine addiction is often progressive and generally marked by recidivism. Addiction to cocaine should be approached as a brain disease, and not a weakness to be viewed with judgmental overtones. In fact, cocaine produces a number of neurochemical alterations in the brain, especially in the reward centers of the midbrain and in the limbic system. When evaluating a patient for treatment,

many factors must be taken into consideration. First, patients presenting for treatment often have complicating factors, such as coexisting psychiatric disorders, family problems, job jeopardy, and medical complications. These problems are often why the person is seeking treatment, and should be fully explored and linked to the addiction. Interpersonal and occupational dysfunction often results from cocaine becoming the addict's number one priority, taking precedence over family and financial responsibilities. Medical problems frequently result from cocaine's destructive action on the heart, brain, and kidneys, while co-occurring psychiatric disorders commonly include paranoia, depression, and anxiety. To a great extent, the presence of these disorders depends on the length of time the individual has been using cocaine, the dose of cocaine taken, and the route of administration. As individuals progressively lose control over cocaine intake, they become more likely to experience interpersonal, medical and psychiatric complications.

COCAINE USE PATTERNS

Cocaine may be taken in various ways that differ in speed of onset, in blood levels, and, consequently, in brain levels. Subjective effects are most intense when brain levels of cocaine are rapidly increasing to high concentrations. Routes of administration, in ascending order of efficiency, are chewing COCA leaves (absorption through the mucous membranes of the mouth), oral ingestion of cocaine hydrochloride, intranasal absorption of cocaine hydrochloride, smoking of alkaloidal (FREEBASE) cocaine (CRACK), and intravenous injection of cocaine hydrochloride. The use of crack is actually the most rapid delivery of cocaine to the brain, and generally preferred over intravenous use.

There are also different use patterns. Some patients rarely use cocaine except at parties and in relatively low doses. Some ethnic and social groups are particularly likely to use cocaine by the intranasal route, a method that achieves lower brain levels than administration via crack (freebase inhalation) or the intravenous route. Women and adolescent users are more likely to use crack, which is inexpensive per unit dose. A vial of crack sufficient to produce a brief, intense period of euphoria averages two to three dollars in some large East Coast cities. Affordability essentially increases the access of this highly addictive drug to our youth, and to all other segments of our population. Many users tend to administer cocaine several times per week in intense bursts, or binges. A binge may last several hours or even several days. In these individuals the binge is usually terminated by exhaustion of supplies or by behavioral, cardiovascular, or neurological side effects. Binges are often perpetuated by the phenomenon of cocaine use producing additional cocaine CRAVING. It is not typical to see individuals able to maintain low or moderate doses of cocaine when used on a daily basis.

The higher the dose of cocaine reaching the nervous system and the longer the period of use, the more likely that there will be some form of behavioral toxicity. Personality change consisting of irritability, suspiciousness, and paranoia may occur. Psychosis with HALLUCINATIONS and persecutory delusions, often associated with the likelihood of violence, is also seen in heavy cocaine users. Auditory hallucinations are the most common, but tactile and gustatory hallucinations are occasionally reported. During the crash period after termination of a binge of cocaine use, there is often DEPRESSION. The period of depression is usually brief, but in some patients it can trigger a major affective disorder, which is a psychiatric syndrome requiring ANTIDEPRESSANT medication. Cocaine addicts often report suicidal thoughts, especially during the crash period. For most patients, cocaine WITHDRAWAL consists of several days of gradually decreasing depression and fatigue with episodes of craving for cocaine.

PHASES OF TREATMENT

Treatment can be divided into three phases: (1) achievement of initial abstinence or detoxification; (2) rehabilitation; and (3) aftercare. The treatment of cocaine abuse or dependence should always be thought of in terms of these phases, and the patient and the patient's family should be told to anticipate a period of treatment lasting at least eighteen months and often three years or longer.

Achievement of Initial Abstinence. Initial abstinence can be difficult to achieve if severe withdrawal symptoms are present, although most patients do not experience the cocaine "crash" because they use irregularly, stopping and restarting cocaine frequently. Although there is a definite cocaine withdrawal syndrome, it has an irregular pattern and does not fit neatly into distinct phases.

Careful studies of patients going through cocaine withdrawal reveal an early severe period of dysphoria, depression, fatigue, and sleepiness. Over the ensuing hours and days gradual improvement occurs. There may also be physical signs, such as a bradycardia (slow heart rate) that gradually returns to normal. These withdrawal symptoms may be accompanied by periodic severe craving for cocaine. If a patient is being treated on an outpatient basis, achieving abstinence can be very difficult.

To assist in the achievement of initial abstinence, researchers have attempted to identify medications that might help reverse brain alterations known to result from chronic cocaine exposure. DOPAMINE, a neurotransmitter involved in natural reward, appears to mediate the "high" associated with cocaine. There is substantial evidence that repeated cocaine use depletes brain dopamine, leading clinical investigators to test dopamine agents in cocaine patients. Bromocriptine stimulates dopamine receptors but is associated with side effects, and has not been proven effective in preventing RELAPSE. Few clinicians currently recommend its use to treat acute cocaine withdrawal. Another dopaminergic medication, AMANTADINE, has been researched in an outpatient study to help patients achieve initial abstinence. It is very important to evaluate potential medications for any disorder by using a comparison or control group. Typically a group of patients is randomly assigned to receive either the drug to be tested or a placebo. Patients are given identical-appearing capsules so that neither the patients nor the physicians know who is receiving the test drug and who is getting the placebo. Such a double-blind trial determined a significant advantage for patients randomly assigned to amantadine as compared with the group receiving a placebo. This advantage was found only during the initial two-week phase of treatment, when the goal is achievement of abstinence, and further research is underway at present to evaluate this dopamine agent. Another outpatient study found desipramine to be helpful in achieving early abstinence and maintaining it for six weeks. This was relatively early in the cocaine epidemic, and the patients were all intranasal users. More severely cocaine-dependent patients have generally failed to respond this well to desipramine.

Rehabilitation Phase. The major emphasis of treatment should be prevention of relapse to compulsive cocaine use. Some clinicians recommend inpatient treatment to establish abstinence and begin rehabilitation in severely addicted patients. Inpatient treatment by itself is never sufficient and must be followed by an outpatient phase of rehabilitative treatment during which time the patient has returned to his or her prior living environment. Outpatient treatment may be especially difficult if the patient lives in a drug environment and is subject to daily cues that trigger cocaine craving. Many clinicians recommend giving all patients an initial trial of outpatient treatment, reserving inpatient treatment only for those who repeatedly fail in less expensive outpatient programs. This approach is generally embraced by managed care organizations. In many areas of the country, access to inpatient treatment is only available for cocaine addicts with serious medical or psychiatric conditions.

Although the effectiveness of inpatient versus outpatient treatment is pertinent to millions of afflicted individuals, there has been surprisingly little actual research in this area. One study made a direct comparison between outpatient and inpatient rehabilitation. Patients at the Philadelphia Veterans Administration Medical Center were randomly assigned to either an 18-day inpatient rehabilitation treatment or outpatient rehabilitation that included a hospital day program. The hospital day program was similar to the inpatient rehabilitation program and based on the TWELVE STEPS with emphasis on group therapy and peer support. Some individual therapy was provided for both groups. Patients came to the day hospital five days per week for more than five hours of therapy per day, and returned home in the evening. Those in the inpatient program remained in treatment seven days per week, twenty-four hours per day. At the end of the twenty-eight-day program, both groups were encouraged to continue treatment in an aftercare program consisting of weekly visits to the outpatient clinic. At the end of four months and at the end of seven months, evaluations were conducted on all patients initialing the study, even if they had dropped out immediately after beginning. The results showed that there were fewer dropouts in the inpatient program, but there was no significant difference between the two groups. Both had a 50 to 60 percent success rate at the two follow-up periods. Success was defined as no cocaine use for the prior thirty days, supported by a negative urine

test at the time of the interview. This study has been cited as supporting the use of less expensive outpatient treatments for cocaine addicts.

Although some individuals are able to stop cocaine use and remain permanently abstinent, most experience slips to cocaine or other drugs. A slip does not necessarily denote relapse or treatment failure, provided the patient is willing to resume counseling and is interested in preventing subsequent use. Slips often occur when patients deviate from treatment recommendations, and treatment compliance can be reestablished in their aftermath. However, slips may turn into "runs" of heavier and heavier cocaine use, resulting in a decision to drop out of treatment and return to active addiction. This is the danger of a slip, and the basis of recommending total abstinence. The use of other addictive agents, such as OPIATES, ALCOHOL, SEDATIVES and MARIJUANA, should also constitute a slip. Although clinicians have recognized the need for abstinence from all addictive substances when treating cocaine patients, it has only recently been demonstrated in a research study that the use of alcohol leads to significantly lower recovery rates.

Based on knowledge of the pharmacological effects of cocaine, there has been an intensive search for medications that serve as effective adjuncts in the rehabilitative phase. Cocaine is known to block the dopamine transporter, a specialized membrane protein that clears cocaine from the synaptic space after it has been released, thus helping to terminate neurotransmission. Cocaine use consequently produces excessive dopaminergic stimulation, contributing to the pleasurable effects of the drug. Cocaine also increases the availability of other neurotransmitters, such as serotonin, norepinephrine, and glutamate. The search for a medication to improve the results of cocaine treatment has focused largely on substances that influence dopamine mechanisms, either presynaptic or at the receptor level, and medications that influence brain systems utilizing GABA, glutamate, and serotonin.

Unfortunately, the results of medication research have been disappointing. Desipramine was initially reported to be of some benefit in this phase of treatment, but subsequent studies involving severe cocaine dependence failed to replicate early reports of success. Carbamazepine was proposed as a treatment based on its ability to block the development of subcortical seizure activity produced by cocaine. Controlled studies, however, have failed to show any benefit for this anticonvulsant medication in prevention of relapse. Bromocriptine was not found to improve recovery rates when used in a relatively high dose, perhaps due to study dropouts motivated by excessive side effects. There have been claims of benefit for acupuncture, but there is no scientific evidence to support is efficacy in cocaine dependence. There have also been unsubstantiated reports in the lay literature that the hallucinogenic drug IBOGAINE produces a long-term loss of craving for cocaine. The lay press has reported three deaths from the use of this drug and animal studies report neuronal toxicity after ibogaine administration. Baclofen, a drug that indirectly affects dopamine neurons through GABA systems in the brain, may be effective against cocaine craving for theoretical reasons, and is currently under investigation.

Psychotherapy during Rehabilitation. In addition to standard treatments provided in most rehabilitation programs, such as the twelve-step program, group and family therapy, there have been studies using specific manual-driven psychotherapy and behavioral therapy. A recent report of a large-scale multi-center study demonstrated superior results with individual drug counseling. Furthermore, the effectiveness of individual drug counseling correlated highly with attendance in twelve-step group meetings.

Reinforcement of Clean Urine. Another treatment approach that has resulted in significant success is using systematic reinforcement of cocaine abstinence. Researchers arranged for patients to be rewarded with vouchers that could be exchanged for desirable goods, restaurant meals or other constructive purchases when they presented drug-free urine. This treatment approach was accepted well by patients, and the results were significantly better than those for a control group receiving counseling alone. A one-year follow-up of patients previously treated for six months in this manner showed that 71 percent were abstinent during the thirty days prior to the follow-up interview.

A similar study has been conducted with opiate-dependent patients who were using cocaine while enrolled in a methadone program. A program of reinforcement of clean urine using vouchers that could be exchanged for desirable objects produced a significant reduction in cocaine use. The use of vouchers to improve retention in treatment, and enhance recovery rates, is the focus of a large gov-

ernment-sponsored effectiveness study currently underway.

Extinction of Cocaine-Related Cues. Even highly motivated former cocaine-dependent patients experience craving after the cessation of cocaine use. While they are in a protective hospital environment, addicts often feel confident that they can remain abstinent. However, upon returning to their previous neighborhoods they encounter environmental cues that typically result in excitement and cocaine craving. These cues usually are people, places, and things that had previously been linked to cocaine use. Many patients say they become so conditioned to the effects of cocaine that simply seeing their drug dealer or a vial of cocaine produces a rush long before the drug gets into their body. Cue craving has recently been shown to produce a discernible signature of brain activity with the use of PET scanning. Treatments have been designed to reduce or extinguish these conditioned responses. They consist of repeatedly reviewing drug-related stimuli and learning various coping skills, such as the relaxation response, visual imagery, and mastery techniques. These techniques are used by behavioral therapists to reduce the symptoms of other disorders, such as phobias or obsessive-compulsive disorder. For cocaine dependence, the patient can be taught the techniques by a therapist. Later, the patient can practice the techniques in the clinic by viewing videos of cocaine use. There is now evidence that patients randomly assigned to these behavioral treatments do significantly better in outpatient treatment than control subjects assigned to standard treatment with the same amount of attention.

Aftercare. After about a month of intense rehabilitation treatment, a patient can graduate to an aftercare program of variable intensity. Sessions may initially be once or twice a week, decreasing gradually to once or twice per month. Urine testing should be continued to monitor drug use. The cocaine metabolite, benzoylecgonine, remains in the urine for several days and can effectively signal the resumption of cocaine use. Patients who admit to a slip or whose urine tests indicate cocaine use should resume intensive counseling. Every attempt should be made to determine why the slip occurred so that it can be avoided in the future. As previously discussed, a slip of this nature should not necessarily be considered indicative of treatment failure, even if it results in a significant binge. It is instead a sign that the patient needs to resume intensive treatment for a chronically relapsing disorder. Most clinicians agree that regular daily attendance in twelve-step groups should supplement professional treatment, at least for the first 90 days of recovery. Thus far, there is no evidence that any medication is helpful in this phase of treatment. Of course, if the patient remains depressed or anxious, or has symptoms of another psychiatric disorder, specific treatment such as antidepressants should be employed.

SUMMARY

Cocaine abuse and dependence represent chronic disorders that require long-term treatment. A brief initial inpatient phase may be necessary, but the major part of treatment consists of long-term outpatient care. Since cocaine addiction is associated with progressive deterioration in functioning, and can produce dangerous medical and psychiatric complications, aggressive treatment is warranted. Various treatment techniques can be used. Most patients receive group therapy and counseling based on the Twelve Steps developed by ALCOHOLICS ANONYMOUS. Professional psychotherapy may be helpful in selected cases, but data are still preliminary. There are also data showing efficacy for behavioral treatments, such as contingent voucher reinforcement of clean urine and extinction of cue craving produced by cocaine-related stimuli. Still, recovery rates from cocaine dependence are disappointingly low, and treatment approaches are being refined. Cocaine use tends to occur in epidemics, especially when there is little perceived danger of it. We appear to be experiencing a dramatic reduction in cocaine use, perhaps because cocaine is widely perceived as dangerous. Therefore, the most effective means of treating cocaine dependence may ultimately involve education of its risks directed toward individuals not yet caught in its grasp.

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Cocaine, Behavioral Approaches No consensus exists about how to treat COCAINE dependence. This statement is particularly alarming given that in 1998 it was estimated that 1.8 million persons in the United States were dependent on cocaine. The abuse of cocaine was first recognized in the medical literature in the late 1800s. Early proposed treatments included various herbal and medical potions, nutritional supplements, hot baths, substitution of MORPHINE, long stays in sanatoriums, education, and psychotherapy. Systematic evaluation of the effectiveness of these early treatments did not occur.

The goals and focus of behavioral approaches for cocaine dependence vary greatly depending on the beliefs held by the treatment provider regarding the causes of cocaine dependence. The efficacy of the various treatments is only beginning to be evaluated. This article describes the primary behavioral approaches used to treat cocaine and discusses the efficacy of those interventions. Although numerous behaviorally-based interventions are being used as treatments for cocaine dependence, this article is limited to providing an overview and discussion of approaches that have received attention in the scientific literature.

OUTPATIENT VERSUS INPATIENT TREATMENT

Studies suggest that inpatient rehabilitation is not cost-effective in most cases of cocaine dependence. It is also not necessary in most cases because withdrawal from cocaine addiction is not physically dangerous, nor does it cause an incapacitating reaction. However, inpatient treatment may be indicated in some instances of cocaine dependence if the patient (1) fails to make progress or deteriorates during outpatient treatment; (2) has severe medical or psychiatric problems; (3) is physically dependent on other drugs, or (4) has a history of criminal involvement. In general, learning to cope with the multitude of environmental circumstances that have contributed to the initiation and maintenance of cocaine abuse is the most important task of the abuser. This task can be accomplished effectively only outside the hospital.

Therapeutic communities, or residential programs with planned lengths of stay of six to twelve months, focus on the resocialization of the individual to society. Resocialization programs at such communities may include vocational rehabilitation and other supportive services. One study has shown that improved cocaine relapse rates for patients with medium- to high-level problems were dependent on longer treatment stays.

COCAINE ANONYMOUS

COCAINE ANONYMOUS (CA) is a community-based self-help group organization modeled after ALCOHOLICS ANONYMOUS (AA). The basic principles are the same as AA's. The program is based on the "disease" model of substance dependence. Achievement and maintenance of abstinence from cocaine is presumed to be facilitated by following the Twelve Steps of CA (which are based on the original TWELVE STEPS of AA).

CA is available to anyone who expresses a desire to stop using cocaine and all other mind-altering substances. All that is necessary to become a group member is that one attend meetings. Meetings vary from large open ones that anyone can attend to small, closed discussions reserved for specific groups. For example, a group of young people, professionals, or women is organized to address specific concerns. At most meetings, experiences are shared and advice and support are given. Two

other components of the CA program are sponsorship and education. A sponsor is a person who has been in recovery for a substantial period of time and who is available at any time to provide support and guidance to the person attempting to recover. Education about the "disease" is provided through pamphlets, books, films, and other literature. CA is recommended by many treatment professionals as the treatment for, or as an important adjunct of treatment for, persons with cocaine problems.

GROUP THERAPY

Many professionals suggest that group therapy is an invaluable component of cocaine abuse treatment. Most groups are structured to include persons of different backgrounds and at different stages of recovery (1) to help deal with feelings of uniqueness, (2) to expose those in the early stage of treatment to positive role models, and (3) to help instill hope for success. Those who promote group therapy view peer pressure and support as necessary to overcome ambivalence about abstaining from cocaine. Providing support for others and the development of intimate social interaction (e.g., sharing of feelings) is facilitated and presumed to be therapeutic.

Topics of discussion in group therapy vary depending on the group members and the orientation of the therapist. Topics may include early abstinence issues, guilt resolution, marital conflict, or lifestyle changes. Education about adverse effects of cocaine is often included. Group therapy occurs in outpatient or inpatient settings. It is sometimes used as the sole source of treatment or combined with individual counseling and other treatment components. Researchers have acknowledged a number of possible limitations to group therapy. They include loss of confidentiality for the individual, likelihood of avoidance of group therapy because of social anxiety, and negative peer influences.

Research on the efficacy of group vs. individual therapy alone or in combination continues. A European review of 22 controlled outcome studies regarding comparisons between individual and group psychotherapy treatments in general found that there is no superiority of one treatment over the other. The study noted, however, that group therapy has an economic advantage over individual therapy. Another study has shown that there are no significant differences in demographic, personality, or addiction severity variables or in treatment retention or 9-month outcome between cocaine abusers who choose individual therapy and those who choose group therapy.

SUPPORTIVE-EXPRESSIVE AND INTERPERSONAL PSYCHOTHERAPY

Psychotherapy is usually suggested as a component of cocaine-treatment programs, both inpatient and outpatient. Typically, the therapy is based on psychodynamic theories of substance abuse. This means that intrapersonal factors and underlying personality disturbances are considered causes of cocaine abuse. It is presumed that cocaine is used to cope with painful emotional states, and that issues such as separation-individuation, depression, and dependency must be resolved to maintain abstinence. The therapist tends to adopt an exploratory role that promotes insight into interpersonal and intrapersonal conflict underlying the cocaine dependence. Increased insight is presumed to result in a reduction in the underlying problems, which, in turn, should help promote cocaine abstinence.

The psychotherapeutic approaches for cocaine abusers are generally similar to the approaches for abusers of other drugs, although treatments for ALCOHOLISM and drug abuse have evolved somewhat differently and the models used may conflict at certain points. A great deal of discussion has been generated about these conflicts in combined treatment for alcohol-and drug-dependent patients, but, overall, the literature is positive about the merits of combining approaches.

One common type of psychotherapy for cocaine dependence is supportive or supportive-expressive psychotherapy. This therapy in combination with pharmacotherapy has demonstrated some efficacy in research with HEROIN-dependent persons. Initially, supportive psychotherapy focuses on acknowledging the negative consequences of cocaine use, accepting the need to stop using, and helping manage impulsive behavior. The therapist and user explore ways to stay away from other users and high-risk environments. The focus of treatment then shifts to insight-oriented psychotherapy in which the therapist's role is to facilitate the exploration of underlying reasons for the cocaine abuse. Long-term abstinence depends on the degree to which the underlying psychic disturbances are re-

solved. A study from the 1990s has led some researchers to conclude that low-intensity psychotherapy was ineffective with the majority of their subjects.

Interpersonal psychotherapy (IPT) was originally developed for and found to be effective with DEPRESSION and was adapted for opiate addicts and, later, cocaine abusers. This psychotherapy for substance abusers is based on the premise that drug abuse is one way in which an individual attempts to cope with problems in interpersonal functioning. An exploratory stance focuses on interpersonal relationships and the impact of drug abuse on these relationships. In helping the patient stop his or her substance abuse, the practitioner selects the important components of treatment. They may include documenting the adverse effects of the drugs compared with their perceived benefits, identifying the thoughts and behaviors that precede drug use, and developing strategies to deal with drug-related cues and high-risk situations. Only after attaining abstinence are interpersonal difficulties directly addressed, including the roles of drug use in these relationships.

A key strategy with IPT is to develop more productive means for achieving the desired social gratification or tension reduction for which the drug abuse substitutes. In a multiple drug abuser, this substitution may differ markedly for various drugs. For example, the abuser may be using cocaine to reduce social isolation and to "meet exciting new people" but may be abusing alcohol because the cocaine "crash" is reduced by the alcohol. Since only the cocaine, and not the alcohol, is directly related to the social deficit, only the cocaine abuse will directly benefit from interpersonal therapy. In general, the interpersonal impact will be somewhat different for the abuse of licit drugs such as alcohol, illicit drugs such as heroin and cocaine, and drugs such as benzodiazepines. Among cocaine addicts, for example, the licit drugs such as alcohol are often used in response to interpersonal tension, while the illicit drugs such as heroin lead to consequences of increased interpersonal tension, rather than being used in response to tension. In summary, IPT must identify the relationship of each particular drug to the interpersonal setting as either primary association or secondary to other drug effects and as either a tension reliever or inducer.

COGNITIVE AND BEHAVIORAL THERAPY

Behavioral perspectives of cocaine dependence view drug taking as a learned behavior that begins and continues because of the reinforcing effects of the drug. These reinforcing effects are determined, in part, by basic biological events in the brain. This means that, to some extent, most persons are susceptible to becoming dependent because cocaine produces a reaction in the brain that increases the likelihood that drug taking will recur. The other factors that determine whether a person will become dependent on cocaine are environmental factors (e.g., peers, acceptance by others, and no apparent negative consequences). Research has clearly demonstrated that cocaine seeking and use are learned responses that occur regularly under specific conditions (e.g., certain times of day, events, internal states). This outcome translates into treatment that focuses on changing these "using" conditions and creating new conditions that encourage abstinence from cocaine.

Cognitive and behavioral therapy is a behavioral approach to treating cocaine dependence that is often conducted through group therapy. The idea behind the therapy is to make drug use less attractive and to create alternatives to drug use by changing an individual's internal and external environment. Some therapy is modeled on techniques that individuals have used themselves to abstain from using or cut back on cocaine use. The approach attempts to help patients to recognize situations in which they are most likely to use cocaine, to avoid these situations when appropriate, and to cope more effectively with problems and problematic behaviors associated with drug abuse. For example, individuals learn how to cope with boredom, anger, frustration, and depression, and how to handle social pressure to use drugs. Sometimes individuals rehearse social situations in therapy sessions, to better equip them for handling such situations when they encounter them. Individuals are also urged to give up other drugs, especially alcohol, because of its association with promoting cocaine use and its effect on weakening one's resistance to use. The possibility of a lapse is acknowledged in this therapy and ways to deal with temporary lapses in abstinence are covered so that the individual can work to prevent total relapse. Family and friends are also encouraged to join therapy groups

as many researchers believe that such support is one of the most effective ways to promote abstinence. Cognitive and behavioral therapy is considered particularly useful because of its compatibility with a range of other treatments patients may receive, such as pharmacotherapy.

A behavioral therapy component that is showing positive results among many cocaine-addicted individuals is contingency management. Contingency management uses a voucher-based system to give positive rewards for staying in treatment and remaining cocaine free. Based on drug-free urine tests, the patients earn points, which can be exchanged for items that encourage healthy living, such as joining a gym or going to a movie and dinner. Some vouchers can also be exchanged for retail goods.

Another contingency-based method that sometimes works is CONTINGENCY MANAGEMENT. With this method, the cocaine addict writes a letter that contains a damaging admission of cocaine use. The addict then agrees that the letter can be made public if his or her urine shows up cocaine-positive after testing. Researchers believe that this type of negative incentive may be effective among cocaine users who have something to lose, as in good employment. Such incentive therapies have shown that cocaine use can be influenced by manipulating the consequences of using.

Another behavioral approach focuses on the conditioned stimuli (environmental events) associated with cocaine use and the way those events affect relapse and deter abstinence attempts. This approach focuses intensely on the persons, places, and things that have frequently been paired with cocaine use. Theoretically, things like drug-using friends, paraphernalia, white powder, and places where cocaine is used can produce cravings for cocaine and ultimately result in cocaine use. Therefore, with repeated exposure to those events under conditions where cocaine is not available (i.e., an extinction procedure), the events gradually lose their ability to elicit the cocaine craving and presumably reduce the probability of cocaine use.

One other behavioral approach that has received increasing attention is Relapse Prevention Treatment (RPT), originally formulated for treating alcohol dependence. RELAPSE PREVENTION requires specific interventions based on precipitants that have been identified as associated with the risk of returning to abuse of a specific drug. These precipitants, which include negative emotional states, interpersonal conflict, social pressure, and specific drug-related cues, may be quite different for different drugs of abuse. For example, in a methadone-maintained patient, the precipitants for using heroin or cocaine may be closely related to being with particular ''friends'' and then ''getting high.'' This ''getting high'' on heroin can be pharmacologically blocked by large doses of METHADONE; large methadone doses will not have a similar effect on cocaine use. Self-monitoring is used to identify risk situations for the specific drug, and then coping strategies are developed using rehearsal of coping behaviors such as anger management and social skills. Preventing relapse focuses on ensuring that brief lapses to cocaine use do not become full relapses. A lapse may be seen as a discreet isolated event that is not uncommon in recovery and that does not nullify all progress. Reduction of this ABSINENCE VIOLATION EFFECT by reframing the concept in this way may work with all drugs of abuse, although in multiple-drug abusers, sequential lapses in each drug must be prevented by carefully emphasizing the importance of abstinence and not giving ''permission'' for experimenting with isolated use of the various abused drugs.

In the first test of its efficacy with cocaine dependence, RELAPSE PREVENTION was superior to IPT in retaining individuals in treatment and in facilitating greater rates of cocaine abstinence. A second trial of RPT provided additional support for its efficacy. One-year follow-up data showed RPT to be superior to case management in facilitating higher levels of cocaine abstinence. In a study that compared standard group counseling (STND) with individualized relapse prevention (RP), individuals who commited themselves to a goal of absolute abstinence on starting a continuing care program had better cocaine use outcomes in RP than in STND. However, individuals with looser abstinence goals fared better with STND.

Another two behavioral approaches, coping-skills training (CST) and neurobehavioral treatment, have received support as potentially effective treatments. CST is similar to RPT in that it involves teaching specific drug refusal and coping skills important for accessing alternatives to drug use and for coping with events that place the abuser at high risk. One year-long study found that during the first six months of the study individuals who had CST and relapsed used cocaine on significantly

fewer days than did the control group using meditation and relaxation as a coping skill. The study was conducted in the context of high-risk situations. Both groups did equally well in the final 6 months.

Neurobehavioral treatment emphasizes many of the elements of RPT and coping-skills training to assist the abuser to abstain from cocaine and avoid relapse. The "neuro" prefix denotes specific treatment focus on difficulties that may arise due to the neurobiological changes that accompany abstinence from cocaine.

MOTIVATIONAL THERAPY

Researchers have noted a high dropout rate in most studies of addiction treatment and that of those who do remain in treatment, most succeed in breaking the habit. As a result of this success among those who remain in treatment, some researchers believe that the commitment to change from addictive behavior is the greatest factor affecting improvement in the cocaine-dependent individual. Motivational therapy takes advantage of this desire for change and is designed to help addicts realize the extent of their problem and help increase their desire to quit. It also prepares them for other treatment. Motivational elements used in such therapy are described by the acronym FRAMES (feedback, responsibility, advice, menu of options, empathy, and self-efficacy).

ECLECTIC TREATMENT

Many treatment providers use an eclectic approach to treat cocaine dependence; that is, a combination of approaches. For example, many programs based on a disease or a psychodynamic model may use certain behavioral procedures such as contingency contracting or relapse prevention strategies.

In a collaborative cocaine treatment study conducted by the National Institute on Drug Abuse, researchers found that group drug therapy plus individual drug counseling was more effective than cognitive therapy plus GDC, supportive-expressive therapy plus GDC, or GDC alone.

In general, a limitation of eclectic approaches is that mixed messages may be given to the patient. Moreover, the intensity and quality of each component may not be as high as approaches that are more unilateral in focus. For example, behavioral approaches spend a great deal of time counseling and assisting the abuser to make the behavioral changes needed to achieve and maintain abstinence. Eclectic approaches may spend only a small portion of time on those changes. The small time spent focused on those changes may not be sufficient to facilitate change, and it may give the abuser the message that those changes are relatively unimportant.

CONCLUSIONS AND FUTURE DIRECTIONS

There is no one treatment for cocaine abuse that has proven more effective than any other. The treatment of cocaine addiction is complex, and it must address a variety of problems. Like any good treatment plan, cocaine treatment strategies need to assess the psychobiological, social, and pharmacological aspects of the patient's drug abuse, and it is important to match the best treatment regimen to the needs of the patient. Programs that provide several treatment options may prove the most effective.

Evaluating programs for cocaine addiction has proven difficult. There are a number of limitations inherent in many cocaine addiction studies that prevent researchers from drawing strong conclusions from the work; these limitations have included self-selection of treatment, the lack of urinalysis data, insufficient follow-up time, a lack of independent evaluation, and the unreliable information provided by the addicts themselves.

Research continues on specific issues that may influence treatment outcome. These issues include (1) the use of other drugs including ALCOHOL, (2) the presence of other psychiatric problems, and (3) the severity and duration of the abuse. In general, researchers believe that recovery from cocaine addiction will be difficult unless the individual has something to lose and unless the individual believes that he or she has the power to change and make positive choices.

(SEE ALSO: *Adjunctive Drug Taking; Causes of Substance Abuse; Disease Concept of Alcoholism and Drug Abuse; Treatment Types*)

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Cocaine, Pharmacotherapy The pharmacological treatment of COCAINE abuse is defined as the use of medication to facilitate initial abstinence from cocaine abuse and to reduce subsequent relapse. The initiation of abstinence from cocaine abuse involves reduction in the withdrawal symptoms associated with cessation of cocaine. This WITHDRAWAL syndrome resembles depression but includes a great deal of anxiety and craving for cocaine. CRAVING for cocaine often persists for several weeks after abstinence has been attained, and places or things associated with cocaine use in the past, called *cues*, can continue to stimulate cocaine craving for many months. Because of this persistence of what is known as *conditioned craving*, relapse to cocaine abuse can occur after the patient has become abstinent. Preventing relapse is an important function of medication treatment.

An objective of the use of medications in cocaine dependence is to reverse changes that are caused in the brain after chronic cocaine use. These brain changes, called *neuroadaptation*, have been demonstrated in animal models of cocaine dependence. Chemical analyses of animal brains exposed to cocaine chronically show abnormalities in the NEUROTRANSMITTER receptors on brain cells. The brain cell receptors that are affected by cocaine include DOPAMINE receptors and SEROTONIN receptors (Harvard Mental Health Letter, December 1999). Neurotransmitters such as dopamine and serotonin may be involved in the conditioned craving that creates the risk of relapse. Researchers are also looking for hereditary factors that may determine individual differences in susceptibility, which may lie in genes that control the manufacture of neurotransmitter receptors (Harvard Mental Health Letter, December 1999).

Direct and indirect evidence that there are changes in brain receptors can be found in human studies. Prolactin is a hormone that is controlled by the neurotransmitters dopamine and serotonin. In some heavy cocaine abusers, prolactin levels are

abnormally high after abuse has stopped and remain elevated for a month or more. This evidence suggests that both dopamine and serotonin brain systems are perturbed by cocaine and that the abnormality persists for some time. Other evidence of persistent abnormalities in the dopamine systems comes from brain imaging studies directly examining dopamine receptors. Positron-emission tomography (PET) studies have shown a marked reduction in dopamine receptors on brain cells that are ordinarily very rich in such receptors. This abnormally low amount of dopamine receptors persists for at least two weeks after a patient stops using cocaine. That several medications may reverse these neurochemical receptor changes has been an important rationale for their use.

In addition to direct biological indicators of neuroadaptation, neuropsychological tests have documented sustained deficits in thinking, concentration, and learning among chronic cocaine abusers. These deficits may persist for weeks after cocaine use has stopped. Researchers believe that some neuropsychological deficits may be related to reduced blood flow to the brain in abusers. One PET study showed reduced cerebral blood flow in patients that had been given cocaine (American Journal of Drug and Alcohol Abuse, May 1999).

The biological abnormalities in the brains of abusers clinically may be manifest by a characteristic withdrawal syndrome. The very early phases of this syndrome, commonly called the "crash," may involve serious psychiatric complications, such as paranoia with agitation and depression with suicide. These complications require medications for symptomatic management, including ANTIPSYCHOTIC agents, such as chlorpromazine and haloperidol, or large dosages of BENZODIAZEPINES to calm highly agitated patients. Many patients self-medicate these crashes using such sedating substances as benzodiazepines or alcohol. Because this crash phase is usually relatively brief, rarely lasting more than several days, there is generally no role for sustained medication. The more important role for medications occurs during the later phase of withdrawal from cocaine, which may persist for several weeks. This later phase resembles a depressive syndrome, with substantial anxiety and craving to use cocaine. The neurobiological changes noted in both human and animal studies after chronic cocaine use correspond in time to the occurrence of this syndrome. This temporal correspondence has provided a further rationale for the use of ANTIDEPRESSANT medications in the treatment of cocaine dependence and withdrawal.

A wide range of pharmacological agents besides antidepressants have been tried as treatments for cocaine abuse and addiction. In general, agents include drugs that affect the production, release, reabsorption, and breakdown of dopamine, serotonin, and other neurotransmitters (Harvard Mental Health Letter, December 1999). Researchers are also evaluating medications that work as a vaccine to prevent the effects of cocaine (Vaccine Weekly, May 4, 1998).

Combination pharmacotherapies are also being researched for cocaine-dependent individuals who abuse other substances. Multiple-drug abuse in cocaine abusers often involves problems with ALCOHOL, OPIOIDS and/or BENZODIAZEPINES. The medical consequences of using these drugs in various combinations are often more severe than using each drug alone, and combinations of treatment options may be needed for many of these drugs. Specific treatments may include pharmacotherapies targeted toward cocaine as well as other drugs of abuse, such as NALTREXONE for opioid abuse and DISULFIRAM for alcohol abuse. Opioid-derived medications have also been explored. The use of opioid-derived medications to treat cocaine dependence has an ironic twist, because Sigmund Freud had suggested that cocaine might be an appropriate treatment for morphine (an opioid) addiction. Clearly substituting one drug of abuse for another drug of abuse is a risky treatment approach, but new ideas are emerging on the use of opioids with lower abuse potential than morphine, such as BUPRENORPHINE for patients dependent on both opioids and cocaine.

Evaluation of medications in controlled studies using double blinding and random assignment is very important, because a substantial placebo response may occur in cocaine abusers when they enter treatment, even if they are given a simple sugar pill. In double-blind, placebo-controlled studies, neither the patient nor the physician knows whether the patient is receiving active medication or placebo. Controlled studies provide the clearest indication of an efficacious medication when it is found to be significantly better than a placebo given to similar patients in a randomized and blinded manner. Randomization simply means that patients who are potential subjects for a study are

randomly assigned to get either the active medication or the placebo. Choices about who will get active medication and who will get the placebo are made by chance alone and not decided by the physician based on drug-abuse severity or any other criteria. In uncontrolled tests, patients are given the medication and their response is compared with their behavior before starting treatment.

ANTIDEPRESSANTS

In controlled studies, several antidepressants have been found superior to placebo. One such antidepressant was desipramine. Desipramine was felt to promote cocaine abstinence by reducing craving. In one study of the efficacy of desipramine, cocaine use declined several weeks before cocaine craving was reduced. This delay suggested that desipramine reduced the recurrence of craving after cocaine abstinence had been attained, and thus its anticraving action might be more important for the prevention of relapse than for the initiation of abstinence. One pilot study suggested that another another antidepressant, venlafaxine, may be an effective treatment for patients with a dual diagnosis of depression and cocaine dependence (American Journal of Drug and Alcohol Abuse, February 2000).

DOPAMINERGIC AGENTS

In theory, dopaminergic agents may be useful in ameliorating early withdrawal symptoms after cocaine binges, because these agents appear to have their onset of action within a day of starting. These agents include AMANTADINE, bromocriptine, and METHYLPHENIDATE. Bromocriptine has been studied by several groups of investigators and has shown efficacy for some and not for others. Several trials have examined amantadine at 200 and 300 milligrams (mg) daily and found that it reduces craving and use for several days to a month. Methylphenidate was shown effective in reducing cocaine cravings in cocaine users with attention-deficit/hyperactivity disorder (ADHD). One theory for addiction among ADHD cocaine abusers is that they are medicating themselves. Methylphenidate acts on receptors like cocaine, but it acts much more slowly (Harvard Mental Health Letter, December 1999). Side effects have limited the utility of several other dopaminergic agents.

MISCELLANEOUS AGENTS

A number of other agents have been utilized to treat different aspects of cocaine abuse and dependence. Several authors report a decrease in euphoria and/or paranoia with such neuroleptics (ANTIPSYCHOTIC medications) as flupenthixol. Neuroleptics are said to reduce the activity of dopamine (Harvard Mental Health Letter, December 1999). Flupenthixol may be particularly useful as a treatment for cocaine abusers with schizophrenia (American Journal of Drug and Alcohol Abuse, August 1998).

Studies have begun on the development of a cocaine vaccine designed to suppress the psychoactive effect of the drug. Such a vaccine works by producing antibodies that bind to cocaine in the bloodstream and prevent it from traveling to the central nervous system, thus neutralizing the effect of the drug. Studies have found that it was possible to override the effects of the vaccine with massive amounts of cocaine, but researchers believe that such consumption would be unlikely with addicts actively working to overcome addiction. Researchers have viewed the vaccine as a complementary therapy to behavioral therapy.

MULTIPLE-DRUG USE

According to the National Institute on Drug Abuse, most cocaine-dependent people abuse other substances. More than half are alcohol dependent. Opioid and sedative dependency has also been widespread over the years. The reasons for cocaine abuse by heroin addicts are to "improve" the euphoria from heroin. These findings suggest that control of heroin abuse in many patients may directly reduce cocaine abuse, and the reduction in cocaine abuse reported by several surveys of methadone-maintenance programs support this assertion.

Combination pharmacotherapies of cocaine anticraving agents with methadone or naltrexone for heroin addiction and with disulfiram or naltrexone for alcoholism have been tried with some success. While buprenorphine, a mixed opiate agonist-antagonist, and methadone have been effective in reducing opiate use, further studies are required to substantiate efficacy in reducing cocaine use in opiate addicts. However, one small study showed that buprenorphine in combination with desipramine or

amantadine facilitated some cocaine abstinence. Buprenorphine and disulfiram was also found more effective than buprenorphine alone in treating heroin addicts with a cocaine habit (*Alcoholism and Drug Abuse Weekly*, June 19, 2000). Disulfiram is used in the treatment of alcohol addiction, and taking it before using cocaine may block the pleasurable effects of cocaine and invoke such negative effects as anxiety and paranoia, effects that may help discourage cocaine use (*Alcoholism and Drug Abuse Weekly*, June 19, 2000). The antidepressant desipramine also shows some promise in promoting opioid and cocaine abstinence in opioid-maintained patients (Oliveto et al., September 1999).

An important clinical need with patients dependent on opiates, alcohol, or sedatives in addition to cocaine is for detoxification. While cocaine withdrawal is not associated with major medical complications, withdrawal from these other drugs can be medically significant and often needs specific pharmacological interventions.

(SEE ALSO: *Causes of Substance Abuse; Drug Metabolism; Research, Animal Model*)

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Drug Abuse: 2000 and Beyond Drug addiction is a medical and public health problem that affects everyone, either directly or indirectly. A recent study estimated that drug abuse and addiction cost the United States more than $110 billion per year. If one adds the cost of nicotine to this figure, the number dramatically soars. Improved

prevention and treatment are the best ways to reduce that cost. Fortunately, advances in science have revolutionized our fundamental understanding of the nature of drug abuse and addiction, and what to do about it.

Extensive data show that addiction is eminently treatable if the treatment is well delivered and tailored to the needs of a particular patient. There is an array of both behavioral and pharmacological treatments that can effectively reduce drug use, help manage drug cravings and prevent relapses, and restore people as productive members of society.

Three decades of scientific research and clinical practice have yielded a variety of effective approaches to drug addiction treatment. Extensive data document that drug addiction treatment is as effective as treatments for most other similarly chronic medical conditions. In spite of scientific evidence that establishes the effectiveness of drug abuse treatment, many people believe that treatment is generally ineffective. In part, this is because of unrealistic expectations. Many people equate addiction with simply using drugs, and they therefore expect that addiction should be cured quickly and permanently, and view treatment is a failure if it is not. In reality, because addiction is a chronic disease, the ultimate goal of long-term abstinence often requires sustained and repeated treatment episodes.

Drug-abuse treatment programs using medications and/or behavioral techniques can and do work. The most successful treatment programs are a complex mix of medical, psychosocial and rehabilitation services that attempt to deal with the unique needs of each individual. However, effectiveness of treatment can differ because of complex variables such as the type(s) of drug(s) to which a person is addicted, the dysfunctional lifestyles of many addicts, and time and treatment resources available to addicts and treatment personnel. Many Americans affected by drug addiction have been restored to healthy and productive lifestyles through appropriate treatment.

NEW AND IMPROVED TREATMENTS

The National Institute on Drug Abuse (NIDA) has already made considerable progress in developing a variety of effective behavioral and pharmacological addiction treatments and making them widely available to the public. For example, NIDA has taken the lead in developing readily available nicotine addiction therapies. They have also brought to the world the most effective medications to date for heroin addiction, including methadone and LAAM (levo-alpha-acetylmethadol), and have standardized behavioral interventions that have been effective in treating both adults and adolescents.

NIDA supports research to develop additional new and improved pharmacological and behavioral treatments. To this end, NIDA sponsors both a medications development program and a behavioral therapies development program. NIDA's medications development program brings the critical mass of knowledge of medicinal chemistry, molecular biology, brain function, and behavior to bear on the urgent public health problem of drug addiction to provide new medications as an effective adjunct to conventional treatment by helping to stabilize addict and allow them to succeed in their overall treatment program. Specifically, new medications are being researched to:

block the effects of abused drugs;
reduce the craving for abused drugs;
moderate or eliminate withdrawal symptoms;
block or reverse the toxic effects of abused drugs;
or prevent relapse in persons who have been detoxified from drugs of abuse.

Because behavioral interventions are the most common, and sometimes the only, treatments administered to individuals with drug addiction, NIDA also has a robust behavioral therapies development program to complement its medications portfolio. Researchers are working to develop new behavioral treatments for drug abuse and addiction and enhance the efficacy of existing ones. Psychotherapies, behavior therapies, cognitive therapies, family therapies, and counseling strategies are among the approaches currently being studied under this program. Once these treatments are proven to be safe and effective in small trials, they will be tested in larger and more diverse populations through NIDA's new National Drug Abuse Treatment Clinical Trials Network. This network will enable the rapid, concurrent testing of a wide range of promising science-based medications and behavioral therapies across a spectrum of real-life

patient populations, treatment settings, and community environments.

CONCLUSION

Addiction is a treatable disease. However, there is no "one size fits all" treatment program. Treatment is typically delivered in outpatient, inpatient, and residential settings, all of which have been shown to be effective in reducing drug use and are appropriate for a specific type of patient. Drug addiction treatment can include behavioral therapy (such as counseling, cognitive therapy, or psychotherapy), medications, or a combination of both. Behavioral therapies, such as cognitive behavioral coping skills treatment, offer addicts ways for coping with their drug cravings, teach them to avoid drugs and relapse, and help them deal with relapse if it occurs. The best programs provide a combination of therapies and other services, such as referral to other medical, psychological, and social services, to meet the needs of the individual patient.

ALAN I. LESHNER

Heroin, Behavioral Approaches Psychological treatments are an important component of comprehensive drug-abuse treatment. Medications such as METHADONE can be used to address physical dependence and other biological aspects of addiction, but HEROIN abuse is also a disorder involving maladaptive learned behavior that must be stopped and replaced by healthier behaviors. Psychological therapies help drug abusers to understand their feelings and behaviors and to make changes in their lives that will lead to ending drug use and maintaining abstinence. Drug abusers also may have psychiatric problems, such as DEPRESSION and ANXIETY, and they may have problems interacting with other people or dealing with anger and frustration. These problems can also be addressed by psychological therapies. In addition, heroin abuse is a chronic relapsing disorder (i.e., many people who try to stop end up returning to drug use). Relapse to drug use following treatment is commonly attributed to environmental (e.g., associating with drug-using friends), psychological (e.g., feeling depressed or angry), and/or behavioral (e.g., having poor social skills) factors that are typically the focus of psychological interventions.

A variety of psychological treatments, often in combination with pharmacological approaches, have demonstrated effectiveness in the treatment of heroin abuse. The purpose of this article is to survey the most prominent psychological interventions currently used in the treatment of heroin abusers. Following a brief discussion of the development of heroin abuse, we describe the factors that lead people to seek treatment, the range of problems that may be characteristic of heroin abusers, and the psychological treatments—including THERAPEUTIC COMMUNITIES, motivational incentive therapies, counseling, psychodynamic and cognitive-behavioral psychotherapies, family therapy, and SELF-HELP approaches. The chapter concludes with a discussion of the effectiveness of these interventions.

DEVELOPMENT OF HEROIN ABUSE

Initial heroin use is motivated by curiosity and the desire to use it without becoming addicted. Heroin is injected into a vein (although it is sometimes inhaled), and the user experiences an immediate rush, characterized by feelings of relaxation and well-being. As use escalates, withdrawal symptoms (e.g., cramps, irritability) may appear as the drug is eliminated from the body. At this point, individuals may start using the drug both for its positive effects and for alleviating uncomfortable withdrawal symptoms. Drug use may also be motivated by an attempt to cope with feelings of STRESS, hopelessness, or depression. Whatever the causes of initial use, the frequent and repeated acquisition of heroin soon becomes a priority; some addicted individuals may resort to illegal activity (e.g., stealing; prostitution) to buy illicit drugs. In addition heroin abusers are often concurrently addicted to ALCOHOL and/or other drugs, including COCAINE and BENZODIAZEPINES (e.g., Valium, Zanax) that they may have started taking before or after they began using heroin. It is in the context of this addictive lifestyle that heroin abusers come to the attention of treatment providers. Heroin abusers are usually ambivalent about seeking treatment; they like taking drugs and have difficulty seeing any reason to stop. They are most likely to begin treatment following a crisis of some sort—a legal, physical, family, financial, or job-related

problem caused by their drug use. They are typically referred to specific treatment sites by friends, family, or the legal system, which may mandate treatment as a part of probationary sentences. The cost, location, and availability of treatment slots are all factors that affect selection of treatment setting.

TREATMENT SETTINGS

Treatment for heroin dependence is offered in publicly funded clinics that accept patients with limited resources, including those who receive public assistance. It is also treated in private programs that take patients with higher incomes and/or medical insurance. Treatment for heroin abuse is often defined by the setting in which it is delivered, not by the actual content of treatment, which may or may not differ across treatment settings. For example, outpatient and inpatient clinics may offer remarkably similar services for drug abusers. One exception is the THERAPEUTIC COMMUNITY, where the treatment philosophy and approach are uniquely associated with long-term recuperation in a residential setting. Treatments are also labeled with regard to the relative role of psychological versus pharmacological interventions used. With METHADONE MAINTENANCE, for example, counseling and psychotherapy are viewed as secondary, although complementary, to the daily oral administration of methadone—a drug that replaces heroin within the dependence mode. At the opposite end of the spectrum are residential therapeutic communities and TWELVE-STEP self-help programs, in which the entire intervention consists of social and behavioral modeling, with no use of medications. Drug-abuse treatment may also be distinguished by whether it is offered in a hospital versus a community clinic outpatient setting. Outpatient clinics usually emphasize psychological techniques, by providing counseling and psychotherapy services. Hospital chemical dependency units usually offer medical detoxification that involves prescribed medications along with some combination of psychological approaches. These detoxification services are important for helping heroin- dependent people make the transition to a drug-free state. However, it is also important that they continue in treatment at the same or another state program after the detoxification has been completed. Those who follow this recommendation are more likely to remain abstinent and to continue working on the lifestyle changes needed for long-term successful outcomes. In this chapter, we will describe the content of psychological interventions for heroin abuse independent of the settings in which they are typically administered.

ASSESSMENT

By the time drug abusers seek treatment, they often have a number of problems that need to be solved, only the first of which is stopping drug use. Within any treatment setting, comprehensive assessment is essential to focus treatment on the areas where change is needed. It is first important to understand the types and amounts of drugs that are typically taken in order to assess the severity of the drug-abuse problem. Drug-use information is assessed through the patient's self-report and urinalysis testing. Urinalysis testing provides objective information about whether the individual has or has not used drugs recently and can also be used to verify the truthfulness of self-reports. An understanding of psychological and environmental factors that precede and follow drug use (e.g., when, where, and why drugs are taken; where and how the drugs are acquired), known as a functional analysis, is also necessary for the development of strategies to initiate abstinence and prevent relapse. Evaluation of psychiatric disorders is essential for determining appropriate treatment intervention. Depression and ANTISOCIAL PERSONALITY, for example, are quite common among heroin abusers (Brooner et al., 1997). Some problems, however, such as depression, may go away when drug use stops. Finally, social functioning, employment history, and illegal activity all have implications for psychological interventions and treatment prognosis and need to be thoroughly assessed. Indeed, being employed and having good social support (e.g., from a spouse who does not abuse drugs) are excellent predictors of treatment success if they are already present, and areas that need attention in treatment if they are not. The ADDICTION SEVERITY INDEX (ASI; McLellan et al., 1992), a structured interview that assesses drug use, physical and emotional health, employment, social support, and legal status, is often used by clinicians and researchers to evaluate the broad range of factors that are related to drug abuse and may improve with treatment.

PSYCHOLOGICAL AND BEHAVIORAL TREATMENTS OF HEROIN ABUSE

This section will survey common psychological and behavioral approaches to the treatment of heroin abuse. Although each differs in regard to its philosophy and goals, all share an interest in eliminating the drug use of the heroin abuser and the substitution of healthier behaviors.

Therapeutic Communities. Therapeutic communities (TCs) are long-term (6–24 month) residential programs developed specifically for helping drug abusers change their values and behaviors in order to sustain a drug-free lifestyle. The assumption behind these communities is that drug abusers, who have typically been involved in a special illicit sub-culture for most of their lives, need to learn how non-drug-abusing individuals function in society. The goal is to rehabilitate the drug abuser into a person who can conform to society's values and goals, assume social and job responsibilities, and make contributions to the community. During treatment, the drug abuser lives in a special residential community with other drug abusers and with therapists who may be ex-addicts in recovery. A behavioral shaping/incentive system is set up so that desirable behaviors are rewarded through community privileges and increased responsibilities. In addition, patients learn through observing peers and staff, who serve as role models for appropriate behavior, sometimes called "right living."

Patients progress through three stages. In the first stage, orientation (0–2 months), the patient assimilates within the therapeutic community by attending seminars concerning the philosophy and rules of the program. The second stage is called primary treatment (2–12 months) and characterized by increasing work responsibilities and group leadership roles. This stage includes three phases. In the first phase (2–4 months), patients conform to the TC policies by following the rules, engaging in low-level work assignments, and attending group meetings. By the second phase (4–8 months), patients work at more responsible jobs, actively participate in group meetings, and begin to assume the responsibility of a role-model for other patients. In the third phase (8–12 months), patients engage in top-level jobs (e.g., coordinating services in the program), colead support and treatment groups, and become social leaders in the community. The final stage, reentry (12–24 months), focuses on preparing the patient to separate from the TC and rejoin the outside community. It is expected that after leaving patients will establish their own households and obtain regular employment or continue their education. In summary, TCs attempt to rehabilitate the drug abuser by instilling a whole new set of attitudes and behaviors that conform to those expected by a non-drug-abusing society. Treatment programs modeled after therapeutic communities are becoming increasingly popular for implementation in prison systems. Typically, prisoners with a drug-abuse history are invited to join the program 6–12 months prior to their scheduled release date. In most successful programs, involvement with residential treatment continues after release from prison, a time when prisoners most need help with reentering the community and establishing a drug-free lifestyle.

Drug-Abuse Counseling. This intervention approach is practiced in methadone maintenance programs, where patients are required to see a counselor throughout the course of treatment—and may also be provided in outpatient community-clinic programs. Counselors are usually professionals with a college degree in counseling, although ex-addicts who have personal experience with recovery from drug abuse may also provide counseling. Counselors have several roles. First, they monitor treatment compliance (that the patient is attending regularly and providing urine specimens for drug testing as requested), confront any violations of program rules, and enforce penalties and privileges. Second, based on problems and deficits identified during the assessment phase, counselors formulate a treatment plan that specifies goals for the patient. For example, a treatment plan may contain recommendations to abstain from drug use, obtain employment, and participate in self-help groups. Counselors work with their patients using several strategies to implement such a treatment plan. Goal setting helps patients learn to set reasonable goals that will lead to a responsible drug-free life (e.g., finding a job, starting a bank account, obtaining a driver's license) and to outline specific steps required to attain chosen goals. In problem-solving training, counselors and patients work together to address both immediate and long-standing problems in the patient's life. The primary goal is for patients to learn the strategies for solving everyday problems and for making decisions. Rec-

reational planning may be used to encourage patients to engage in new social and recreational activities that might substitute for their typical lifestyle of searching for drugs or hanging out with drug-using friends. Finally, counselors are expected to refer patients to other community-helping agencies for services that they cannot provide themselves. For example, patients who are unemployed may be referred to an employment-counseling service. In summary, counseling attempts to comprehensively address the problems of drug abusers using practical, goal setting, and problem-solving techniques.

Motivational Incentive Therapy. The goal of motivational incentive therapy is to offer a therapy that can more effectively compete with the powerful enticement of drugs and make abstinence a more attractive option. It does this by offering immediate and tangible benefits to the addict for remaining abstinent. In a motivational incentive program, drug abusers in treatment can earn points that are worth money each time they submit a urine sample that tests negative for specified drugs (e.g., heroin and cocaine). The incentive program is designed to promote sustained abstinence. To do this, the number of points earned for each consecutive drug-free sample increases over time and "resets" to the original lower number if the patient relapses to use and submits a drug-positive sample. In general, the more money that is offered, the more successful the incentive program. For example, in some of the most successful research programs, patients have been able to earn up to $1000 if they remained continuously abstinent for 3 months. Although this amount may seem high, it is reasonable compared to the costs of continuing drug abuse to society. Patients like the incentive program because they can use the money earned to improve their life. For example, they can pay bills or exchange gift certificates for groceries and other retail items. The incentive program is not intended to last indefinitely; 3 to 6 months is typical. However, the program helps keep patients in treatment and promotes abstinence. During periods of sustained abstinence engendered by an incentive program, counselors and clients can work on making the lifestyle changes that will promote more enduring abstinence after the incentive program ends.

Psychotherapy. This type of psychological treatment, usually practiced by trained clinical psychologists, psychiatrists, or psychiatric social workers during a one-on-one interaction with the patient, uses interpersonal skills to promote insight and behavior change. Psychotherapy was developed for use with neurotic and emotional disorders, but has been adapted for use with drug abusers. Several specific types of psychotherapy are practiced by various therapists, depending on their training, with psychodynamic and cognitive-behavioral being two prominent types. In each of these therapies, comprehensive assessment, empathic listening, nonjudgmental understanding, and patience are necessary tools to help the patient become involved in a therapeutic relationship and provide a context for behavior change.

Psychotherapy can also be practiced in groups, and group treatment is frequently defined as a separate type of treatment. Groups are a popular way to conduct treatment and may be found in virtually any treatment setting, including hospital and outpatient chemical dependency programs, methadone programs, and therapeutic communities. The content of therapy, however, can vary widely from one group to another in the same way that differing approaches are used for individual psychotherapy. Regardless of therapeutic approach, group therapies do differ from individual therapies in some specific ways. Groups provide a context for mutual empathy, encouragement, and support among people who share similar problems. Patients in groups may benefit from the experience of others in solving these problems and by entering reciprocal helping relationships. The interactions among group members also provide a context in which the therapist can facilitate improved social skills for those who may need them.

Psychodynamic Therapy. Psychodynamic therapy with heroin abusers employs supportive, analytical techniques to explore heroin use and the addictive experience from the patient's point of view. Drug use is viewed as a symptom of underlying emotional problems and/or relationship difficulties. Thus, psychodynamic therapy rarely confronts or attempts to modify drug use directly, and for this reason, it is usually implemented after stable abstinence from drugs has been achieved. Therapy focuses instead on the patient's thoughts, feelings and relationships (past and present) with parents, spouse, friends, and other significant individuals—from which the therapist tries to identify common patterns or themes. As therapy progresses, the therapist-patient relationship becomes the focal point,

as this relationship often replicates themes from interactions with others, which the therapist points out. The primary means of behavior change results from the patient recognizing these common, often maladaptive, interaction themes and determining to change them. Thus, the goal of treatment is for the patient to understand the origin and function of their feelings and behavioral patterns, and to use this awareness to change the manner in which they cognitively interpret, emotionally respond, and behaviorally interact with individuals in their environments. For example, a psychodynamic therapist might observe that anger is a continuing theme in a patient's life and be sensitive to situations when the patient shows anger toward the therapist. When this happens, the therapist will help the patient understand the circumstances leading to the anger and relate these circumstances to other situations when the patient had been angry. Eventually, the patient and therapist might explore the origins of the patient's anger (perhaps toward his or her parents) and the relationship between the patient's anger and engaging in self-destructive behavior (e.g., drug use). As the patient develops more adaptive ways of coping with thoughts, emotions, and relations to others, heroin and other substance use becomes less necessary and desirable. In summary, psychodynamic therapy views long-term abstinence from drug use as an indirect result of resolving the causes of drug use. In this way, it is believed that a more permanent cure will result.

Cognitive-Behavioral Therapy: Relapse Prevention. Cognitive-behavioral therapists are concerned with direct interventions that will change behavior and thinking without necessarily requiring or expecting insights into the causes of behavior. Recognizing that relapse is a serious problem in drug abuse, these therapy approaches have been specifically adapted for use with heroin and other drug abusers in a therapy called Relapse Prevention, to teach them the skills necessary to initiate and sustain abstinence (Marlatt & Gordon, 1985). A functional analysis derived in the assessment phase allows the therapist to understand the thoughts, behaviors, and environmental conditions that precede and follow heroin and other drug use and to help the patient recognize the environmental (e.g., drug-using friends), cognitive (e.g., irrational thinking), emotional (e.g., anger), and behavioral (e.g., starting arguments) factors that may either reduce the likelihood of stopping or increase the likelihood of returning to drug use. Based on this functional analysis, the cognitive-behavioral therapist and the patient decide which factors (e.g., thoughts, places, people) are most likely to sustain ongoing drug use or act as triggers for relapse during abstinence; then specific treatments are based on this analysis (Carroll et al., 1994).

Patients and therapists may work together to devise strategies for avoiding drug-using friends and staying away from places in which the patient has bought and used drugs in the past. In some cases, patients may even want to change their phone numbers or move to new locations. In addition to environmental changes, heroin abusers may be taught new skills designed to help them cope with high-risk situations that could trigger relapse. For example, patients who use drugs when they feel stressed may be taught specific relaxation techniques that can counteract stressful feelings. Patients may also learn drug-refusal skills to handle situations where they actually encounter drugs (although it is better to avoid such situations altogether) and to use specific strategies for coping with situations in which the return to drug use is likely (e.g., calling a nonusing friend; leaving the situation; making an appointment with their therapist). In addition, cognitive-behavioral therapists may address the patient's thought patterns that precede heroin use and call attention to dysfunctional thinking. For example, patients may have unrealistic thoughts ("I must be loved and accepted by everybody or else I am a failure and might as well use drugs") or illogical thoughts ("I will never be able to stop using drugs because I am an addict"). The cognitive-behavioral therapist aims to change negative cognitions to adaptive, positive thinking ("I do not need everybody's approval"; "I can learn to gain control over my behavior").

Sometimes a pervasive maladaptive behavior pattern underlies drug abuse that can be addressed with a cognitive-behavioral approach. For example, with a patient who has trouble controlling anger and tends to use drugs after angry confrontations, the cognitive-behavioral therapist may place the patient on an anger-control skills-training program. The patient would be instructed to avoid situations likely to induce anger (e.g., confrontations with a supervisor) and would be taught specific strategies for dealing with potential anger-producing situations. For example, relaxation might be employed to gain control over anger. Fur-

ther, the patient might be taught new self-statements to replace thoughts that have typically preceded feelings of anger (e.g., "It would be nice to get a raise, but it isn't the end of the world if I do not get it"). In summary, cognitive-behavioral therapy focuses directly on behavior change without expecting or requiring insight into the cause of the problem. To the extent that underlying emotional and interactional dysfunctions often exacerbate drug use, however, both the cognitive-behavioral and the psychodynamic therapist will end up dealing with the same issues—albeit in slightly different ways.

Family Therapy. Heroin abusers are often raised in dysfunctional families and may replicate the maladaptive behavior patterns learned from their families within their own personal and romantic relationships. In addition, the patient's heroin abuse may have had a disruptive effect on that family. These observations suggest the importance of including the family in the treatment process, and this is particularly true for adolescents who become involved with drugs while still living with their families. For older drug abusers, it is often difficult to involve the family in treatment, and family resistance/avoidance is one of the first issues that the therapist must address. Family therapy is a specialized type of psychotherapy that has its own methods, in which practitioners must be trained. Thus, it is generally conducted by a psychologist or other health professional who has been trained in one of several specific familial treatment approaches. Although there are several theoretical perspectives to family therapy (e.g., psychodynamic, cognitive-behavioral, family systems, etc.), the goals of these types of interventions are to help the family recognize maladaptive patterns of behavior, to learn better ways of solving family problems, to better understand each other's needs and concerns, and to identify and modify family interactions that may be helping to maintain drug use in the targeted family member (or members).

Self-Help Groups. ALCOHOLICS ANONYMOUS (AA) was created in 1935 by recovering alcoholics so that alcoholics could help each other abstain. NARCOTICS ANONYMOUS (NA) and COCAINE ANONYMOUS (CA) were later based on the tenets of AA but geared toward drug addictions. The newest group is Methadone Anonymous (MA), which accommodates drug addicts who use methadone. The core beliefs espoused by self-help groups are commonly adopted by many treatment programs, and drug-abuse patients are often referred to self-help groups as an adjunct to other treatments. Active members of self-help groups attend frequent meetings, some as often as once per day. At these meetings, members speak to each other about their drug use and drug-related problems; they offer mutual advice and support without the help of any trained therapists.

The philosophy, treatment goals, and procedures of self-help groups are contained in a book called *The 12 Steps to Recovery*. This book, often referred to as "The Big Book," outlines a series of tasks designed to promote abstinence and long-term recovery among alcoholics and drug abusers. The first step in recovery is to admit that one has a problem with drugs and/or alcohol and that outside help is needed to solve the problem. The sources of help to be called upon are other group members and a higher spiritual power (e.g., God), who will supply the spiritual strength necessary to stop drug use. The twelve-step program also advocates specific practical changes in lifestyle; these revolve around regular and frequent attendance at group meetings and concentration on the goal of abstinence (e.g., remembering the motto "one day at a time"). Once stable abstinence is achieved, the drug user is encouraged to restore relationships with friends and family that have been damaged by former drug use. For some, however, the self-help community becomes the primary source of friendships and social support.

Sponsorship is another technique used to promote and sustain abstinence. Specifically, all group members are encouraged to work with a sponsor who is typically an older, long-standing, group member who models appropriate behavior, guides new members through the twelve-step process, and provides a source of support for the new member to turn to in times of crisis. Later, the new member may sponsor someone else. To the extent that self-help programs permit former drug abusers to receive support from peers, associate with new groups of non-drug-using friends, and engage in alternate recreational activities with newly developed social contacts, the goals and even processes are similar to therapy. However, these goals are accomplished through group support and modeling using a treatment plan laid out in the twelve-step code rather than through formal meetings with a professional therapist.

EFFECTIVENESS OF PSYCHOLOGICAL TREATMENTS FOR HEROIN ABUSE

An end to drug use is the primary outcome measure for evaluating the effectiveness of drug-abuse treatment. Urine testing is usually included as a routine part of any drug-abuse treatment, to provide objective information on whether the treatment is being successful at motivating the patient to stop drug use and maintain abstinence. Changes in criminal behavior, employment status, family problems, and physical and emotional health are also relevant to understanding the effectiveness of treatment. Many of these collateral difficulties improve once drug use is stopped, although more improvement would be expected in treatment programs that offer services to specifically address these collateral problems. Using this array of outcome measures, studies have been conducted to evaluate the relative efficacy of treatments for heroin abusers. These studies have typically focused on the treatment setting rather than the content of treatment that is delivered within each setting. Further, some treatment settings have received much more evaluation than others. Methadone maintenance and TCs, for example, have received lots of attention, whereas hospital chemical-dependency programs have been infrequently evaluated and self-help programs have not been evaluated at all (Gerstein & Harwood, 1990).

Large scale followup studies such as the Treatment Outcome Prospective Study (TOPS), the Drug Abuse Treatment Outcome Study (DATOS), and the Drug Abuse Reporting Program (DARP), which have surveyed outcomes from methadone, therapeutic community, and outpatient modalities, have found that drug abusers who enter treatment display less drug use and better social adjustment during and following treatment than they did prior to treatment and also have better outcomes than groups of patients who applied for treatment but never followed through (Hubbard et al., 1989: Simpson & Sells, 1990; Simpson & Curry, 1997). These studies also found that effectiveness does not seem to be related to type of treatment but rather to duration of stay in treatment. Several types of treatment can be effective, but only with those patients who remain for prolonged periods of time. Thus, methadone maintenance and therapeutic-community treatments produce similar degrees of success with those who stay—but more patients tend to stay in methadone than in TC treatment. Finally, the success of drug-abuse treatment in general is better for patients who exhibit the fewest psychiatric symptoms and the greatest social stability (McLellan, 1983).

When evaluation focuses on treatment setting rather than on treatment content, it becomes difficult to determine which components of treatment are responsible for outcome results. This is especially true since treatment programs for heroin abuse are typically comprehensive and multimodal, encompassing a variety of techniques that may include psychological and behavioral interventions, medications, and self-help. The few well-executed studies that have attempted to evaluate the impact of specific psychological interventions on heroin abusers have been conducted with methadone maintenance programs. These studies have shown that methadone-maintenance treatment outcome is enhanced by a variety of psychological interventions, including counseling (McLellan et al., 1988, 1993), individual psychotherapy (Woody et al., 1983), family therapy (Stanton & Todd, 1982), cognitive-behavioral/relapse prevention aftercare (McAuliffe, 1990), and motivational incentive/contingency management therapy (Higgins, et al., 1993; Petry, 2000; Silverman et al., 1998) as evidenced by reduced drug use and crime, plus improved social and psychological functioning.

SUMMARY

Research has shown that several different types of treatment for heroin abusers can be effective. Heroin abusers who enter treatment do better than those who apply but do not follow through with treatment. Heroin abusers who remain in treatment the longest achieve better treatment outcomes than those who drop-out early. In addition, heroin abusers who exhibit the fewest psychiatric symptoms and demonstrate the most social stability appear to benefit most from treatment. Finally, specific psychological interventions have enhanced the effectiveness of methadone maintenance treatment. As previously noted, heroin abuse is a chronic, relapsing disorder: It appears that long-term treatment and perhaps repeated treatment may be necessary to eliminate drug use and to successfully address the broad range of psychosocial difficulties that usually accompany this disorder.

(SEE ALSO: *Addiction: Concepts and Definitions; Causes of Substance Abuse; Coerced Treatment for Substance Offenders; Drug Testing and Analysis; Opioid Dependence; Opioid Complications and Withdrawal; Tolerance and Physical Dependence; Treatment, History of; Treatment Types; Wikler's Pharmacologic Theory of Drug Addiction*)

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Heroin, Pharmacotherapy HEROIN abuse has been a social problem for many years. Heroin was trademarked after its first synthesis and use by the Bayer pharmaceutical company in Germany in 1898. It is derived from MORPHINE, the natural alkaloid complex that is found in opium. Although heroin is taken into the body by a number of routes, the most common is injection. The rapid absorption of injected heroin into the bloodstream causes a large "high" and a "rush," at first (before tolerance occurs), and all the heroin is absorbed by this route.

Another method, smoking heroin, has been called "chasing the dragon," perhaps as an allusion to Chinese opium smoking; in this method, heroin is placed on a metallic foil and a match lit under it. When the heroin vaporizes, the vapor is inhaled

through a straw; liquid heroin rolls around on the foil—hence the chase. A third method of heroin use, which waxes and wanes in popularity as the purity of illicit street heroin changes, is insufflation (snorting). This method minimizes the risks of intravenous drug use, including blood-borne infectious diseases such as hepatitis and HIV/AIDS, but it does not produce a rush because absorption into the bloodstream is slow. Heroin can also be injected into a muscle or under the skin (known as skin popping).

At first, heroin users have few lingering effects after a dose. The drug effects wear off after about six hours. Over time, however, addicts develop tolerance to the dose and dependence on the drug. Addicts will begin using heroin because they see people (friends, family, peers, role models) using it or because they feel a need to try it. As the frequency of use increases, they begin to experience withdrawal symptoms when they are not using the drug. At this point, they are physically dependent on heroin and will require larger and larger doses of heroin to achieve the same high or any high at all. Many addicts report that tolerance develops to such an extent that they cannot use enough for a high but must continue to use it to just feel normal (i.e., not be in withdrawal). It takes several weeks for a naive user to become dependent with this type of regular use.

HISTORICAL OVERVIEW OF TREATMENTS

When heroin was first commercially marketed by the Bayer Company as a morphine-like cough suppressant, it was thought to have fewer side effects than morphine. It was also used in the "treatment" of morphine addiction since it enters the brain more rapidly than does morphine. Instead, heroin introduced a new, more potent addiction. An over-the-counter industry in the legal sale of morphine and codeine elixirs also existed until opiates were outlawed by the HARRISON NARCOTICS ACT of 1914 and subsequent laws were passed during World War I (1914–1918).

Treatment of heroin abuse in the United States was initially targeted at removing the drug user from the environment of use. The federal prison in Lexington, Kentucky, became the site where incarcerated heroin addicts in federal custody were sent. Much of the current knowledge about opiate abuse was gained from the careful observations and carefully controlled studies of the researchers there. After incarceration, the addicts often returned to their towns of origin, and most of them turned back to drug abuse. The resulting clinical observation has been that imprisonment alone (with no drugs available) is an ineffective treatment of heroin abuse.

Historically, many of the medications used to treat heroin withdrawal in the general public have been largely ineffective; in some cases, the cure has been worse than the disease. Among the numerous ineffective treatments have been Thorazine, BARBITURATES, and electroshock therapy. In one method, belladonna and laxatives were used, because of the incorrect supposition that narcotics needed to be "rinsed" from the bodily tissues in which they were stored. At one institution that used this treatment, six of 130 addicts died during such opiate detoxification. Commenting on these methods, two of the researchers at Lexington noted: "The knockout feature of these treatments . . . doubtless had the effect of holding until cured many patients who would have discontinued a withdrawal treatment before being cured, and the psychological effect of doing something for patients practically all the time has a tendency, by allaying apprehension, to hold them even though what is done is harmful" (Kolb & Himmelsbach, 1938). Since the research conductetd at Lexington from the 1930s to the 1950s, which showed that opiate withdrawal was not fatal (unless complicated by other disorders or treatments), more standardized methods of detoxification have been developed.

A true advance was the development of methadone as a long-acting, orally effective opioid. Methadone was developed in Nazi Germany and was given the trade name Dolophine by the Eli Lilly company (from *dolor*, pain). The advantages of methadone over heroin include methadone's effectiveness when taken by month; its long action, which allows single daily doses; and its gradual onset and offset, which prevents the rapid highs and withdrawal seen with heroin. Methadone-maintenance treatment was developed in the 1960s in New York City and has become an accepted treatment for opioid dependence. With the discovery that HIV infection can be transmitted by intravenous drug users, the benefits of methadone in decreasing intravenous heroin use have become even more evident.

PHARMACOLOGICAL TREATMENT APPROACHES

The most common and first-line treatment approach is to try to get the addict to stop using heroin by detoxification. *Detoxification* refers to using medications to treat withdrawal symptoms. The heroin withdrawal symptoms are similar to the symptoms of a severe flu. Although these withdrawal symptoms are rarely medically dangerous for those in good health, they are extremely uncomfortable, and, in many addicts, they make the alternative, using heroin, more attractive than detoxification. Severe withdrawal is associated with signs of sympathetic nervous system arousal as well as increased pulse, blood pressure, and body temperature. Addicts experience sweating, hair standing on their arms (i.e., gooseflesh—hence the expression "cold turkey"), muscle twitches (from which the expression "kicking the habit" comes), diarrhea, vomiting, insomnia, runny nose, hot and cold flashes, and muscle aches. A host of psychological symptoms accompany the withdrawal distress. After addicts have been detoxified, they may be treated with medications that make it less likely they will use heroin again; these medications that prevent relapse may work by blocking heroin's effects. Medications can also be used to treat underlying psychiatric problems that contributed to the addict's use of drugs.

An alternative approach is METHADONE MAINTENANCE, which does not initially aim to stop the addict from using opioids but instead to substitute oral methadone use for heroin abuse. Methadone is a clear liquid, usually dissolved in a flavored drink, that is given once a day and is prescribed by a physician. Used as a way to treat addicts' withdrawal symptoms and drug craving, the prescription of methadone is closely controlled by state and federal regulations.

Opiate Detoxification. The simplest approach to detoxification is to substitute a prescribed opioid for the heroin that the addict is dependent on and then gradually lower the dose of the prescribed opioid. This causes the withdrawal to be less severe, although the withdrawal symptoms may last longer. A typical procedure entails first verifying that addicts are dependent on opioids (by some combination of observed withdrawal, a withdrawal response to naloxone, or evidence of heavy opioid use). The addicts are then given an appropriate dose of methadone, which treats the withdrawal symptoms. They are monitored for oversedation due to methadone or undermedication of withdrawal symptoms. Intravenous users of street heroin admitted to the hospital usually tolerate well a starting methadone dose of 25 milligrams. The methadone dose is then gradually lowered over the next several days. It is typical to taper a starting methadone dose of 25 milligrams over a period of seven days.

Another approach avoids the difficulties of prescribing an opioid to an addict. It involves using the antihypertensive CLONIDINE to treat withdrawal symptoms after the addict has stopped using the opiates. Clonidine suppresses many of the physical signs of opiate withdrawal, but it is less effective against many of the more subjective complaints during withdrawal such as lethargy, restlessness, and dysphoria. Clonidine's side effects of low blood pressure, sedation, and blurry vision make it unpleasant to take and unlikely to be abused by addicts. Although clonidine has not been approved by the Food and Drug Administration for opiate detoxification, it is widely used for this purpose and has demonstrated efficacy. It is most effective when used in addicts who are not addicted to large doses of opioids.

Opiate Antagonists. The opiate antagonist NALTREXONE is used clinically to accomplish rapid detoxifications and to help detoxified addicts stay off opioids. Naltrexone binds more strongly than heroin to the specific brain receptors to which heroin binds. If, therefore, addicts who are dependent on heroin take a dose of naltrexone, the naltrexone will replace the heroin at the brain receptor and the addicts will feel as if all the heroin has been suddenly taken out of their body. The effect of this rapid reduction in effective heroin (at the receptor) is withdrawal. The withdrawal is usually more severe than that which comes from simply stopping the heroin, but it also has the effect of accomplishing a detoxification more quickly. Thus, a combination treatment of clonidine to suppress the intensity of withdrawal symptoms and naltrexone to accelerate the pace of withdrawal has been used for rapid detoxification.

Naltrexone is primarily used after detoxification to prevent addicts from returning to opioid use. Because naltrexone binds to opioid receptors more tightly than does heroin, opioid addicts on naltrexone who use heroin will find the heroin effect

blocked by naltrexone. Addicts maintained on naltrexone who use heroin will only be wasting their money. One effect of naltrexone is thus to extinguish the conditioned response to heroin injection. Naltrexone is prescribed in the form of a pill that can be given as infrequently as three times a week. It has few side effects in the majority of patients who take it, and, contrary to some rumors, it does not suppress other "natural highs."

Opioid Maintenance. Methadone is the most common opioid used for the maintenance treatment of opioid addicts. Methadone satiates the heroin user's craving for heroin in order to prevent heroin withdrawal. The more important therapeutic effect of methadone, however, is tolerance to it. Addicts maintained on a stable dose of methadone do not get high from each dose because they are tolerant to it. This tolerance extends to heroin, and methadone-maintained addicts who use heroin experience a lesser effect because of the tolerance. Tolerance accounts for the fact that methadone-maintained addicts can take methadone doses that would cause a naive (i.e., first-time) drug user to die of an overdose. Generally, methadone-maintained addicts do not appear to be either intoxicated or in withdrawal. Tolerance is admittedly incomplete, and methadonemaintained addicts have some opioid side effects that they do not become tolerant to—for example, constipation, excessive sweating, and decreased libido. There is no known medical danger associated with methadone maintenance, however.

Methadone is dispensed as part of licensed programs, usually on a daily basis. It is generally well received by addicts, and the risk of incurring withdrawal symptoms if methadone treatment is interrupted provides a strong incentive for addicts to keep appointments. The ritual of daily clinic attendance has the additional therapeutic benefit of beginning to impose structure on the chaotic lives of most opiate addicts. Methadone treatment is often augmented with medical, financial, and psychological support services to address the many needs of opioid addicts.

Despite the philosophical debates about the appropriateness of using methadone, there is a large body of evidence indicating that methadone-maintained addicts show decreases in heroin use, crimes committed, and psychological symptoms. The major drawbacks to methadone maintenance include the great difficulty of achieving detoxification from methadone, the methadone side effects, and the possibility of increased use of other illicit drugs such as cocaine.

An opiate addict initially coming in for treatment will usually be put through detoxification and possibly put on naltrexone maintenance. Addicts with intact family supports, good jobs, or strong motivation are more likely to benefit from naltrexone maintenance than those who are more impaired. Younger addicts and adolescents are urged to try nonmethadone approaches, so as to avoid developing a methadone addiction. Methadone maintenance is usually reserved for patients who have failed at previous detoxifications. An exception is made for pregnant women, in whom methadone maintenance is the treatment of choice, with detoxification of the infant from methadone accomplished after birth. Opiate detoxification is risky in pregnant women because of the adverse effects on fetal development in the first and second trimesters, and the risk of miscarriage.

Other nonmethadone medications for maintenance treatment of opioid dependence have not yet been widely used. BUPRENORPHINE is a partial opioid agonist medication that has the advantages of being safe, even at higher doses, and being associated with less severe withdrawal symptoms than methadone after discontinuation. Another medication recently approved for treating opioid dependence is LAAM (levo-alpha-acetylmethadol). LAAM is broken down in the body to very long-acting active metabolites, and therefore it can be prescribed as infrequently as three times a week.

THE INTEGRATION OF PHARMACOLOGICAL AND PSYCHOSOCIAL TREATMENTS

No medication will prevent an addict who wants to use heroin from doing so. Naltrexone maintenance can be discontinued, and addicts who discontinue it are able within one to three days to use heroin without the naltrexone blockade. Similarly, methadone maintenance is ineffective in addicts who are unable or unwilling to meet the requirements of clinic attendance (which sometimes requires payment of fees) and staying out of prison. Addicts whose lives are in disarray require medications as part of a comprehensive treatment program that also addresses their other needs. In a street addict who chronically uses drugs, these may

include needs for counseling, medical attention, vocational rehabilitation, and a host of other services. There is evidence that methadone treatment is more effective if a higher "dose" of psychosocial treatment is provided along with it.

Detoxification is a first step toward recovery because it makes the addict available to further psychosocial and medical treatments. There is evidence that mild physiological abnormalities due to withdrawal of opiates linger for as long as three months after detoxification. This "long-term abstinence syndrome" is thought to contribute to the craving for opiates that occurs after detoxification. Naltrexone maintenance is most effective in addicts who have jobs and stable social supports—for example, in anesthesiologists who have become addicted to hospital medications. Because naltrexone itself is not reinforcing and many heroin addicts have a host of psychosocial problems, many clinics have reported that naltrexone maintenance alone was minimally effective in the treatment of long-term addicts.

SUMMARY

Opioid addiction is, in many ways, a physical problem as well as a psychological and behavioral problem. Addicts become physically addicted to opiates and, in the later stages of addiction, become preoccupied with relieving the physical symptoms of withdrawal. They become highly attuned to the bodily signals that withdrawal is coming. Heroin addicts spend most of their waking life procuring, using, and withdrawing from heroin—three times a day, seven days a week, fifty-two weeks a year—for years.

The medications used to treat opioid abuse are powerful agents that interrupt this cycle. Although medications alone rarely cure an addiction, they are critically important to breaking the cycle of preoccupation with opioid use and enabling addicts to benefit from comprehensive drug-abuse treatment.

(SEE ALSO: *Coerced Treatment for Substance Offenders; Ibogaine; Opioid Dependence; Opioid Complications and Withdrawal; Pregnancy and Drug Dependence; Substance Abuse and AIDS; Treatment Types*)

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Marijuana, An Overview Although marijuana is the most widely used illicit drug in the U.S., fairly little is known about how to effectively treat individuals who become dependent on this drug. Increasingly, however, the findings of controlled trials designed to evaluate the effectiveness of alternative counseling approaches are appearing in the literature. Additionally, recently acquired knowledge about the actions of a marijuana-like compound that occurs naturally in the brain will

enhance our understanding of the nature of marijuana dependence and possibly set the stage for the development of pharmacological interventions.

Prevalence of Marijuana Dependence. The most widely used illicit substance in the U.S., it is estimated that seventy-two million people have ever used the drug and eleven million are doing so currently (i.e., at least once in the past month). Nearly seven million reported using marijuana weekly or more often in 1998, and approximately two million individuals begin use of marijuana each year (SAMHSA, 1999).

Epidemiological studies conducted in the last two decades permit an estimation of the prevalence of marijuana dependence in the United States. In the 1980s, the Epidemiological Catchment Area (ECA) study involved in-person interviews with 20,000 Americans in five urban areas (Anthony & Helzer, 1991). The study's purpose was to determine the prevalence of psychiatric symptoms for forty major psychiatric diagnoses including drug abuse and dependence. Based on the criteria for the marijuana dependence diagnosis utilized in that study (indications of tolerance or withdrawal plus pathological use or impaired social functioning lasting for at least one month), 4.4 percent of adults were found to have been dependent on marijuana at some point in their lives. About a decade later, interviews conducted with over 8,000 individuals for the National Comorbidity Study led to a very similar estimate that 4.2 percent of the general U.S. population meet the diagnostic criteria of marijuana dependence (Anthony, Warner, & Kessler, 1994).

For those who have used marijuana at least once, the relative probability of ever becoming dependent on the substance is estimated at 9 percent (Anthony, Warner, & Kessler, 1994). This risk level appears modest when compared with risk estimates of dependence for those who've used other substances at least once (tobacco-32%; alcohol-15%; cocaine-17%; heroin-23%). However, among individuals who have smoked marijuana more frequently, the risk of developing dependence is higher. Among those who've used it five or more times, the risk of dependence is 17 percent (Hall, Johnston, & Donnelly, 1999). For daily or near daily users, the risk may be as high as one in three (Kandel & Davies, 1992).

Treatment Approaches with Marijuana-Dependent Adults. A series of controlled trials conducted since the mid-1980s have focused on evaluating interventions for marijuana-dependent adults. Stephens and Roffman (1994), in a 1986–1989 study funded by the National Institute on Drug Abuse, compared the effectiveness of a 10-session cognitive-behavioral group intervention with a 10-session social support group discussion condition. The cognitive-behavioral treatment focused on strengthening the participant's skills in effectively coping with relapse vulnerabilities. The social support treatment emphasized the use of group support for change. The participants were 212 marijuana smokers who averaged over ten years of near daily marijuana use. Following the completion of treatment and for the next 2.5 years in which participants were periodically reassessed, there were no significant differences between conditions in terms of outcomes (abstinence rates, days of marijuana use, problems related to use). During the final two weeks of counseling, 63 percent of the total sample reported being abstinent. While only 14 percent were continuously abstinent after one year, 36 percent had achieved improvement (i.e., either abstinence or reduction to 50 percent or less of the baseline use level and no reported marijuana-related problems) at that point. At 30 months post-treatment, 28 percent reported abstinence for the past 90 days. Thus, both counseling approaches were modestly effective in helping a significant portion of participants either achieve abstinence or improvement. These findings called into question the hypothesized superiority of a cognitive-behavioral approach with marijuana-dependent adults and argued for additional research on treatment approaches.

In a second NIDA-funded study conducted by Stephens and Roffman (1989–1994) with 291 adult daily marijuana smokers, a three-group design permitted the comparison of two active treatments with a delayed treatment control condition (Stephens, Roffman, & Curtin, in press). One of the active treatments involved 14 cognitive-behavioral skills training group sessions over a four-month period, emphasizing both the enhancement of coping capacities in dealing with situations presenting high risk of relapse and the provision of additional time for the building of group cohesion and mutual support. The second active treatment involved two individual motivational enhancement counseling sessions delivered over a one month period. The latter approach appeared promising inas-

much as a growing literature in the addiction treatment field was supporting the effectiveness of short-term interventions (Bien, Miller, & Tonigan, 1993), utilizing motivational interviewing strategies (Miller & Rollnick, 1991), designed to strengthen the individual's readiness to change (e.g., providing participants normative comparison data concerning their marijuana use patterns). The first session in this condition involved the counselor reviewing with the participant a written Personal Feedback Report generated from data collected during the study's baseline assessments. The counselor used this review as an opportunity to seek elaboration from the participant when expressions of motivation were elicited, to reinforce and strengthen efficacy for change, and to offer support in goal-setting and selecting strategies for behavior change. One month later, the second session afforded the opportunity to review efforts and coping skills utilized in the interim period. In both conditions, participants had the option of involving a supporter. Following treatment, there was no evidence of significant differences between the two active treatments in terms of abstinence rates, days of marijuana use, severity of problems, or number of dependence symptoms. At the 16-month assessment, 29 percent of group counseling participants and 28 percent of individual counseling participants reported having been abstinent for the past 90 days. Both active treatments produced substantial reductions in marijuana use relative to the delayed treatment control condition. The results of this study suggest that minimal interventions may be more cost-effective than extended group counseling efforts for this population.

The third study, funded by the Center for Substance Abuse Treatment (1996–2000) and conducted in three sites, also employed a three-group design with a delayed treatment control condition (Donaldson, 1998). One of the active treatments involved nine individual counseling sessions delivered over a 12-week period, with the initial sessions focusing on motivational enhancement and the later content emphasizing cognitive-behavioral skills training and, as needed, case management. The other active treatment involved two individual motivational enhancement therapy sessions delivered over a one-month period. (This condition replicated the brief intervention in the above-reported study conducted by Stephens and Roffman). At the 9-month follow-up, both active treatments produced outcomes superior to the 4-month delayed treatment control condition. Further, the 9-session intervention produced significantly greater reductions in marijuana use and associated negative consequences compared to the 2-session intervention. Abstinence rates at the 4- and 9-month follow-ups for the 9-session intervention were 23 percent and 13 percent, respectively. These differences between the two active treatments were apparent as early as 4 weeks into the treatment period and were sustained throughout the first nine months of follow-up. As was the case in the two studies discussed above, the findings of the CSAT-funded research point to modest efficacy of counseling interventions with marijuana-dependent adults. More positive outcomes from the 2-session motivational enhancement intervention were found in the Stephens and Roffman (in press) study than in the CSAT-funded investigation.

In a study funded by NIDA, Budney and colleagues randomly assigned sixty marijuana-dependent adults to one of three 14-week treatments: motivational enhancement, motivational enhancement plus coping skills training, or motivational enhancement plus coping skills training plus voucher-based incentives (Budney, Higgins, Radonovich, et al., in press). In the latter condition, participants who were drug abstinent—documented with twice-weekly urinalysis screening—received vouchers that were exchangeable for retail items (e.g., movie passes, sporting equipment, educational classes, etc.). The value of each voucher increased with consecutively negative specimens. Conversely, the occurrence of a cannabinoid-positive urine specimen or failure to submit a sample led to a reduction of each voucher's value to its initial level. Participants in the voucher-based incentive condition were more likely to achieve periods of documented continuous abstinence from marijuana during treatment than were participants in the other two conditions. Additionally, a greater percentage of participants in the voucher-based condition (35%) were abstinent at the end of treatment than was the case in the skills training (10%) or motivational enhancement (5%) conditions. The absence of long-term post-treatment assessment data limits comparisons of this study's outcomes with those from the other trials discussed above. However, based on their earlier research with voucher-based incentives in treating cocaine-dependency, the authors are hopeful that

future studies will demonstrate successful long-term outcomes in marijuana-dependent participants who achieve and maintain abstinence during treatment.

In reviewing the above work, it appears that some participants who sought treatment have been substantially aided in either quitting or cutting back. However, it is also apparent that the majority of those treated in the these studies reported above did not achieve their initial goal of durably abstaining from marijuana. Given the evidence of the drug's dependence potential and adverse health consequences (Hall, Johnston, & Donnelly, 1999), continuing development and testing of marijuana dependence interventions is clearly warranted.

Support Groups. Marijuana Anonymous groups, a self-help fellowship based on the principles and traditions of Alcoholics Anonymous, exist in a number of states and internationally. In addition to in-person meetings, MA sessions are also held on-line. The organization's web site address is: www.marijuana-anonymous.org, and its toll-free telephone number is 800-766-7669.

User Characteristics Predictive of Treatment Success. Stephens, Wertz, and Roffman (1993) reported predictors of successful outcomes in their first marijuana treatment trial. Higher levels of pretreatment marijuana use predicted higher use levels following treatment. Indicators of lower socioeconomic status predicted more reports of problems associated with marijuana use post-treatment. Finally, individuals who prior to treatment indicated greater self-efficacy for avoiding use had more successful post-treatment outcomes.

Reaching the Non-Treatment-Seeking Heavy Marijuana Smoker. With funding from NIDA (1997 through 2000), Stephens and Roffman are conducting a clinical trial (''The Marijuana Check-Up'') with 188 non-treatment-seeking adult marijuana smokers who have been randomly assigned to a motivational enhancement intervention (The Personal Feedback Session), a marijuana educational intervention (The Multimedia Feedback Session), or a brief waiting period. This study is adapted from a brief intervention (''The Drinker's Check-Up'') in the alcoholism field (Miller & Sovereign, 1989).

In conducting The Marijuana Check-Up, a variety of recruitment strategies were used to attract participants, including posters, radio and newspaper ads, and outreach at various community events (Stephens, et al., 1998). Project publicity targeted adults over the age of 18 who used marijuana and had concerns or were interested in obtaining information. These strategies highlighted the objective, non-judgmental, and confidential approach of the study. All announcements emphasized that the MCU was not a treatment program. Those who inquired were told that although this program did not offer counseling for persons who wanted to quit or reduce their use, it would likely be useful in helping an individual better assess their experiences with marijuana.

The first MCU session involved a structured interview that included an assessment of the individual's use patterns, perceived benefits and adverse consequences associated with both continued use and reductions or cessation of use, and self-efficacy in accomplishing cessation. In the second session, feedback to the client from the initial assessment was largely normative and risk-related in nature. Utilizing motivational interviewing skills, the therapist elicited the client's views concerning benefits and costs associated with both his or her current marijuana use pattern, as well as various pathways of change. When appropriate, the discussion turned to goal-setting for reduction or cessation of use and the identification of useful behavior change strategies.

Based on the finding that 64 percent of participants met diagnostic criteria for cannabis dependence and, of those who did not, 89.4 percent met criteria for cannabis abuse (American Psychiatric Association, 1994), it was evident that the check-up modality offered a useful method for reaching the non-treatment-seeking heavy marijuana user. Upon joining the study, fewer than a third had resolved to quit or cut back on their use. They were using marijuana on more than 80 percent of the days prior to the interventions and typically getting high two or more times per day.

The check-up modality may also show promise in affecting behavior change. While the study is still ongoing, preliminary analyses of outcomes indicated that participants in the motivational enhancement condition (the personal feedback session) were more likely to both reduce the amount of marijuana smoked per day and the number of days of use than were those in the educational or wait-list control conditions.

Marijuana Withdrawal. A mild syndrome of withdrawal from marijuana has been reported,

with symptoms that may include: restlessness, irritability, mild agitation, insomnia, decreased appetite, sleep EEG disturbance, anxiety, stomach pain, nausea, runny nose, sweating, and cramping (Budney, Novy, & Hughes, 1999; Crowley, Macdonald, Whitmore, et al., 1998; Haney, Ward, Comer, et al., 1999; Jones, Benowitz, & Bachman, 1976). Commonly, these symptoms lessen within a week to 10 days.

The Future of Marijuana Interventions. Currently underway or recently completed controlled trials testing various models of marijuana dependence treatment with adults and adolescents will undoubtedly contribute new information to what is currently known. The ''leading edge'' of such studies include counseling interventions in which contingency management components, variations in motivational enhancement strategies, brief and extended cognitive-behavioral therapies, treatments involving family members, and alternative dosages and distributions of counseling episodes are being evaluated.

The treatment of marijuana dependence may also ultimately be informed by knowledge of human biology. As an example, there is some evidence for the role of genetics in determining whether the marijuana user will become dependent. In a study of more than 8,000 male twins, genes were shown to influence whether a person finds the effects of marijuana use pleasant (Lyons, Toomey, Meyer, et al., 1997). Comparable findings were demonstrated for females (Kendler & Prescott, 1998). While factors in an individual's social environment clearly influence whether he or she ever tries marijuana, becoming a heavy user or abuser may be more determined by genetically transmitted individual differences, perhaps involving the brain's reward system. Research in this area may eventually identify individual risk factors for marijuana dependence that people can use in making decisions about their own use of this drug.

Finally, considerable evidence for a biological basis to marijuana dependence has accumulated since the identification of a specific cannabinoid receptor in the brain (Devane, Dysarz, Johnson, et al., 1988) and the discovery of anandamide, a compound that binds to and activates the same receptor sites in the brain as delta-9-tetrahydrocannabinol (THC), the active ingredient in marijuana. (Devane, Hanus, Breuer, et al., 1992). Subsequently, researchers discovered a cannabinoid antagonist, a compound that blocks anandamide action in the brain (Rinaldi-Carmona, Barth, Heaulme, et al., 1994). Taken together, these discoveries have made it possible to systematically study the effects of chronic exposure to marijuana. With greater understanding of the cannabinoid neurochemical system's physiology, the potential for developing and testing pharmacological interventions for marijuana dependence is advanced.

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Polydrug Abuse, An Overview Polydrug abuse (also called multiple-drug abuse) refers to the recurring use of three or more categories of PSYCHOACTIVE substances. It is a pattern of substance abuse that is most commonly associated with illegal drug use and youth. Most polydrug users also smoke TOBACCO, but NICOTINE has only recently begun to be recognized as a drug of abuse to be addressed with polydrug users.

While the term *Polydrug User* is usually reserved for people with a rather varied and nonspecific pattern of drug use, many drug users who have a preferred (a primary) drug of abuse are also polydrug users. In fact, it is uncommon for users of any illicit drug to restrict their substance use to only the one drug. For example, an individual may be a regular COCAINE user but also use ALCOHOL, TRANQUILIZERS, and MARIJUANA.

WITHDRAWAL

The intensity of withdrawal symptoms and their medical risk depends on the particular substances used and the degree to which dependence has developed. Withdrawal is most often clinically significant in those who have developed severe dependence on a primary drug of abuse; the medical risks of such withdrawal vary substantially with the type of drug. For example, much greater risks exist for BARBITURATE than for HEROIN withdrawal. The re-

cent use of other drugs in addition to the primary drug of abuse complicates the withdrawal process. In such cases, careful medical assessment is important in the planning of withdrawal management for polydrug users.

Polydrug users who typically dabble among the available drugs without developing severe dependence on any of them usually have no clinically serious problems when they stop using drugs. They may experience some discomfort, agitation, or sleeplessness but they do not normally require medical treatment. Social stability and support would be important, however, as the risk of relapse could be high during this period of discomfort.

ASSESSMENT

There are two main purposes of assessment: (1) to determine what specific treatment would be most suited to the specific needs of the polydrug user; and (2) to determine baseline levels of functioning against which progress in treatment can be measured. Assessment must address many areas of functioning in addition to drug use. These include the following: medical and psychiatric problems; family and other social relationships; school or work problems; leisure activities and skills; criminal activities and legal problems; and financial status.

Drug use must be carefully assessed in the polydrug user, because of the variety of drugs used and the need to evaluate the risks associated with the particular pattern of use. The usual procedure is to divide drug use into categories based on pharmacological similarities. These categories typically include: alcohol; marijuana; HALLUCINOGENS (e.g., LSD); heroin; other OPIOIDS (e.g., CODEINE); cocaine; other STIMULANTS (e.g., AMPHETAMINES); TRANQUILIZERS (e.g., BENZODIAZEPINES such as Valium) and other sedative hypnotics (e.g., barbiturates); and solvents (including glue). Because accurate estimates of doses are very difficult to obtain from polydrug users, their drug use is usually assessed as the number of times each drug has been used within a specified time period. Other important factors to consider in assessing drug use are risks related to HUMAN IMMUNODEFICIENCY VIRUS (HIV) infection—especially injection drug use, and drugs used in combination.

A further consideration in assessment is the client's commitment to change. Polydrug users may be, at best, ambivalent about the need for change. The assessment process offers an excellent opportunity to enhance the polydrug user's motivation for change by providing feedback and support, as well as by helping the person to clarify goals and values.

TREATMENT APPROACHES

Many different treatment approaches are available, but they reflect differing conceptual or theoretical perspectives on the origins of drug-use problems as well as on the best ways to treat them. Most of these approaches were not developed for the polydrug user but, instead, were adapted from other substance-abuse treatments. The approaches described may be presumed to be quite widely available except where restrictions are noted. Research evidence concerning their comparative effectiveness for polydrug users is extremely limited.

Approaches Based on the Disease Concept. According to one variant of the disease concept, alcoholism and drug addiction are incurable diseases. Those affected are considered unable to control their use of the substance, because of an allergiclike, biological reaction. This approach has only one solution to the problem—to get the user to abstain from any use of the drug.

Twelve-Step Groups. The treatment approaches most commonly associated with the disease concept are those based on ALCOHOLICS ANONYMOUS (AA), which was started in 1935. The TWELVE-STEP approach developed by AA has been adapted for application to other primary drugs of abuse, e.g., NARCOTICS ANONYMOUS (NA) and COCAINE ANONYMOUS (CA). Like AA, these approaches rely exclusively on self-help peer-group procedures. Members voluntarily embark on a lifetime journey of recovery, armed with a set of principles and the support of peers who share a common problem and a desire for change. The central features of these approaches are the following: an acceptance of being powerless over the drugs; a belief in a higher power; a commitment to make restitution to those who have been harmed; and personal responsibility to maintain abstinence. Polydrug users may affiliate with any of such groups, depending on the particular drugs most commonly used. They may, however, have some difficulty in identifying with the majority of group members as peers. Often a buddy or two with the same problems and concerns become a special subgroup.

Chemical-Dependency Programs. Some treatment programs, most notably residential programs, have adapted the twelve-step approach as the basis of their treatment. Chemical-dependency (CD) programs are the most prominent example. These programs are an extension of the four week MINNESOTA MODEL (for ALCOHOLISM) to a broader range of substances of abuse. Some have a particular focus for young polydrug users.

The CD approach usually involves a three- to six-week structured and intensive residential-treatment phase, which includes lectures and discussions about the harmful effects of drug use; group-therapy sessions that focus on breaking down denial and personal issues related to drug use; an orientation to the twelve-step approach; recreational and physical activity; and family counseling sessions. The residential phase is followed by an extended aftercare program, typically involving attendance at AA, NA, or CA meetings. Many CD programs specialize in the treatment of polydrug users who also have coexisting psychiatric problems.

The number of CD programs has grown rapidly in the past decade, particularly in private hospitals. Because of their residential phase, these CD programs are among the most expensive form of treatment available to polydrug users.

Systems Theory–Based Approaches. Systems theory holds that individuals function within a variety of social systems (e.g., the family and peer groups) and that these systems act to influence behavior and to resist changes that are not in the interest of the broader system. From this perspective, drug use may be seen as serving some useful purpose within the "identified client's" social systems. Attempts to change that drug-use behavior without ensuring that the system will support and maintain such a change may be doomed to failure.

Family Therapy. Family therapy is the most common application of systems theory to the treatment of polydrug users. This is because research has linked various forms of family dysfunction to the development of drug-use problems. Also, many polydrug users are children and young adolescents and their drug use is a major family issue.

In family therapy, the family rather than the polydrug user becomes the client. Treatment addresses family-system issues, which include family roles, patterns of communication, and structural factors such as the alliances that may exist within and among parts of the family system. The presenting problem of drug abuse may be dealt with directly within the framework of the family approach. It may otherwise be treated as a symptom of the family's dysfunction—where the expectation is that the drug use will disappear with resolution of the more fundamental family problems.

In family therapy, all or most of the family members typically attend the treatment sessions. One-person family therapy is a variation on this practice, in which the treatment focuses on changes to the family system via one member of that system. This practice is, however, very limited in comparison with the more common approach of involving most or all the other family members.

Peer-Network Therapy. Peer-network therapy focuses on the peer or friendship social system. Polydrug users are typically young and their drug use is often a social activity. Much research evidence links all drug use to peer associations. This may be caused by peer influence or because drug users seek out other drug users. Either way, it is widely believed that changes in peer associations are a necessary step for polydrug users who would attempt to discontinue drug use.

Peer-network therapy involves systematically examining the relationship of drug use to association with particular peers. Strategies involve avoiding certain peers; strengthening peer relationships in which drug use is not a factor; reestablishing old relationships that may have been ignored while drug use was occurring; using a buddy system to facilitate developing new peer relationships; and structuring leisure activities to help the client meet new friends who share similar attitudes and goals concerning drug use. Typically, changes in the peer system are introduced via the identified client, but peer-network therapy may also involve sessions that include other members of the peer network.

Peer-network therapy is still a relatively novel approach to the treatment of polydrug users, although many treatment programs are placing increased emphasis on changes to peer networks as part of their overall treatment strategy.

Peer Counseling. Polydrug use is the most common pattern of substance abuse for many novice drug users. For such individuals, early intervention programs based on peer counseling, and provided in school or neighborhood settings, may be appropriate. Peer counseling capitalizes on the tendency for adolescents to be most influenced by their peers. Peer counselors are selected on the basis of their

ability to act as good role models. They are trained to emphasize practical strategies to assist polydrug users to change their lifestyles in ways that support becoming drug free. They also act as facilitators or group leaders in peer counseling groups, in which adolescents learn from each other.

Social Learning Theory–Based Approaches. Social learning theory suggests that drug use is a learned behavior and that it may be changed by the therapeutic application of principles of learning theory. Treatments based on social learning theory usually begin with a functional analysis of the drug use. This involves a detailed analysis of the circumstances in which drug use occurs and the apparent benefits to the user. The basic assumption is that drug use serves useful purposes (functions) in the life of the user and that understanding these functions of drug use is a critical step in planning treatment.

Coping Skills Training. One such treatment approach is based on substituting alternative methods of obtaining the same benefits that drug use provides. If the individual becomes more sociable and outgoing on drugs, social-skill training is provided; if drug use reduces tension, stress-management techniques are offered. This approach is sometimes referred to as coping-skills training, because improved coping in one or more life areas usually becomes the primary treatment goal. Coping-skills training can address a variety of skill deficits from improved problem solving, to coping with depression, to increased assertiveness. The objective is to provide the polydrug user with alternative methods of coping with difficult life situations.

Since the 1970s, this type of approach has become the primary alternative to more traditional approaches based on the disease concept or psychotherapy.

Contingency Management. Contingency management involves structuring unpleasant consequences to occur when drugs are used. The assumption is that these adverse consequences will compete with the benefits the user gets from the drug use, thereby reducing the likelihood that drug use will continue. Contingency management procedures are most effective when the occurrence of the drug use behavior can be reliably determined and the prescribed consequences reliably administered. Urine screening is the most common means of monitoring whether any drug use has occurred. Clients are typically required to provide urine specimens according to a random schedule that minimizes the opportunity to plan drug use to escape detection. A variety of types of consequences can be used. For example, clients may avoid the loss of a job, regain custody of children, or avoid breach of probation by consistently providing "clean" urines. While many treatment programs emphasize the consequences of drug use, few do so in the very systematic way required by contingency management.

Cue Exposure. Cue-exposure techniques focus on the circumstances that precede or "cue" drug use. Frequent repetition of patterns of drug taking may result in certain cues becoming conditioned so that the user experiences cravings for the drug in the presence of these cues. For example, observing drug-use paraphernalia or being in a setting in which drugs have frequently been used in the past, may cause the polydrug user to experience cravings. These cues can be the cause of relapse. Treatment involves repeatedly exposing the individual to these cues in a controlled manner (e.g., with a supportive person present) until the cue no longer elicits the craving response. Conditioning is more apt to occur for a specific drug than across a variety of drugs. Hence cue exposure may be most relevant for polydrug users with a pronounced primary drug of abuse.

Approaches Aimed at Major Psychological Change. These approaches assume that the cause of drug use lies in the psychological makeup of the polydrug user. From this perspective, drug use is a self-destructive or deviant act brought about by serious underlying psychological problems or the adoption of anti-social values. Treatment is aimed at correcting the underlying problem for which drug use is thought to be merely a symptom.

Psychotherapy. Psychotherapy is an intensive and extended counseling approach in which the therapist explores the past events in the client's life with the aim of uncovering emotionally upsetting events or identifying themes or patterns of behavior that interfere with the effective social and psychological functioning of the individual. The drug use itself would seldom be the focus of the treatment sessions. Rather, the goal of psychotherapy would be psychological growth to change the personality of the polydrug user.

Psychotherapy can be provided on a one-to-one or group basis. It is typically provided on an outpatient basis but has also been provided within the framework of long-term residential programs for

young drug users. Psychotherapy can be a comparatively expensive form of treatment, because it requires highly skilled therapists and typically takes longer to complete than other therapies. It may be most relevant when the polydrug user also has a psychiatric problem (e.g., depression).

Therapeutic Communities. THERAPEUTIC COMMUNITIES (TC's) are long-term residential programs of twelve to twenty-four months duration. There are several types of TC, all of which share a common belief that clients gain from living together in a therapeutic environment for an extended period of time. The most prominent TC model is based on the Synanon program developed for heroin addicts in the late 1950s. Since that time, many variations of this model have evolved and the target treatment population has been broadened to include polydrug users.

The treatment approach is typically targeted to hard-core drug users who are judged to have serious personality deficits or chronic antisocial values. The problem is presumed to be the person, not the drug or the individual's social environment. The treatment is extremely intensive, often involving harsh confrontation and emotionally charged encounters. The intent is to break through the protective shell that the polydrug user has developed—in response to past deprivations and abuse—and to resocialize the individual to adopt new values and patterns of behavior. Consistent with its self-help origins, treatment within the TC is usually provided by recovered addicts.

Psychobiological Approaches. Psychobiological approaches involve interventions which have a biological (often neurological) mechanism of action. Examples include treatments that involve the administration of a drug (pharmacotherapies) and ACUPUNCTURE, although the latter has had little application to the treatment of polydrug users. These approaches are based on the assumption that it is possible to change drug-use behavior by biological methods even though the drug-use problem may not have biological origins. For example, a drug may be used in treatment to eliminate the positive effects of an abused drug, thereby reducing the likelihood that its use will continue.

Pharmacotherapies. Drugs are used in the treatment of substance-abuse problems for a variety of purposes. These include substituting for the drug effect; blocking or changing the drug effect; or treating a condition that is believed to underlie, or at least contribute to, the substance-abuse problem. Most pharmacotherapy approaches are intended to address the misuse of specific substances, which limit their application to polydrug users; however, many polydrug users have preferred drugs of abuse for which a pharmacotherapy approach may be appropriate. In such instances, it will usually be necessary to combine the pharmacotherapy treatment with some other approach to ensure that treatment addresses all the individual's drugs of abuse.

Methadone treatment is the best-known of the drug-substitution approaches. Methadone substitutes for heroin (and other opioid drugs) prevent the onset of withdrawal symptoms in addicts. This serves to stabilize the user with regard to the desire or need to continue heroin use until the addict develops sufficient confidence and a strong enough support system to become drug free.

Other drugs used in treatment (e.g., NALTREXONE) act on the brain to block or reduce the pleasant sensations associated with the use of particular drugs. The assumption is that if the so-called beneficial effects of the drug are eliminated or reduced, it is less likely to be used. So-called anti-alcohol drugs (ANTABUSE and Temposil) take this notion one step further, by altering the metabolism of alcohol so that its effects become very unpleasant (the individual gets sick if alcohol is consumed while the drug is in effect). For all these approaches, strategies to ensure that the individual actually takes the prescribed drug are very important since the polydrug user can easily obtain the desired drug effects just by not taking the treatment drug.

Finally, some polydrug use reflects an attempt at self-medication to cope with symptoms of untreated psychiatric problems. The appropriate diagnosis and treatment (with medication) of such problems may reduce the client's need to self-medicate. Examples of this form of pharmacotherapy include medications for the treatment of anxiety, mood disorder, and psychotic disorders.

THE IMPORTANCE OF MATCHING TREATMENT TO CLIENT NEEDS

This chapter has described a broad range of treatment approaches available to the polydrug user. In practice, treatment programs often combine elements of the various approaches described.

None of the approaches can claim general superiority over any other. Any one of them may be the most appropriate treatment choice for a particular individual under certain circumstances. It is important to assess the needs and wishes of the polydrug user carefully before selecting the treatment that seems most likely to be most helpful.

(SEE ALSO: *Addiction: Concepts and Definitions; Adolescents and Drug Use; Causes of Substance Abuse; Comorbidity and Vulnerability; Contingency Contracts; Disease Concept of Alcoholism and Drug Abuse; Methadone Maintenance Programs; Prevention; Treatment Types*)

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GARTH MARTIN

Polydrug Abuse, Pharmacotherapy

Although many individuals present with abuse or dependence upon a single PSYCHOACTIVE SUBSTANCE, increasing numbers of drug users are presenting with dependencies upon two or more such substances. The DIAGNOSTIC AND STATISTICAL MANUAL of the American Psychiatric Association (DSM-IV) and the INTERNATIONAL CLASSIFICATION OF DISEASES of the World Health Organization (ICD-10) define a condition called "polydrug dependence" or "multiple drug dependence," in which there is dependence on three or more psychoactive substances at one time. Polydrug dependence is particularly common among adolescents and young adults. However, if one includes NICOTINE and CAFFEINE dependence, over half of patients with psychoactive-substance dependence are polydrug-dependent.

The use of specific, preferred combinations of drugs is typically seen in polydrug users. OPIOIDS and COCAINE are often used together, as are ALCOHOL and cocaine or nicotine and alcohol. Alcohol, BENZODIAZEPINES, and cocaine are often used together by opiate users, especially METHADONE users. Illicit-drug users often show nicotine and caffeine dependence. Some individuals will use whatever psychoactive substances are available. One useful distinction is the difference between simultaneous and concurrent polydrug use. In simultaneous polydrug use, the drugs are used together at the same time for a combined effect, such as heroin and cocaine mixed and injected as a "speedball." In concurrent polydrug use, the various drugs are used regularly but not necessarily together. An example is a heroin user who uses benzodiazepines and alcohol to get another kind of high. In other cases, the polydrug abuser may self-medicate with one drug to offset the side effects of another. Cocaine abusers often take diazepam (Valium) to relieve the irritability that follows cocaine binges. Heroin addicts sometimes take benzodiazepines to relieve the anxiety that characterizes the early stages of opioid withdrawal. A more recent development is the abuse of antidepressant medications among heroin users. The tricyclics appear to be abused more frequently than either the SSRIs or the MAO inhibitors.

TREATMENT

The treatment of the polydrug user presents a particular challenge to the clinician. The simultaneous and concurrent use of multiple drugs may increase the level of dependence, increase drug toxicities, worsen medical and psychiatric

comorbidities due to the drugs, and intensify withdrawal signs and symptoms upon cessation of drug use. The basic principles of treatment of polydrug use are similar to those for the treatment of any single psychoactive-substance dependence. Patients require a complete medical and psychiatric assessment, treatment of active problems, detoxification, then rehabilitation with attempts to reduce subsequent use of the drugs. One of the complications of treating polydrug users is that the patient's history may be unreliable— many cannot remember what they have used and others do not know the identity of drugs they have purchased on the street.

In providing treatment for the polysubstance user, there are two options: (1) sequential treatment for the dependencies, with initial treatment of the major dependency or the dependency with greater morbidity; or (2) simultaneous treatment of all dependencies. Unfortunately, few objective data exist as to which type of treatment is optimal for which patients. Most clinicians rely on their own experience, the capabilities of the treatment setting, and the wishes of the patient. One rule of thumb that has been suggested for complex detoxifications is to focus initially on the CNS depressant drug(s) and not be overly concerned with the opioid component. The patient can be stabilized with regard to the opioid with methadone, and given phenobarbital to prevent the potentially life-threatening symptoms of sedative withdrawal.

The treatment of polysubstance dependence often involves more than one type of treatment modality. A common example is an alcohol-dependent, opioid-dependent, cigarette smoker who is receiving METHADONE MAINTENANCE for opioid dependence, abstinence-oriented treatment for alcoholism, and no specific treatment for nicotine dependence. The different treatment philosophies—methadone substitution, abstinence, and no treatment—necessarily conflict. In such cases, good communication and flexibility among the various treatment providers and with the patient are important to ensure optimal, coordinated treatment.

DETOXIFICATION

During the initial treatment of polysubstance abuse and dependence, the primary goals include cessation of substance use and the establishment of a substance-free state. If necessary, detoxification occurs, as well as management of medical and psychiatric problems. Detoxification is the removal of the drug in a fashion that minimizes signs and symptoms of withdrawal. It can be pharmacological or drug free. Pharmacological methods for detoxification include (1) a slow decrease in the dose of the drug or of a cross-tolerant agent (e.g., methadone for heroin withdrawal, diazepam for alcohol withdrawal, NICOTINE GUM for smoking cessation) and (2) stopping the drug and using an alternative agent to suppress signs and symptoms of withdrawal (e.g., CLONIDINE for opioid withdrawal, atenolol for alcohol withdrawal). For many drugs, pharmacologically assisted detoxification is not necessary. Simple alcohol withdrawal can be treated with supportive care. However, the presence of polysubstance dependence usually increases the need for pharmacological agents to assist in withdrawal.

There are few controlled studies on the clinical course and optimal therapies for detoxification from multiple psychoactive substances. Patients can be detoxified from all psychoactive substances together, or maintained on one or more drugs while being detoxified from others. When the drugs used are all part of the same class (e.g., alcohol and sedatives; methadone, CODEINE, and heroin), a complete detoxification is more common. When the drugs used are from different classes, partial or sequential detoxification usually occurs. An example of the latter situation is an opioid, cocaine, alcohol, and nicotine user who is detoxified from alcohol and cocaine, but maintained on methadone and allowed to continue tobacco use. Sometimes a partial detoxification is indicated because of the need for continued psychotropic medication for medical or psychiatric illnesses, such as continued opioids for chronic pain or benzodiazepines for anxiety.

Given the cross-tolerance of most SEDATIVE-HYPNOTICS with ethanol, methods that are effective for the detoxification from alcohol or sedatives alone are usually effective for the combinations of alcohol and sedatives. Loading techniques, with long-acting benzodiazepines, such as diazepam or CHLORDIAZEPOXIDE, or with BARBITURATES, such as PHENOBARBITAL, are well documented as effective. The advantages of these methods include matching the medication used for withdrawal to the individual patient's tolerance and the avoidance of overmedication. The anticonvulsant car-

bamazepine (Tegretol) has been shown to be effective for the treatment of combined alcohol and sedative withdrawal.

Although the mechanisms of action of various drugs differ, there are common neurological substrates of certain behavioral effects and of withdrawal signs and symptoms. The autonomic hyperactivity and some of the CNS excitation common to several withdrawal syndromes are mediated by the locus ceruleus of the brain. Medications such as alpha-2 antagonists (clonidine) and benzodiazepines, which inhibit locus ceruleus activity, have been shown to attenuate the symptoms of nicotine withdrawal. However, clonidine will not block the seizures that result from alcohol or sedative withdrawal.

LONG-TERM TREATMENT

In the long-term phase of treatment, the patient undergoes rehabilitation and reestablishment of a lifestyle free of drug dependency. Pharmacological treatment is sometimes used to assist rehabilitation. Pharmacotherapies may reduce drug craving, decrease protracted withdrawal symptoms, or decrease positive reinforcing effects of the drugs. Types of pharmacological therapies used in long-term treatment and rehabilitation include (1) maintenance (e.g., methadone maintenance for the treatment of opiate dependence); (2) blockade (e.g., NALTREXONE treatment for opioid dependence); (3) aversive therapy (e.g., DISULFIRAM for alcoholism, possibly naltrexone for alcoholism); and (4) psychotropic drug treatment of coexisting psychiatric disorders, such as lithium for bipolar alcoholics, or methylphenidate for cocaine-dependent patients with ATTENTION DEFICIT DISORDER.

The use of pharmacological agents as adjuncts in the treatment of polysubstance dependence is an area of active investigation. One medication that may prove useful in the treatment of combined cocaine and opioid dependence is buprenorphine (Buprenex). This partial mu agonist, used as a surgical analgesic, has shown efficacy as a substitute in the long-term treatment of opioid dependence. Compared with methadone, buprenorphine may produce less dependence and fewer withdrawal symptoms upon cessation. Buprenorphine treatment also may reduce cocaine use in some individuals dependent on both opioids and cocaine. Animal studies of the effects of buprenorphine on "speedball" self-administration are consistent with the findings of clinical trials of buprenorphine in polydrug abusers. Other research suggests that buprenorphine is effective in patients dependent on both cocaine and heroin because it improves regional cerebral blood flow. Desipramine has been reported as being effective in reducing cocaine use in methadone patients. Disulfiram, which is efficacious in the treatment of alcoholism, may also reduce cocaine use in individuals using both alcohol and cocaine.

Newer pharmacological agents that are being investigated for possible use in long-term treatment of polydrug abuse include a medication mixture of flupenthixol, a dopamine antagonist, and quadazocine, an opioid antagonist. The mixture targets combined stimulant/opioid abuse. A combination of these two drugs appears to be more effective in treating combined abuse of heroin and cocaine than either antagonist alone. Another agent that may have therapeutic potential is gamma-hydroxybutyric acid, a compound that affects the brain's dopaminergic systems. It may also be a neurotransmitter. Gamma-hydroxybutyric acid, first used as an anesthetic, emerged as a drug of abuse around 1990. It is still used by bodybuilders, partygoers at "rave" dances, and polydrug abusers. As of 2000, preliminary evidence supports its use in the treatment of alcohol and opiate dependence.

(SEE ALSO: *Comorbidity and Vulnerability; Treatment-Treatment Types*)

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Tobacco, An Overview Ever since tobacco use became popular, some users have been trying to quit. Sometimes they sought treatment because the tobacco was too expensive, because companions complained about the tobacco use, because they did not like the smoke in the air, or, in the case of SMOKELESS TOBACCO (chewing tobacco or spitting snuff), because they did not like the tobacco juice on the floor. Sometimes treatment was sought out of concern for health problems.

Cigarette smoking is the most common form of tobacco use, and smoking is one of the nation's most critical public health problems. Tobacco use causes more than 430,000 deaths each year in the United States and is the leading preventable cause of death. Most adults in the United States have either smoked cigarettes or used some other tobacco product. In 1997, 71 percent of the population aged twelve or older had tried cigarettes at some time in their lives. This article focuses on the treatment of cigarette smoking but will include a brief discussion of the treatment of smokeless tobacco use, for which many of the same principles apply.

According to the Surgeon General's report on reduction of tobacco, existing types of smoking intervention can be used to reduce smoking. Researchers believe that widespread dissemination of the approaches and methods shown to be effective, especially in combination, would substantially reduce the number of young people who will become addicted to tobacco, increase the success rate of young people and adults trying to quit using tobacco, decrease the level of exposure of nonsmokers to environmental tobacco smoke, reduce the disparities related to tobacco use and its health effects among different population groups, and decrease the future health burden of tobacco-related disease and death in this country.

There are a number of different methods used in the treatment of nicotine addiction. Behavioral counseling and nicotine replacement therapy have proven the most effective forms of intervention for nicotine addiction, particularly when they are combined. Non-nicotine medications, such as antidepressants, anxiolytics, and nicotine antagonists, are among the medications also used in treatment, though their efficacy is still under investigation.

TRENDS IN SMOKING CESSATION

Although the prevalence of smoking among the American public decreased in the late 1900s, the current number of smokers is still substantial. In the late 1990s, about one-quarter of adult Americans, or about 48 million people, smoked. Most of these people wanted to quit but were unable to do so because they found it too difficult. According to some figures from the late 1990s, only an estimated 2.5 percent of all smokers successfully quit each year.

EFFECTS OF SMOKING CESSATION

There are a number of physiological effects that take place in the human body after cessation of smoking. About twenty minutes after cessation, the blood pressure and pulse rate return to normal, and the body temperature increases to normal. About 8 hours later, the carbon monoxide level in the blood drops to normal, and after 1 day, an individual's chance of a heart attack decreases. After two days, nerve endings start to regenerate, and the ability to smell and taste is improves. After two weeks, an individual's circulation improves and the functionality of the lungs increase by a maximum of 30 percent. After a year of smoking abstinence, the risk of coronary heart disease is reduced to half that of a smoker, and after five years of cessation, the risk of death by lung cancer is cut in half. After fifteen years, the risk of coronary heart disease is equal to that of a nonsmoker.

RESEARCH ON CESSATION OF TOBACCO USE

Although the scientific study of smoking treatments dates from the mid-1900s, "nonscientific" and "scientific" treatments often overlap. Until the 1980s, there were still many observers who doubted that tobacco use was based on an addiction to or dependence on nicotine. In the 1950s and 1960s, many experts believed that smoking was "just a bad habit." Experts at that time failed to appreciate that tobacco use was a form of drug use; instead, they saw smoking as the kind of habit that could be broken by taking certain behavioral steps. This attitude was the origin of the so-called behavioral techniques for stopping smoking.

In the early part of the twentieth century, self-help movements were very popular and were directed against alcohol and other drug problems. Such efforts at behavioral changes have a long history in society. Perhaps because they are so commonplace, people tend not to seek professional help for dealing with minor behavioral problems. As a result, it should not be surprising that over the years much of the "treatment" for cigarette smoking has been self-administered. However, researchers find that self-help treatments have not generally been proven effective for most people. In one study of 5,000 smokers, only 4.3 percent of individuals who had quit on their own remained abstinent for one year after they attempted to quit. Self-help treatments, combined with such intensive treatment as behavioral counseling, nicotine replacement, or the combination of the two, is likely to be more effective.

No single treatment stands out as being the single best way for all smokers. In general, however, researchers have found that nicotine replacement therapy combined with behavioral counseling has shown the best results in the treatment of nicotine addiction.

GROUP VERSUS INDIVIDUAL THERAPIES

Much of the instruction and support that is part of smoking treatment can be done individually—one-on-one—with clients or can be delivered to a group of clients. Group programs have been used to provide hypnotism, educational therapies, behavioral therapies, and combined therapies. There is no clear scientific evidence indicating which delivery system is best, but it is clear that group programs can be less expensive than individual programs and that some clients have strong personal preferences for how they wish to receive treatment: Some enjoy the group support and like to share their experiences in a group; others find such involvement with groups unpleasant or embar-

rassing. As for the efficacy of such therapies, researchers have found that the more time counselors spend with smokers in a treatment session, the higher the likelihood of cessation. Longer duration of treatment in weeks and the total number of treatment sessions is also associated with improved odds of smoking cessation.

PHYSICIAN-BASED TREATMENTS

Physicians interested in preventive medicine make special efforts to encourage and support smoking cessation in their patients. In 1964, only about 15 percent of current smokers reported that a physician had advised them to quit smoking. By 1987, about 50 percent of current smokers had received such advice. Sometimes just the advice of a physician to quit and the setting of a quitting date can lead to successful smoking cessation. Physicians can also be helpful by referring patients to smoking treatment programs. Specialists who deal with patients already suffering from a smoking-related disease can be in a good position to help those who are well motivated to quit, but cardiac or lung patients often fail to stop smoking. Being diagnosed with a smoking-related disease is no guarantee that the patient will quit smoking.

The Importance of "Minimal" Interventions. In medical settings, there has been research on the value of interventions (e.g., brief advice, pamphlets) that take only a few minutes of the physician's time. Although the effects of these interventions are usually small, they are generally viewed as worthwhile because they can reach so many smokers.

SMOKING CESSATION EFFORTS BY AGENCIES

Many diseases caused by smoking—cancer, heart disease, lung disease—have agencies concerned with furthering research, dissemination of public health information, and treatment of the disease. The Cancer Society, the Lung Association, and the Heart Foundation are voluntary, charitable organizations. Each has developed materials and programs to promote smoking cessation. The measured treatment effects of simple stop-smoking pamphlets are small, but since they can reach many smokers at very low cost, they should be viewed as beneficial elements of the public-health efforts to support smoking cessation. U.S. government agencies concerned with smoking and smoking-related disease have also developed and promoted materials and procedures to foster smoking cessation.

The voluntary agencies have supported smoking cessation efforts in the workplace, by providing smoking-treatment services and by promoting smoking bans in the workplace. EMPLOYEE ASSISTANCE PROGRAMS (EAPs) increasingly offer help to smokers who are trying to quit. In addition to workplaces, many public places, such as restaurants and other public buildings, now prohibit smoking on their premises. Just as social pressures encouraged many smokers to start the habit, social pressures might encourage them to stop. Once it was fashionable to be a cigarette smoker; now it is becoming fashionable to stop smoking.

NICOTINE-REPLACEMENT THERAPIES

Nicotine-replacement therapies can help reduce the nicotine withdrawal symptoms after smoking cessation. Replacement therapies help individuals deal with their smoking gradually by separating the behavioral and pharmacological components of smoking. While physical symptoms of nicotine withdrawal are reduced, the individual can focus on dealing with the behavioral challenges of stopping. The most commonly used nicotine-replacement therapies are a gum that releases nicotine as it is chewed and a patch that slowly releases nicotine into the body through the skin. These therapies are available over-the-counter. Transdermal nicotine patches appear to be preferred by individuals over nicotine gum. They seem to have the fewest side effects and are associated with the greatest long-term abstinence rates.

Nicotine nasal sprays and nicotine vapor inhalers that deliver nicotine through the respiratory system are less common forms of nicotine-replacement therapy. They became available in the United States in 1996 and 1998, respectively. There have been reports of eye, nose, and throat irritation with the nasal sprays, but individuals have been known to build a tolerance to these effects.

Nicotine-replacement therapy is considered an effective treatment for smoking cessation, although the efficacy of the different methods varies when used alone. In addition, a number of negative side

effects could potentially interfere with a patient's success with the therapy.

OTHER DRUG THERAPIES

For someone who has tried repeatedly and yet failed to stop smoking for good, a medicine that could take away the desire to smoke would be welcome. A number of non-nicotine medications have been developed to help aid smokers in the cessation process. Nicotine antagonists help cut down on nicotine withdrawal symptoms—including irritability and anxiety—or mimic the effects achieved by smoking and thus may help decrease an individual's desire for a cigarette. Such antagonists include antidepressants, anxiolytics, and stimulants or anorectics. Other medications make smoking distasteful to the user. Studies on the efficacy of such non-nicotine drug therapies continue.

HYPNOSIS

HYPNOSIS is worth special mention because of its popularity as a smoking therapy. Careful evaluations of hypnotherapies show small or no treatment effects. One of the problems in studying hypnotherapies is that the actual hypnotic procedures involved are not standardized. The kind of procedures used and suggestions made to the hypnotized patient (e.g., "You will not want a cigarette" vs. "The thought of a cigarette will make you feel sick") differ from therapist to therapist. It is important to deal with reputable therapists who charge reasonable fees for their services.

MULTIMODAL THERAPIES

A wide range of behavioral therapies have been tested, and no single method stands out as particularly effective. Multimodal approaches have become widely used, in hopes that something loaded into the shotgun will hit its mark. Currently, there is no reliable way to judge beforehand which smoker will be most helped by a particular technique (the exception being that heavier, more dependent smokers are consistently more likely to benefit from nicotine replacement). The multimodal, something-for-everyone approach is reasonable. There is not room in this article to discuss in detail the variety of behavioral therapies that have been used, but they have in common the use of basic psychological principles of learning.

Contingency contracting involves, for example, the preparation of detailed contracts that spell out punishments that will follow from the return to smoking (e.g., if the patient relapses, he or she will give $100 to someone he or she dislikes).

Aversive conditioning procedures (e.g., rapid smoking, satiation) cause cigarette smoking to be associated strongly with the acute unpleasant effects (such as dizziness and nausea) of smoking very heavily.

Relapse Prevention and the Maintenance of Abstinence. RELAPSE PREVENTION programs have been developed to reduce the problem of relapse or return to smoking. Many of the same behavioral techniques used in multimodal programs are applied to the task of helping prevent relapse and helping prevent the occasional slip back to smoking from becoming a permanent return.

Smoker's Anonymous Programs. Smokers have sometimes organized this type of program to support smoking cessation. The program allows smokers to support each other and teach each other techniques that will help them to stop smoking and to keep from returning to smoking. These programs have not generally become popular. This is in contrast to the great popularity of ALCOHOLICS ANONYMOUS (AA) groups.

RELATION TO TREATMENT OF OTHER DRUG PROBLEMS

Heavy smoking is strongly linked to heavy alcohol and other drug use. Smoking is often found in those with ALCOHOL and other drug problems. Those smokers who fail to stop smoking may have serious alcohol or other drug problems that require treatment before the smoking problem can be resolved.

ON SELECTING A WAY TO STOP SMOKING

Smokers should be advised to take a long view of their efforts to stop smoking, understanding that if one method does not help them, they should try another, and another, until they have stopped smoking. Any one attempt to stop smoking can meet with poor success. With repeated attempts, the smoker may encounter some success. Also, repeated attempts give the smoker experience with

assorted treatment techniques, so that the individual begins to learn for what helps and what does not help. Finally, there may be a kind of "no more nice guy" effect, so that the smoker gets fed up with failing to quit smoking.

It is also important to realize that no two programs are delivered in exactly the same way. The individual characteristics of a therapist and the client's rapport with that therapist can contribute to a therapy's success. The person who wants help to stop smoking should investigate available community resources; the library is good place to start. If the first attempt fails, additional attempts should be planned.

A NOTE ON SMOKELESS TOBACCO

To the extent that chewing tobacco and dipping snuff can cause nicotine to be delivered to the brain in sufficient doses, they present a similar risk of nicotine dependence in the regular user. These products may prove more difficult to treat than cigarette use, because they are sometimes viewed as less risky alternatives to cigarettes. One study quoted in a Surgeon General's report on smoking reported that 77 percent of youth thought that cigarette smoking was very harmful, but only 40 percent rated smokeless tobacco as very harmful. Once the "negative publicity" on smokeless tobacco use reaches a level close to the bad press on smoking, there should be a growing demand for using the smoking therapies as treatments for the use of smokeless tobacco.

In addition to the problems associated with nicotine addiction, smokeless tobacco can cause bleeding gums and sores of the mouth that never heal. It is also associated with cancer. Smokeless tobacco also stains the teeth a dark yellow-brown color, gives the user bad-smelling breath, and can cause dizziness, hiccups, and vomiting in the individual. A further risk associated with smokeless tobacco is that youth who use it are more likely to try smoking than those who do not use it.

(SEE ALSO: *Addictions: Concepts and Definitions; Nicotine Delivery Systems for Smoking Cessation; Tobacco: Treatment Types*)

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Tobacco, Pharmacotherapy Although tobacco use causes a powerful addiction, people who want to stop using it can be helped, and at far less expense than treatment of tobacco-caused diseases—which will kill approximately one in two smokers who do not quit. The effort to find pharmacological agents that would help tobacco users quit is not a new development. In the late 1890s and early 1900s, a number of potent medicines were advertised as being useful for reducing tobacco craving and helping break the habit. Such advertising was possible because at the time there

were no regulations requiring a seller to demonstrate that the product was effective. None of the products offered to the public between the early 1900s and the late 1970s were demonstrably better than placebos in helping smokers quit. Effective pharmacological approaches to treating nicotine addiction, including transdermal patches that deliver nicotine through the skin, and resin complexes (gum) that release nicotine when chewed, were among the important medical advances of the 1980s and 1990s. To understand how pharmacotherapy works, it is necessary to understand the role of NICOTINE in the addiction to tobacco.

Nicotine is a naturally occurring alkaloid present in the tobacco leaf. It is a small lipid and water-soluble molecule, rapidly absorbed through the skin and mucosal lining of the mouth and nose or by inhalation in the lungs. In the lungs, nicotine is rapidly extracted from tobacco smoke within a few seconds because of the massive area for gas exchange in the alveoli; it is passed into the pulmonary veins, and pumped through the left ventricle of the heart into the arterial circulation within another few seconds. Within 10 seconds, a highly concentrated bullet (bolus) of nicotine-rich blood reaches organs such as the brain as well as the fetus of a pregnant woman. Arterial blood levels may be ten times higher than venous levels within 15 to 20 seconds after smoking. Nicotine arterial boli from smoking a single cigarette may be three to five times more concentrated than the low, steady levels obtained from nicotine gum or patch systems. These spikes probably contribute to the pleasure sought by the cigarette smoker, but, fortunately, they are not necessary to relieve withdrawal symptoms. NICOTINE GUM and patches, which provide more steady nicotine levels without arterial spikes, may selectively relieve withdrawal without the highly addictive nicotine spikes produced by cigarettes. Although SMOKELESS TOBACCO users do not obtain the same rapid nicotine increase as smokers, they may, by repeatedly putting new "pinches" in their mouths, achieve stable nicotine levels higher than those typical of smokers.

Most cigarettes on the U.S. market contain 8 to 9 milligrams (mg) of nicotine, and the average smoker obtains 1 to 2 mg per cigarette. In general, the type of cigarette or nicotine delivery rating reported by the manufacturer bears almost no relation to the level of nicotine obtained by the typical smoker, because smokers may change their behavior to compensate for differences in cigarette brands. For example, they may take additional puffs on low-nicotine brands.

Cigarette smoking produces rapid and large physiological changes, but, to a lesser extent, smokeless tobacco produces similar effects. Nicotine gum and patch treatments have the advantages of much slower nicotine delivery, and they produce less severe physiological changes. This slower delivery rate may be less pleasurable to the tobacco user, but the user is less likely to have difficulty giving up the gum or the patch after treatment.

Tobacco-caused cancer may be considered a side effect of nicotine dependence in much the same way that ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) may occur as a side effect of heroin dependence. In both cases, the exposure to the disease-causing toxins or to HIV occurs repeatedly and often frequently because individuals are dependent on a drug that has reduced (if not nearly eliminated) their ability to abstain from the highly contaminated drug delivery system they know may lead to disease and premature death.

The physiological basis of drug dependence became increasingly well understood in the past few decades and especially with regard to nicotine dependence in the 1970s and 1980s. Awareness of the physiology of nicotine dependence can help researchers understand the problems faced by people attempting to give up tobacco and can provide a more rational basis for the development of treatment programs that may prevent the occurrence of cancer and other diseases or contribute to remission in people who have been treated for cancer.

TOLERANCE as a result of repeated nicotine exposure is a crucial factor in the development of lung and other cancers. Essentially, smokers self-administer much greater amounts of tobacco-delivered toxins than would be the case if they had not developed tolerance. In turn, with development of nicotine dependence, smokers come to feel normal, comfortable, and most effective when taking the drug and to feel unhappy and ineffective when deprived of the drug. This process makes it more difficult to achieve and sustain even short-term abstinence.

PHARMACOLOGICAL TREATMENTS

Most smokers have quit on their own or, rather, tried to quit. Although 18 million try each year, less than 7 percent do so successfully. Most of the efforts were "cold turkey," good for a start, but the least effective of all techniques. Long-term abstinence rates are low for people using this method. Treatment programs are helpful in increasing rates of success, and the availability of pharmacological interventions gives clinicians additional useful tools to help the smoker. The major pharmacological approaches are nicotine replacement, symptomatic treatment, nicotine blockade, and deterrent therapy. Nicotine replacement and symptomatic treatment have become part of general medical practice. Until further information is collected, blockade and deterrent therapy must be considered experimental.

Nicotine Replacement. The rationale for nicotine replacement is to substitute a safer, more manageable, and, ideally, less addictive (more easily discontinued) form of an abused drug to alleviate symptoms of withdrawal. An example of a less-addictive substitute is METHADONE MAINTENANCE for opiate abusers. Various forms of nicotine replacement have been developed including polacrilex (gum), transdermal delivery systems (patches), nasal vapor inhaler, nasal nicotine spray (gel droplets), and smoke-free nicotine cigarettes. The forms provide different doses and speeds of dosing. These parameters may be important in offering the smoker levels of nicotine necessary to alleviate withdrawal and cravings for nicotine. Currently, only the nicotine gum and patch are approved for use in the United States.

Several advantages exist in replacing nicotine from tobacco with non-tobacco-based systems such as gum or patches. First, they do not contain all the toxins present in tobacco or produced by burning tobacco. Second, total daily nicotine administration is lower for most patients on nicotine-replacement systems, and the high initial nicotine bolus doses produced by inhaling are not delivered. Third, the clinician can control doses more effectively than with tobacco-based products. The patient cannot, for example, take a few extra puffs per cigarette and defeat the purpose of gradual nicotine-reduction plans.

Nicotine gum may not be absorbed well if the client does not follow directions carefully. From

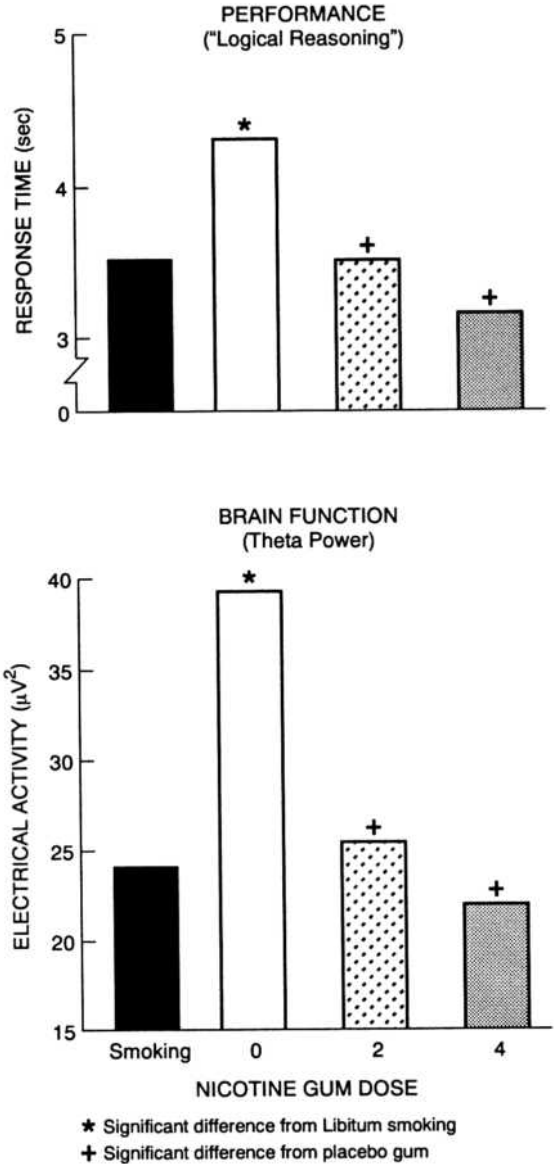

Figure 1
Cognitive Performance and an Electrophysiological Measure of Brain Function during Smoking and Abstinence with Nicotine- or Placebo-Delivering Gum Treatment.

1984 until 1991, about 1 million prescriptions for nicotine gum, the only form of nicotine replacement then available, were filled per year. At the end of 1991, nicotine patches were introduced, and approximately 7 million prescriptions were filled for all replacement systems, with the nicotine patch accounting for nearly 90 percent of new prescriptions for nicotine replacement. The popularity of

the nicotine patch can be measured by the higher rate of compliance than for the only currently available alternative, nicotine gum. Nicotine gum compliance rates tend to be lower because patients may dislike the taste and experience slightly sore mouths, throats, and jaws and gastrointestinal upset. Nevertheless, a study at the Addiction Research Center of the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) found nicotine gum to be effective in treating the cognitive function and corresponding brain electrical function changes of tobacco withdrawal. The effect was stronger at higher dose levels (e.g., 4 mg; see Figure 1). Because of current prescribing practices, this section will concentrate on the nicotine patch.

Four brands of nicotine patch are currently available in the United States. All deliver a given dose of nicotine transdermally, through the skin, over either a 24-hour (Habitrol, Prostep, and Nicoderm) or a 16-hour (Nicotrol) period. No clinical study has directly compared the four brands, but there is no evidence that any one brand leads to consistently higher rates of abstinence than any other. Variations in nicotine-delivery rate and skin contact effects may mean that certain patches work better for some people than others, but there is as yet no way to tell which patch will work better for an individual patient.

The nicotine patch is highly effective, resulting in an overall doubling of smoking cessation rates. Different studies have reported cessation rates of between 22 percent and 42 percent after six months of use. The combination of intense counseling and patch use was associated with higher success rates.

Work is necessary to develop a list of characteristics of those patients most likely to benefit from nicotine patch use. The University of Wisconsin's Center for Tobacco Research and Intervention suggests that patients may benefit if they are motivated to quit and fit into at least one of the following categories:

- Smoke at least 20 cigarettes per day
- Smoke first cigarette within 30 minutes of awakening
- Have experienced a strong craving for cigarettes during the first week of previous attempts at quitting

The nicotine patch should be applied as soon as the patient awakens, and the user should stop all smoking during patch use. The patch should be applied to a hairless part of the body, with a different site every day. The same site should not be used again for one week. Side effects include a local skin reaction at the patch application site in 30 percent of patients and possibly sleep disruption. Because the tobacco-withdrawal syndrome also may include sleep disruption, it is sometimes difficult to determine whether the sleep disturbance is a result of tobacco withdrawal or nicotine patch therapy.

The four patches vary in their recommendations for length of treatment, from six to sixteen weeks. Because no published studies have documented a benefit for longer treatment, some researchers recommend 6 to 8 weeks for most patients, but therapy should be individualized where appropriate. Other researchers have concluded that, in general, the chances of success appear better in longer-term use.

In patients with cardiovascular disease, the nicotine patch may be used cautiously, although there has been no documented association between patch use and acute heart attacks. It should be used in pregnant patients with caution—only after they have failed to quit using nondrug means. Nicotine replacement should not be given to people who continue to smoke, although the advisability of terminating therapy if only occasional cigarettes are smoked is subject to debate.

Nicotine delivered by tobacco products is one of the most highly addictive substances known. Even people highly motivated to quit may have profound difficulty doing so on their own. It is now known that people differ greatly in the severity of their addictions and their ability to cope. Our ability to treat nicotine addiction is continually improving. Even so, many people will require several repeated quitting attempts, regardless of treatment used. Therefore, long-term support by public health organizations and other facilities is essential if we are to prevent the serious diseases that will affect one in two untreated smokers.

Recent data from the 3 million people treated with the nicotine patch during its first seven months of availability in the United States increase optimism that the body can repair much of the damage caused by smoking. Epidemiological data indicate that 2,250 heart attacks would have occurred if these smokers had continued their habit. In fact, the Food and Drug Administration (FDA) received reports of only 33 severe cardiovascular problems. Even assuming underreporting, this de-

crease is so profound that it strongly supports the conclusion of the surgeon general in 1991 that risk of heart attacks rapidly declines after smoking cessation. These people were receiving nicotine via the patch, although probably at a lower level than if they continued smoking, and still their rate of heart attacks was significantly reduced.

Symptomatic Treatment. Nicotine administration and withdrawal produce a number of neurohormonal and other physiological effects. Symptomatic treatment methods are nonspecific pharmacotherapies to relieve the discomforts and mood changes associated with withdrawal. If the potential quitter relapses to escape the suffering of withdrawal, these methods should help to prevent such relapse. There is a long history of pharmacological treatment of smokers. To reduce withdrawal, sedatives, tranquilizers, anticholinergics, sympathomimetics, and anticonvulsants have all been tried at one time and were no more successful in helping smokers quit than was a placebo. CLONIDINE is one agent that has been tried in the treatment of nicotine withdrawal discomfort and is commonly used to treat opioid withdrawal. Glassman and his colleagues (1984; 1988) administered clonidine to heavy smokers on days they abstained from smoking and found that it reduced anxiety, irritability, restlessness, tension, and craving for cigarettes. When they gave clonidine to smokers trying to quit, 6 months later, 27 percent of those given clonidine and 5 percent of those given placebo reported abstinence. Surprisingly, clonidine seemed to be effective only for women. Among men, those given clonidine did no better than those given a placebo. Before recommending clonidine for smokers, practitioners should consider potential side effects. Clonidine has been used to treat hypertension, and abrupt termination has sometimes led to severe hypertension and in rare circumstances to hypertensive encephalopathy and death. More commonly, it may cause drowsiness, potentially dangerous to someone operating machinery or driving.

Among nicotine's effects is the regulation of mood. Smokers have been shown to smoke more than usual during stressful situations; therefore, those trying to quit often relapse (begin smoking again) during stressful situations. These observations suggest that treating the mood changes associated with abstinence with, for example, BENZODIAZEPINE tranquilizers, ANTIDEPRESSANTS, or psychomotor stimulants may improve abstinence rates. The benzodiazepine tranquilizer alprazolam was also examined by Glassman and his colleagues (1984; 1988) and found to reduce anxiety, irritability, tension, and restlessness, but it had no effect on cravings for cigarettes in heavy users abstaining from smoking for one day. More study is necessary on its effectiveness in maintaining tobacco abstinence.

Nicotine Blockade. Nicotine blockade therapy is based on the rationale that if one blocks the rewarding aspects of nicotine by administering an antagonist (or blocker), the smoker who seeks the pleasant effects nicotine produces will be more likely to stop. To be effective, the drug must be active in the central nervous system (brain and spinal cord). Thus mecamylamine, which acts at both central and peripheral nervous system sites, effectively increases rates of abstinence, whereas hexamethonium and pentolinium, which block peripheral nervous system receptors only, have no effect on abstinence. The problem is that there are no pure nicotine antagonists currently available. Drugs like mecamylamine produce side effects, such as sedation, low blood pressure, and fainting, that probably limit their role to that of an experimental tool, not appropriate for clinical treatment.

Deterrent Therapy. The rationale for deterrent therapy is that pretreatment with a drug may transform smoking from a rewarding experience to an aversive one if the unpleasant consequences are immediate and strong enough. DISULFIRAM treatment for alcoholism is an example of this type of treatment. After pretreatment, even a small quantity of alcohol can produce discomfort and acute illness. Silver acetate administration is a potential treatment for smokers. When silver acetate contacts the sulfides in tobacco smoke, the resulting sulfide salts are highly distasteful to most people. Although many over-the-counter deterrent products are available, their effectiveness has not been scientifically validated. Additionally, a severe limitation to this treatment is compliance. It may be difficult to ensure that patients continue to take the medication as needed.

BEHAVIORAL TREATMENTS

Characteristics of tobacco dependence and nicotine addiction suggest that combining nicotine replacement, to reduce the physiological disruptions

of withdrawal, with behavioral treatments, to counter the conditioning cues, reinforcers, and social context cues associated with smoking, may be especially useful in helping people to quit. Adding behavioral treatments may increase both the rate of successful outcomes and the adherence to the pharmacological treatment. Behavioral interventions for smokers have been tried for many years. This section will focus on several of the current major approaches, but it is by no means comprehensive.

Social support has produced mixed results. Enlisting the help of the smoker's spouse and coworkers, or encouraging participation in a group, has yielded generally positive outcomes, but attempts to enhance social support further have been uniformly unsuccessful. Providing *skills training* in coping with stress and negative emotions has also been tried but generally as part of a multicomponent treatment plan. If the person smokes during times of stress and negative emotions, learning other means of dealing with these situations may lessen the need to smoke. Skills training appears beneficial in the short term, especially when combined with aversive smoking procedures (discussed below), but its long-term benefits are less clear. Mixed but generally negative results have been reported, but a problem in assessing skills training is that researchers have not controlled for the differences in treatments available. Some may be more effective than others. The techniques should be available for clients long after learning in order to be beneficial for long-term smoking cessation.

Contingency contracting uses operant conditioning techniques to reinforce quitting or punish smoking behaviors. Procedures include collecting monetary deposits from clients early in treatment and providing periodic repayment as nonsmoking goals are reached, having a client pledge to donate money to a disliked organization for every cigarette smoked, or similar procedures using nonmonetary rewards or punishers. Research indicates that contingency contracting aids quitting at least in the short term. *Stimulus control* procedures gradually eliminate situations in which the client smokes (e.g., only smoke outside) or the time the client smokes (e.g., only on the half hour) to reduce the number of cues for smoking.

Nicotine fading gradually changes brands or cigarette filters the smoker uses, in order to decrease tar and nicotine per cigarette before complete cessation. It is hoped this strategy will decrease later withdrawal symptoms when the client stops smoking. Problems are that the procedure may do nothing to reduce cravings (considered important for relapse prevention) and that the nicotine reduction is not as large as one would expect from ratings of the cigarettes' contents, because people change the way they smoke to receive more nicotine from each cigarette. Improved outcomes may occur with nicotine fading when it is part of multicomponent treatment approach.

Aversion treatments are designed to condition a distaste for cigarettes by pairing smoking with either unpleasant imagery (covert sensitization), electric shock, or unpleasant effects of smoking itself through directed smoking procedures. Directed smoking techniques include satiation, rapid smoking, and focused smoking. In satiation, clients smoke at least at twice their regular rate. Research indicates a low, 15 percent success rate when satiation is used by itself, versus 50 percent when it is part of a multicomponent program. In rapid smoking, clients inhale every 6 seconds until they will get sick, usually for six to eight sessions. As part of a multicomponent program, good outcomes are seen, but success is variable when rapid smoking is used alone, with high immediate abstinence rates, followed by low long-term rates. In focused smoking, clients either smoke for a sustained period at a slow or normal rate or do rapid puffing without inhaling. Long-term outcomes are similar to or slightly lower than for rapid smoking. The utility of aversion procedures is limited because the aversions are rarely permanent, and it is difficult to condition aversion to a substance that has had repeated past use.

CONCLUSIONS

Multicomponent interventions that combine pharmacological and behavioral components appear to be the best treatment strategies, often producing very high short-term (nearly 100% for the best programs) and impressive long-term success rates (at or above 50%). Ideally, the components should complement one another; however, it is not known how the separate components work in combination. It is possible that, because people smoke for different reasons (to prevent withdrawal, to ease anxiety, to relax, to achieve pleasant effects), a program that includes components that target enough different reasons for smoking will be successful in most cases. Second, it is not known which

components work best together or how to target interventions for particular types of people. Third, a concern in designing a multicomponent treatment plan is that too many interventions may decrease patient compliance. Despite these gaps in our knowledge, smoking-cessation programs are improving constantly, and smokers do not have to go it alone in their attempts to quit.

(SEE ALSO: *Addiction: Concepts and Definitions; Nicotine Delivery Systems for Smoking Cessation; Relapse Prevention; Tobacco; Treatment Types*)

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Tobacco, Psychological Approaches

Persistent use of tobacco products is believed to result from the rewarding effects of nicotine, a psychostimulant found in tobacco. Individuals become dependent on tobacco, in part, because of nicotine's positive psychoactive effects (e.g., mild euphoria, stimulation, improved concentration). Continued use of tobacco products is also reinforced by the alleviation of unpleasant withdrawal symptoms that often occur during nonuse or abstinence (e.g., irritability, weight gain). However, tobacco dependence results not only from the pharmacological effects of nicotine that eventually lead to physical addiction, but also from the psychological and behavioral components associated with tobacco use.

Psychological reliance on tobacco is likely to be a result of the psychoactive effects from nicotine and the use of tobacco. For example, a cigarette smoker may smoke to modulate moods or deal with stress. The behavioral components are a result of learning that certain contexts or stimuli are associated with

smoking behavior with consequent desirable effects. After repeated self-administration of nicotine-containing tobacco products, these contexts or stimuli begin to control behavior. Pharmacological treatments are often used to deal with the physical addiction to nicotine. However, psychological or behavioral approaches are used to help smokers learn more adaptive ways to deal with situations other than using tobacco products and to engage in more adaptive behavior in response to stimuli associated with smoking.

This section will discuss assessing whether tobacco users are ready to quit tobacco products, methods to motivate them to quit, and behavioral treatment methods that have been found to be effective, and combining pharmacological and behavioral treatment approaches.

ASSESSMENT OF READINESS TO QUIT TOBACCO USE

The application of behavioral treatments to tobacco-dependent individuals begins with an assessment of preparation for change. Readiness to change negative health behaviors has conceptualized in the transtheoretical model originated by James Prochaska and Carlos DiClemente. This model posits that there are reliable Stages of Change in health awareness and motivation, and that appropriate treatments vary by the stage. There are five stages of change: (1) precontemplation, a period where during the next 6 months, the tobacco user is not considering quitting; (2) contemplation, a period when a tobacco user is seriously considering quitting in the next 6 months; (3) preparation, a period when, a tobacco user who tried quitting in the previous year, thinks about quitting in the next month; and (4) action, a 6 month period after the tobacco user makes overt changes to stop using tobacco products. The last stage, maintenance, is the longest and describes the tobacco-free period after cessation. To assess stage of change, informal questioning or a brief list of structured questions (i.e., the University of Rhode Island Assessment Scale [URICA]), has been employed.

At any time, the majority of smokers are precontemplators, contemplators, or preparers, and these individuals lack the motivation to justify the intensive behavioral techniques described below. The behavioral techniques described later in this section are most applicable to the action stage. At all stages of change, education about nicotine dependence is essential. Education about nicotine dependence should emphasize a couple of major points. First, chronic use of nicotine changes the brain, leading to a complex neurobiological disorder. Second, nicotine withdrawal is a difficult but time-limited syndrome typically taking one to three weeks to subside, with weight gain and cravings persisting longer. Nicotine withdrawal can involve negative mood, insomnia, anxiety, impaired attention and concentration, restlessness, and weight gain. Knowing why one uses tobacco and what lies ahead as well as knowing that effective treatment techniques are available, can help to motivate a quit attempt and enhance self-efficacy, the belief that one has the ability and tools to achieve abstinence from tobacco. For those in the action stage, providing counseling that involves problem solving and developing coping skills is most effective.

PRINCIPLES OF BEHAVIORAL MODIFICATION

Classical behavioral treatments in tobacco cessation are based on the principles of behavioral modification, where the antecedents and the consequences of tobacco-use behavior are examined. Consequences are events that occur after the use of tobacco. If the consequences increase behavior, then the process is termed reinforcement. There are two major types of reinforcement: positive and negative. Positive reinforcement involves the presentation of an event that then increases behavior. Negative reinforcement involves the removal of an event that also results in increased behavior.

Both positive and negative reinforcements initiate and maintain tobacco use. Positive reinforcement from smoking cigarettes, for example, may include improving concentration. Negative reinforcement from smoking cigarettes may include reduction of tension, depressed mood, or prevention of withdrawal symptoms.

If the consequence decreases behavior, then the process is termed punishment. Punishment can involve presentation of an event or removal of an event. For example, the occurrence of social disapproval, negative physical consequences, and increased cigarette taxes may reduce smoking. Similarly, the removal of privileges, such as being

unable to participate in sports can decrease smoking behavior and serve as punishment.

Despite the many negative consequences of tobacco use, it often persists in many who try it. There are many antecedents or events that precede tobacco use, that begin to control or maximize the occurrence of tobacco use, the process called stimulus control. An individual learns that in certain situations, behavior is reinforced; while in other situations, it is either not reinforced or punished. For example, a smoker may learn smoking in bars is reinforced socially as well as by nicotine's effects, whereas smoking in church is not reinforced. Upon repeated experiences, frequenting bars begins to automatically elicit the desire or behavior for smoking, while in contrast attending church does not. In large part, the punishing effects of tobacco use and particularly the reinforcing effects of cessation are relatively remote (i.e., occur years in the future), while the reinforcing consequences of smoking (e.g., mood regulation) are more immediate. The strength of any reinforcer or punishment diminishes the further removed from the actual behavior, and thus tobacco use is often maintained for decades.

Behavioral treatments involve manipulating these antecedents and consequences to reduce the probability of tobacco use. Further, skills that foster non-tobacco use behaviors such as stress management skills and assertiveness are also taught or encouraged.

BEHAVIORAL, SUPPORTIVE, AND OTHER TREATMENTS

Since the 1960s, many behavioral techniques have been developed to help tobacco dependent people quit, but only a few techniques have shown reliable evidence of efficacy. Efficacy is generally defined by comparing abstinence rates (i.e., proportion not using tobacco products) at six months or a year after quitting. In 2000, the Agency for Health Research and Quality (AHRQ) released a second comprehensive evaluation of these techniques using meta-analysis, a method of quantitative literature review. The review identified four areas of behavioral treatment or psychosocial support that were associated with significantly higher quit rates: (a) intra-treatment support; (b) extra-treatment support; (c) problem solving and skills training; and (d) aversive techniques. The first two approaches represent supportive psychological treatments, whereas the latter two emphasize behavioral aspects of smoking and employ some principles of behavioral modification. Before considering them, the actual act of quitting and relevant approaches are detailed. Finally, brief descriptions of some techniques whose clinical efficacy has not been supported will be provided.

QUITTING

Several techniques have been developed to help the individual quit using tobacco products. One technique, quitting abruptly (''cold turkey''), is best executed on a planned quit day and as part of a broader treatment strategy (e.g., involving intra-treatment support). In contrast, gradual reduction involves slowly reducing tobacco use until it reaches zero. Several reduction approaches are available including one where the number of cigarettes smoked each day is reduced (either through lengthening the time between cigarettes or delaying the onset of smoking) and one where situations where tobacco is used are slowly restricted. Unfortunately, a significant number of smokers experience difficulty in reducing the number of cigarettes beyond a certain point. Other gradual reduction methods include using cigarette filters with ventilation holes that can decrease the amount of nicotine obtained from each cigarette or gradually reducing the nicotine content of the cigarette. However, these methods may result in compensatory smoking, that is puffing more or longer, or smoking more cigarettes to make up for reduced nicotine. An important goal of tobacco reduction methods is the reduction of withdrawal signs and symptoms from tobacco, which gradual reduction does in fact achieve. However, gradual reduction may prolong withdrawal symptoms for a period longer than abrupt cessation.

Since the 1980s, a number of pharmacological agents have been developed for the treatment of smokers. Nicotine replacement therapies (e.g., nicotine gum) and novel non-nicotine pharmacotherapies, such as buproprion (Zyban) have been found to significantly reduce withdrawal signs and symptoms. Because of the uniform efficacy of these products, their use has been recommended for most smokers to aid cessation (excluding smokers who have certain medical illnesses, pregnant women, or adolescents).

In summary, tobacco users are typically advised to set a quit date and to take medications to assist in their cessation efforts. If a smoker does not want to use medications, abrupt cessation can be used or if the smoker is concerned about withdrawal, a gradual approach may be taken.

INTRA-TREATMENT AND EXTRA-TREATMENT SUPPORT

The process of quitting smoking can be difficult, and support and encouragement can greatly help. In intra-treatment support, healthcare providers (e.g., physicians) improve quit-rates through support and encouragement (e.g., by recognizing the discomfort of quitting, underscoring that half of all smokers have quit for good, and noting that effective therapies exist). In addition, by providing training in acquiring extra-treatment support, the tobacco user can effectively obtain additional care from family members, friends, and telephone hotlines. Further, supportive others (e.g., spouse) can be contacted with information on tobacco cessation or encouraged to participate directly in treatment with the tobacco user.

PROBLEM SOLVING AND SKILLS TRAINING

Problem solving and skills training involve learning to recognize patterns of tobacco use and situations where use is common through self-monitoring and learning ways to effectively deal with these high risk situations.

Self-monitoring requires an individual using tobacco products to monitor situations and feelings that are associated with tobacco use. Through self-monitoring, the individual begins to recognize specific antecedent conditions that are associated with the use of tobacco. Antecedent conditions for a cigarette smoker often involve environmental contexts or situations (e.g., smoking the first thing in the morning) while others involve internal cues or psychological states (e.g., being under pressure). In these situations, tobacco users are most likely to experience craving or an urge to use tobacco products. Understanding and recognizing these situations and psychological states will promote learning skills to handle them.

Adequate problem solving and coping skills are essential to remaining tobacco free. Problem solving includes learning how to assess potential relapse situations adequately, developing a number of solutions, and trying out these solutions. Solutions involve the use of coping skills. One type of coping skill is learning how to deal with stimulus control or high-risk situations. One method is to avoid stimuli associated with tobacco, such as the smoking section of a restaurant. Also, smokers can put themselves in situations that prevent or discourage tobacco use (e.g., movie theatre, non-smoking restaurant). Unavoidable situations and psychological states can be countered through cognitive strategies such as distraction and positive thinking. Other techniques include using substitutes that may simulate some of the stimulus qualities or effects of smoking (e.g., chewing gum, sucking on straws). In addition, craving to use tobacco products lasts only minutes, and using distractions (e.g., exercising) can occupy the tobacco user until the craving passes. Tobacco users are also taught to practice refusing tobacco or asking others not to use tobacco around them. Often tobacco users have employed nicotine instead of coping skills that could be used to counter stress and negative affect, and training in use of adaptive coping skills can be beneficial.

The deprivation of nicotine and tobacco can be offset by the provision of rewards. Rewards can include saving money that is typically spent on cigarettes to reinforce the cost of the habit and to pay for pro-health activities like vacations. Rewards can also be leisure activities (e.g., reading a book, going to a movie). Finally, rewards can be self-affirming statements such as, ''I did really well today.'' Rewards are initially given for small successes, based on achieving a goal behavior (e.g., not smoking for 72 hours), and occur as soon as possible upon completion of this behavior.

AVERSIVE TECHNIQUES

Rapid smoking is one aversive technique that has been found effective. Smokers are asked to smoke several consecutive cigarettes rapidly so that they will experience immediate adverse, punishing effects (e.g., nausea), thereby reducing the desire to smoke. Similarly, reduced-aversion techniques also facilitate smoking cessation by their unpleasant effects and improve the effectiveness of behavioral treatment. This technique involves focusing on smoking while the person smokes for a sustained

period of time, or on rapid puffing with no inhalation of the smoke.

OTHER TECHNIQUES

Several other techniques for tobacco cessation have failed to show results superior to a non-treatment control group, but may still be useful in treatment programs that employ multiple behavioral techniques. Relaxation or breathing techniques involve deep breathing or meditation in anticipation or response to urges to use tobacco. Programs designed to specifically counter negative affect seek to help the tobacco user to identify negative feelings, assess and appraise the situations that lead to the negative affect, and respond to them realistically and productively. Programs designed to counter increased weight on cessation (on average about seven pounds), have not improved quit rates, and can actually reduce the chances of successfully quitting. Two commercial treatments, hypnosis and acupuncture continue to be popular, but their lack of efficacy and unclear bases for action do not support their use.

INTENSITY OF BEHAVIORAL TREATMENT

A separate question from which behavioral treatments to give is how much or how intense the treatment should be? Treatment intensity involves the number of treatment sessions, the length of these sessions, and also the total amount of time spent throughout treatment providing behavioral treatments and support. The AHRQ guideline recommends that an intensive treatment should include four or more sessions, with each session lasting at least ten minutes, and that the total contact time should be longer than thirty minutes. Providing additional contact time and support will increase quit rates, but need to be weighed against the financial costs and likely loss of patient participation if the contact is spread of many weeks.

RELAPSE PREVENTION

Once a tobacco user has quit consuming tobacco, the challenge is to prevent relapse, the return to regular tobacco use. Relapse is distinguished from a slip, which is smoking one or few cigarettes after a period of abstinence. However, slips, especially during the initial weeks of quitting, generally lead to relapse. Therefore, smokers or tobacco users are instructed not allow themselves use of any tobacco products (e.g., not one puff). Maintaining abstinence involves developing both behavioral and cognitive skills that go beyond the initial challenges of nicotine withdrawal. Long term abstinence may be supported through health-oriented lifestyle changes such as increased levels of physical activity, proper eating, obtaining enough sleep and rest, and managing or changing levels of stress in adaptive ways.

CONCLUSION

Many of the techniques used in psychological treatment for smoking cessation have been described in this article. Studies show that smoking interventions are most effective when multiple techniques are used, and that increasing treatment contact can further improve treatment outcome. Unfortunately, nicotine is a highly addictive drug, and relapse to smoking cigarettes or other tobacco use remains high, in spite of behavioral treatment and pharmacological interventions. Following treatment, most tobacco users begin to relapse with only twenty to thirty percent still tobacco free after six months from quitting.

Use of both pharmacotherapies and psychological treatments for smoking cessation can increase success rates, with combinations used to target different aspects of nicotine addiction. For example, pharmacotherapies such as nicotine gum or bupropion reduces the physical dependence aspects of smoking, which then allows the tobacco user to focus on the behavioral or psychological aspects of smoking. The intensity of behavioral treatment and whether pharmaceutical treatments are prescribed depends on the characteristics of the smoker (e.g., degree of dependence).

In order to help tobacco users receive treatment appropriate to their stage of change and tobacco and health histories (i.e., level of nicotine dependence, previous quit attempts), a stepped care model has been proposed. In the stepped care framework, a process called tailoring is used so that the most appropriate treatment is given. Those in the precontemplation, contemplation, and action stages are given information about the health risks of tobacco use, the benefits of cessation, resources for a later quit attempt, and a follow-up is planned to reassess their readiness to quit. When tobacco

users make an initial quit attempt a minimum of intra-treatment behavioral support is used, in conjunction with self-help materials and if necessary pharmacotherapy is recommended. Tobacco users who have failed to quit with less intensive treatments can then be "stepped up" to a program involving more contact, different behavioral interventions, and pharmacotherapies. In all cases, planned follow-up is essential to determine if additional treatment is needed.

Of final note, most cigarette smokers quit on their own, without treatment, but their quit rates are the lowest of any approach (e.g., compared to behavioral or pharmacological treatments). If smokers do seek treatment, they tend to obtain help from their physician or from health-care providers who often do not have time to provide intensive behavioral treatment. Therefore, availability and use of the behavioral techniques for smoking cessation are being increasingly adapted to these various methods or settings for tobacco cessation (e.g., teaching smokers how to obtain extra-treatment support, such as through telephone counseling). Telephone counseling is a particularly promising source of treatment support that can provide intensive counseling without the need for costly travel and missed work. Recently, awareness that tobacco cessation is not possible for all individuals, at least as the initial goal in treatment, has given rise to studies of tobacco use reduction. The role for psychological treatments in this burgeoning area is not clear but will doubtless be important. In the long run, however, societal pressures (e.g., banning smoking in public places) and economic pressures (e.g., increasing taxes on tobacco products) will likely have the greatest impact in reducing tobacco use and in encouraging cessation.

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Twelve Step Facilitation (TSF) Twelve Step Facilitation (Nowinski & Baker, 1998; Nowinski, Baker, & Carroll, 1992) is a manual-guided, twelve-step based treatment program that includes a range of interventions that are organized into a "core" or basic program, an "elective" or advanced program, and a brief conjoint program for the substance abuser and a significant other. Interventions in the core program are most appropriate for what could be termed the "early" or initial stage of recovery from alcohol or drug dependence, meaning that stage of change in which an individual takes their initial steps from active substance abuse toward abstinence.

TSF is a highly structured intervention whose sessions follow a prescribed format. Each begins with a review of the patient's recovery week, including any 12-step meetings attended and reactions to them, episodes of drinking or drug use versus sober days, urges to drink or use, reactions to any readings completed, and any journaling that the patient has done. The second part of each TSF session consists of presenting new material, consisting of material drawn from the core, elective, or conjoint program. Each session ends with a wrap-up that includes the assignment of recovery tasks: readings, meetings to be attended, and other pro-recovery behavioral work that the patient agrees to undertake between sessions.

The various TSF interventions, or 'topics' are of two types: Core and Elective. Core sessions include Introduction & Assessment, Acceptance, People, Places, & Routines, Surrender, Getting Active. Elective (advanced) sessions include: Genograms, Enabling, Emotions, Moral Inventories, Relationships. There is also a conjoint program.

Patients need not necessarily be dependent on either alcohol or drugs in order to benefit from a 12-step oriented treatment; rather, they must merely satisfy the basic criterion for becoming member of a 12-step fellowship as set forth by Alcoholics Anonymous, namely, "a desire to stop drinking," or to stop using drugs (Alcoholics Anonymous, 1952). However, 12-step fellowships do advocate abstinence, as opposed to controlled use of alcohol or drugs. Historically, these fellowships were founded and exist to provide support and advice, and to facilitate the personal growth of individuals whose own efforts to control their use of alcohol and/or drugs have failed and whose lives have became "unmanageable" as a consequence of substance abuse (Alcoholics Anonymous, 1976).

EARLY RECOVERY

Based on an assessment of the patient's lifestyle, prior treatment experiences, periods of sobriety, and circumstances surrounding relapse, an individual treatment plan is devised, typically including one or more elective topics plus the core TSF program. Broadly speaking, early recovery can be broken down into two phases: acceptance and surrender. Acceptance refers to the process in which the individual overcomes "denial." Denial refers to the personal belief that one either does not have a substance abuse problem, and/or that one can effectively and reliably control drinking or drug use. Acceptance represents a significant insight: That one has in fact lost the ability to effectively control use of alcohol or drugs. Acceptance is marked by a realization that one's life has become progressively more unmanageable as a consequence of alcohol or drug use, and furthermore that individual willpower alone is an insufficient force for creating sustained sobriety and restoring manageability to one's life. Given this realization, acceptance implies that the only sane alternative to continued chaos and personal failure to admit defeat (or one' s ef-

forts to control use), and to accept the need for abstinence as an alternative to controlled use. This is Step I of Alcoholics Anonymous: "We admitted we were powerless over alcohol—that our lives had become unmanageable" (Alcoholics Anonymous, 1976).

As important as insight is, alone it is not sufficient for recovery, and that is where the concept of surrender comes in. Surrender refers to a willingness to take action, and specifically to embrace the twelve steps as a guide for recovery and spiritual renewal. These are Step 2 and 3: We came to believe that a Power greater than ourselves could restore us to sanity; We made a decision to turn our will and our lives over to the care off God as we understood Him (Alcoholics Anonymous, 1976).

AA and NA are programs of action and lifestyle change, as much as they are programs of insight and spiritual renewal. Surrender follows acceptance and represents the individual's commitment to making whatever changes in lifestyle are necessary in order to sustain recovery. Surrender requires action, including frequent attendance at AA and/or NA meetings, becoming active in meetings, reading AA/NA literature, getting a sponsor, making AA/NA friends, and replacing people, places, and routines that have become associated with substance abuse and therefore represent a threat to recovery, with alternative relationships and habits of living. In TSF the action and commitment that are the hallmarks off surrender are guided to some extent by the facilitator; but they are also heavily influenced by individuals the patient encounters and begins to form relationships with within 12-Stop fellowships. One especially significant relationship that TSF actively advocates for in early recovery is that of the sponsor, who is someone already in recovery and active in a fellowship who offers guidance and support to the newcomer.

SPIRITUALITY

Twelve step fellowships regard spirituality as a force that provides direction and meaning to one's life, and they equate spiritual awakening with a realignment of personal goals, specifically a movement away from radical individualism and the pursuit of the material, toward community and the pursuit of serenity as core values.

The twelfth step of AA states: "Having had a spiritual awakening as the result off these steps, we tried to carry this message to alcoholics, and to practice these principles in all our affairs" (Alcoholics Anonymous, 1952). AA and its sister 12-Step fellowships have a long spiritual tradition, in that they challenge individuals to believe in a center of power that is greater than personal willpower. This "Higher Power" may be the fellowship itself. Substituting faith in the group (or some other higher power) for faith in personal willpower, is the essence of 12-Step recovery, and it has been likened to a form of spiritual conversion or awakening (Fowler, 1993). 12-Step fellowships believe that those who thoroughly follow their program of recovery will eventually benefit spiritually: That they will re-evaluate themselves in terms of how they relate to others, their personal goals, and their sense of purpose in life.

EFFICACY OF 12-STEP BASED TREATMENT

TSF has been found to be effective in producing significant and sustained reductions in alcohol use (Project MATCH Research Group, 1997; Seraganian et al., 1998). A further finding from Project MATCH, and supported by other research (Fiorentine, 1999), is a correlation between attendance at 12-step meetings and abstinence from alcohol and drug use. Finally, greater involvement in 12-step fellowships (e.g., getting a sponsor, taking on responsibilities) has been found to correlate positively with recovery (Emrick, 1993). Taken together, these studies offer empirical support for the efficacy of these widely used models of treatment, particularly when therapists are trained to deliver this manualized approach competently.

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TREATMENT TYPES This section provides the reader with brief descriptions of some of the diverse ways that people with substance-related problems can be helped. *Treatment Types* presents descriptions of distinct interventions that are applicable to dependence on each of a variety of drugs. In practice, though, treatment programs are hybrids, incorporating features from several distinct treatment modalities and adapting them to specific needs having to do with age, gender, ethnic, racial, and socioeconomic factors, provider preference, and the economic realities that govern delivery of treatment.

Neither this section nor the one above on *Treatment* is exhaustive. A number of substance dependence interventions employed in other countries and by certain U.S. ethnic groups (such as sweat lodges among some Native American tribes) are not covered. Nevertheless, the entries included here should allow the reader to become reasonably familiar with what is considered mainstream treatment in the United States today.

This section contains the following articles: *An Overview; Acupuncture; Approaches based on Behavioral Principles; Aversion Therapy; Behavior Modification; Cognitive Therapy; Contingency Management; Family Therapy; Group Therapy; Hypnosis; Long-Term versus Brief; Minnesota Model; Nonmedical Detoxification; Outpatient versus Inpatient; Pharmacotherapy, An Overview; Psychological Approaches; Self-Help and Anonymous Groups; Therapeutic Communities; Traditional Dynamic Psychotherapy*; and *Twelve Steps, The*.

An Overview According to the 1998 National Household Survey on Drug Abuse, of the 23.1 million Americans who used an illicit drug in the past year, 1.9 million reported some health problem due to their illicit drug use, 3.5 million reported an emotional or psychological problem due to their drug use, and 4.1 million were dependent on an illicit drug. An estimated 963,000 had received treatment or counseling for their drug use. In addition to those dependent on illicit drugs, another 9.7 million Americans are estimated to be dependent on alcohol, including 915,000 youths age 12-17. Current treatment capacity, including public and private facilities for illicit drug and alcohol treatment, is about 1.7 million treatment episodes a year—clearly short of the need.

Prior to referring an addicted patient to treatment, it is important to address certain questions: (1) What are the possible treatment alternatives? (2) What treatment modalities are best suited for a particular patient? (3) What is the efficacy of the preferred treatment? and (4) Is the chosen treatment available to the patient? As will be noted, the information base needed to answer these is often not available.

TREATMENT ALTERNATIVES

Treatment Setting. Excellent treatment can be delivered within both outpatient and inpatient settings. A more expensive inpatient program does not offer the best treatment for all individuals. The appropriate placement of a drug-dependent individual in a treatment program requires the consid-

eration of several factors, including drugs that are being used, level of psychiatric distress, potential medical complications, family or other support, and availability of child care. Intensity of treatment is not necessarily a function of setting since some outpatient treatment programs provide more intense treatment than do inpatient ones.

Inpatient Programs. Usually, inpatient settings are of three types: (1) detoxification units within medical hospitals, (2) dual-diagnosis programs within psychiatric hospitals, and (3) rehabilitation programs. The first two settings are best utilized when there is a risk of serious medical problems (e.g., seizures) or psychiatric difficulties (e.g., suicidal ideation). Medical units generally employ pharmacologic detoxification protocols that are based on the type of drugs abused and the patient's concomitant medical condition. The length of stay is usually less than two weeks. Although many patients mistakenly believe that after detoxification no further intervention is necessary, detoxification is only the beginning of treatment. The next treatment placement should be based on the needs of the patient, but, unfortunately, it often depends on other factors (e.g., community resources or the patient's insurance coverage or ability to pay).

Dual-diagnosis programs are usually based in psychiatric hospitals and are designed to treat patients with both serious psychiatric illnesses and substance-use disorders. Treatment may include individual, group, and family therapy, pharmacotherapy, relaxation techniques, and education. ALCOHOLICS ANONYMOUS (AA) or NARCOTICS ANONYMOUS (NA) groups may also be offered. Individuals may reside in these hospital units from several weeks to several months.

Rehabilitation units are usually free-standing facilities that are often based on the AA TWELVE-STEP model of treatment. Some carry out uncomplicated pharmacologic detoxifications, but many patients are already detoxified at entry. Some rehabilitation programs are staffed to offer psychiatric evaluation or treatment (or both). Therapy usually consists of education, group therapy, individual meetings, and at times, specialized groups (e.g., a women's group), usually provided by drug or alcohol counselors. Social workers may provide family therapy. Traditionally, the standard length of stay was twenty-eight days, but lack of data to support the advantages of this length and reimbursement issues have often compelled programs to reduce treatment to less than fourteen days.

Outpatient Programs. Outpatient treatment generally consists of drug-free treatment or, in cases of opiate addiction, methadone treatment. The time for outpatient drug-free treatment can range from once a week to daily daylong activities. In comprehensive treatment programs, individuals may be initially enrolled in an intensive outpatient program consisting of many structured daily activities (e.g., group therapy, individual therapy, self-help groups, educational groups, stress-management groups) and "graduate" over a certain period (ranging from one to six months) to weekly or biweekly clinic visits. Random urine testing is usually an integral part of these programs. Completion of the intensive portion of the program is usually determined by documented behaviors such as length of abstinence, attendance in groups, and keeping scheduled appointments. Initiation of change—for example, the avoidance of drug-using friends or the desire to return to work or school—may suggest readiness for a less-intensive program.

Some outpatient programs have the necessary staff and expertise to provide medically supervised detoxification. Appropriate patient selection is crucial, however. There has been a growing recognition that many patients seeking drug treatment have additional psychiatric disorders (Rounsaville, Weissman, & Kleber, 1983; Weiss et al., 1986; Rounsaville et al., 1991), and, consequently, psychiatrists have been increasingly employed in drug-free outpatient settings to both assess patients and, when necessary, provide additional psychiatric treatment.

Methadone maintenance programs are designed for patients who have been addicted to opiates for at least one year. These patients often have lengthy drug-use histories and have been unable to maintain abstinence after repeated detoxifications. Verification of opiate addiction may be determined by using a naloxone challenge test or by observing withdrawal symptoms. Because of the risk of transmitting the human immunodeficiency virus (HIV), pregnant and HIV-positive opiate-dependent individuals may be given admission priority in some programs. As is the case with drug-free treatment programs, methadone programs vary in the comprehensiveness of their services. Some additional psychosocial services provided by a methadone

program may include the teaching of job-hunting skills, family therapy, and parenting groups.

Residential Programs. Residential programs can be used as a bridge between inpatient and outpatient programs or as an alternative to them. Intermediate-care facilities, similar to those developed at HAZEL-DEN, allow individuals to live within a residential setting, be employed during the day, and receive comprehensive treatment, including group therapy, individual counseling and monitoring, and education. Both behavioral models and the principles of Alcoholics Anonymous are applied. The average stay is approximately four months.

THERAPEUTIC COMMUNITIES provide treatment within highly structured, hierarchical residential settings that stress the importance of community and recovering staff in treatment. More recently, professionals with or without prior drug histories are providing managerial expertise and treatment. Within therapeutic communities, behavior is shaped by using rewards and penalties (Kleber, 1989). Drug abusers are constantly confronted by their peers in a variety of situations regarding their functioning within the program. Jobs range from low to high status and are allocated to individuals on the basis of the length of their stay in the community, their competence, and their ability to behave responsibly. Traditional therapeutic communities recommend stays of twelve to twenty-four months whereas newer programs are experimenting with stays of three to six months.

Treatment Modalities. Treatment interventions can be categorized in terms of behavioral, self-help, psychological, or pharmacological approaches. Although a specific treatment setting may emphasize one type of intervention, additional modalities are often employed. Generally, programs proficient in using diverse treatment methods are more likely to change their therapeutic interventions if the initial approaches appear ineffective.

Behavioral Approaches. Various behavioral treatments, using the psychological theories of operant and respondent conditioning, have been designed to treat substance abuse. Experimental psychologists found that behavior could be shaped if positive consequences occurred as a result of the changed behavior. Used with drug abusers, operant conditioning is complicated since many positive and negative reinforcers may promote continued drug use. These reinforcers include: (1) the positive sensations related to the drug itself, (2) the avoidance of actual or conditioned withdrawal symptoms, (3) the perceived reduction of distressing psychologic symptoms, (4) the fear of losing a social network centered on drug use, and (5) the anxiety associated with having to confront painful issues once drug use ceases.

Several clinicians have attempted to counter drug-promoting reinforcers with other reinforcers that were contingent on non—drug taking behavior. Higgins et al. (1993) developed a voucher system in which negative urine screens were rewarded with vouchers that could be used to purchase a variety of community-based items viewed as prosocial and consistent with a drug-free lifestyle. When compared to a control group that had received standard drug counseling, it was found that the behavioral group remained in treatment longer and had more discrete periods of abstinence.

Operant techniques can be applied in various treatment settings by using fairly simple yet effective reinforcers. For example, methadone programs may offer drug abusers take-home doses for negative urine results. Because compliance is more likely to occur if the positive reinforcement is temporally linked with the desired behavior, take-home doses immediately offered after two weeks of negative urine tests work better than if the take-home doses are delayed until a prolonged period of abstinence has been accomplished. CONTINGENCY MANAGEMENT and respondent conditioning are two alternative behavioral interventions that are occasionally used for treating substance abuse. Contingency contracting applies negative contingencies to undesirable behavior. For example, patients who are concealing their drug use from their bosses, family members, or anyone else may be asked to sign a "contract" that allows their therapist to inform one or more specific individuals if their drug use resumes.

Respondent conditioning may involve the use of noxious stimuli. For example, individuals may be given a chemical that induces nausea (e.g., apomorphine) while receiving an injection of their drug of choice or while handling drug-related paraphernalia. The drug may come to induce unpleasant feelings as a result of its association with the noxious stimuli. Poor patient acceptance, ethical issues, and insufficient data regarding efficacy limit the use of these AVERSIVE TREATMENT approaches.

Self-Help Approaches. These interventions have evolved from the personal experiences and ideas generated by Bob Smith and Bill Wilson, two alcoholics who cofounded Alcoholics Anonymous. The organization has grown until, in 2000, it estimated that it numbers more than 99,000 groups worldwide. Although AA's approach to gaining SOBRIETY (the Twelve Steps) and its principles (the Twelve Traditions) are commonly integrated into many treatment programs, it remains unclear which patients benefit most from self-help programs, particularly when they are used without other interventions. The concepts of AA have also been applied to other psychoactive-substance use disorders (e.g., in the programs of COCAINE ANONYMOUS and Narcotics Anonymous).

Psychological Approaches. Psychological approaches are used to try to understand the psychological or cognitive issues that promote drug use and, with this knowledge, to provide appropriate treatment interventions. As Zweben (1986) emphasized, the goals of recovery-oriented psychotherapy change as addicted individuals progress in their recovery. The manner in which recovery "progresses" has been clearly conceptualized by Gorski and Miller (1986) in their six-stage developmental model. Each of the stages has a primary goal, and different types of psychological interventions become appropriate, depending on the goal.

During the first two phases, pretreatment and stabilization, the focus is placed on challenging the denial of patients regarding the consequences of their disease and, subsequently, on addressing the symptoms of acute and post-acute withdrawal. For therapists to engage patients into treatment, they need to be skillful at both confrontational and supportive approaches. During the third and fourth stages of early and middle recovery, the patients' major goals are to learn to function without drugs or alcohol and to develop a healthy lifestyle. For these stages, a cognitive approach focused on Relapse Prevention is useful. Marlatt and Gordon (1985) stressed that drug relapse was often due to ineffective coping with high-risk situations. Although individuals have their own unique list of high-risk situations, the situations are usually related to interpersonal conflicts, social pressure, conditioned cues, or negative emotional states. The therapeutic work of this approach is to develop effective coping responses as well as learn to handle a "lapse" (i.e., a single drink or drug administration) such that it does not degenerate into a "relapse" (i.e., problem use).

The final stages, late recovery and maintenance, emphasize personal growth in areas such as self-esteem, spirituality, intimacy, and work while individuals are maintaining a drug-free lifestyle. When there are deficits in these areas, insight-oriented therapy may be helpful. The reasons for continued inadequate functioning can be extremely complex and may involve unresolved issues from childhood. Kaufman and Redoux (1988) emphasized that uncovering core conflicts and confronting maladaptive defenses might elicit intense anxiety. Unless patients were in the late recovery stage, they might revert to their former maladaptive mode of coping—namely, using drugs.

The developmental model should be used as a guideline in understanding the recovery process rather than as a paradigm that is directly applicable to all patients. Additionally, there may be exceptions to when certain psychological interventions should be utilized. For example, an individual with major depression might not benefit from relapse-prevention techniques until the depression has been treated. *Pharmacologic Approaches.* Medications can serve as useful adjuncts in a comprehensive treatment plan. The appropriate use of these agents depends on the patient's medical and psychiatric status, prior treatment experience, and the clinical setting. Generally, the novel as well as established pharmacotherapies can be put into four classifications: (1) AGONISTS, (2) ANTAGONISTS, (3) antiwithdrawal agents, and (4) anticraving agents.

Agonists bind and activate receptors on cell membranes, and these operations then lead to a cascade of biologic activities. Drugs themselves are usually agonists and may generate strong physiologic responses (i.e., full agonists) or weak responses (i.e., partial agonists). The use of a specific agonist is limited to treatment of abuse of a drug from the same pharmacological class. Agonists are generally used for detoxification or for medication maintenance, and, when chosen for these purposes, they are likely to be well absorbed orally and slowly eliminated from the body. Slowly metabolized medications are less likely to produce a severe withdrawal syndrome but are more likely to produce a protracted, albeit less intense, one. Because agonists induce positive drug effects, they are well ac-

cepted. This, however, also means that they have the potential for abuse.

The most commonly used agonist for both maintenance and for opiate withdrawal is methadone, which itself is an opiate. BUPRENORPHINE, a partial opioid agonist, is being evaluated in the mid-1990s and may have less potential for abuse and be associated with fewer withdrawal symptoms than methadone when used for opiate detoxification. L-ALPHA-ACETYLMETHADOL (LAAM), also an opiate drug, has recently (1993) received FDA approval for use in treating opiate abuse. Unlike methadone, which must be taken daily, LAAM can be given three times a week, thereby decreasing the number of clinic visits for the patient as well as the risk of medication diversion. Few agonist drugs have been developed for other types of drug abuse, although NICOTINE, delivered transdermally, is being used with some success to treat tobacco dependence.

Antagonists prevent agonists (i.e., the abused drug) from producing their full physiologic response, either by blocking the receptor site or by disrupting the functioning of the receptor. Short-acting antagonists are most commonly used for treatment of acute intoxication or overdose and long-acting ones for rapid detoxification and relapse prevention. The benefits of antagonists are that they produce no euphorigenic effect, have no potential for abuse, and produce no withdrawal syndrome. Although generally only antagonists that block the specific receptor activated by the specific drug can be used for drug-abuse treatment, research is suggesting that the opiate antagonist NALTREXONE may play a role in diminishing alcohol drinking after a single drink.

Commonly used opioid antagonists include naloxone and naltrexone. Naloxone reverses the respiratory depression associated with opiate overdoses. Naltrexone is used after detoxification to maintain abstinence. Unfortunately, relatively few patients take an antagonist as prescribed because of its lack of pleasant effect, its lack of effect on withdrawal if the patient ceases taking the medication, and at times the persistence of craving (Kleber, 1989). Development of a monthly, long-acting injectable formulation may soon increase compliance when it reaches the market.

Antiwithdrawal medications are given to minimize the discomfort associated with detoxification from drugs that induce physiologic dependence. Agents used for opiate detoxification include methadone, CLONIDINE, and lofexidine; although effective for opiate detoxification, the latter two have not received FDA approval for this indication. The use of the dopamine agonists bromocriptine and AMANTADINE have been suggested for the manifestations of cocaine withdrawal, but their efficacy remains unclear. The most appropriate antiwithdrawal regimen for a particular clinical situation is not always the one chosen. This situation may be due to federal and state regulations, physician or patient bias, reimbursement issues, and the lack of available expertise within a community in the use of particular methods (Kleber, 1994).

The development of anticraving agents to treat drug dependence is a new treatment strategy. Earlier conceptualizations of craving focused on the physical aspects (i.e., the individual "craved" the drug because he or she was experiencing physical withdrawal symptoms). Thus the emphasis was placed on developing antiwithdrawal rather than anticraving drugs. During the last decade, as cocaine use soared, clinicians noted that craving could be psychologically based and be a significant relapse trigger (Gawin & Kleber, 1986). Much research was consequently done to find useful anticraving medications. Although desipramine remains promising, no medication has been unequivocally shown to be an effective anticraving agent for cocaine addiction.

ASSESSMENT OF TREATMENT OUTCOME

Although treatment for substance abuse can work, which treatment setting or modality will work best for each patient cannot invariably be predicted. Using a number of outcome studies, researchers at the Institute of Medicine (Gerstein & Harwood, 1990) reached several conclusions regarding the efficacy of various treatment modalities:

1. *Methadone Programs* Opiate-dependent individuals maintained on methadone exhibit less illicit drug use and other criminal behavior than do individuals discharged after being in the program for a period of time or not treated at all. For opiate-dependent individuals, there are higher retention rates in methadone programs

as compared to other programs, and patients tend to do better if they are stabilized at higher doses. Problems include continued use of nonopiate drugs, especially cocaine, and difficulty withdrawing.

2. *Therapeutic Communities* The length of stay within these communities, even for those who do not complete the program, is the best predictor of treatment outcome measured by drug use, criminal behavior, and social functioning. Graduates from therapeutic communities have superior outcomes when compared to dropouts. Dropout rates are unfortunately as high as 75 percent, although data suggest that even those who do not graduate derive some benefit if they have stayed for a period of time.
3. *Outpatient Nonmethadone* Programs As with individuals in therapeutic communities, individuals who graduate from these programs have better outcomes than those who drop out, and individuals who enter the programs have better outcomes than those who were contacted but did not begin the programs. These programs tended to treat less severely dependent patients.
4. *Chemical Dependency Programs* There were inadequate data to evaluate the efficacy of residential or inpatient programs (so-called 28-day MINNESOTA MODEL programs) designed to treat drug problems, and there were no data regarding whether hospital or free-standing programs were more effective.

Hubbard (1992) found that individuals referred from the criminal justice system performed as well in treatment as did other patients entering without such pressure, and that drug-abuse treatment provides a favorable cost-benefit ratio to society within one year of completion of treatment.

Recognizing that treatment success is multifactorial, investigators have sought comprehensive yet practical ways to characterize both patients and treatment programs. One instrument increasingly used to assess patient functioning is the ADDICTION SEVERITY INDEX (ASI) (McLellan et al., 1980). Using the ASI, the interviewer rates the severity of the patient's problem across six domains: alcohol and drug use, medical status, employment and support status, family and social relationships, legal status, psychiatric status. By giving the ASI at admission and repeating it over time, treatment success can be assessed in a standardized manner. Using this instrument, McLellan et al. (1984) found that opiate-addicted patients with severe psychological problems did worse over time when placed in a therapeutic community compared to those placed in methadone programs. As this study illustrates, it is critically important to assess "nondrug" variables when evaluating treatment response, and to carry out a comprehensive assessment prior to, during, and after treatment.

In the past few years, there has been greater emphasis on understanding how the specific aspects of treatment programs (e.g., therapeutic skills of the counselors, treatment modalities used, psychosocial services offered) influence treatment outcome. In regard to treatment services, McLellan et al. (1992) developed a rapid interview, the Treatment Services Review (TSR), which provides an evaluation of the amount and type of psychosocial services provided to patients during treatment. The investigators have suggested that this type of review might be useful when comparing different programs or for determining if the needs of individual patients were met during treatment. A recent study by McLellan et al. (1993) found that methadone-maintained patients who received enhanced psychosocial services did significantly better than those who received standard or minimal services.

No single study, no matter how comprehensive, can address all of the factors that influence treatment outcome. Instead, studies will need to focus on specific subpopulations of patients when comparing various treatment interventions as well as the impact on treatment of factors often overlooked (e.g., the patient's stage of recovery and the extent of program hours).

RECOMMENDED TREATMENT POLICIES

Since many Americans are still in need of treatment for drug abuse problems, rational treatment policies need to be established on the basis of our current knowledge regarding the extent of the problem and what interventions work. Such policies should address the following issues (Kleber, 1993):

1. Available treatment needs to be expanded. Although there are approximately 6 million individuals in need of drug treatment, the current system can treat less than 2 million a year.

2. Patients need to have access to a wide variety of treatment modalities. Since no one treatment is suitable for all patients, a community with a diversity of treatment services can more likely offer appropriate interventions to its population.
3. For treatment improvement to occur, there must be more funds dedicated to research along with efficient dissemination of new technologies. Without new research, progress will not be achieved. Without training and education of staff regarding new research findings, treatment will not improve.
4. Pressure must be exerted to encourage drug-addicted individuals to enter treatment. As noted earlier, those who enter under pressure from the criminal justice system do as well as those entering voluntarily. The family, employer, or criminal justice system can all be instrumental in getting individuals to enter and remain in treatment. This pressure must be sustained since when it remits, the individual often drops out of treatment.
5. The treatment needs of special populations (e.g., prisoners, pregnant women, HIV-infected individuals) require greater attention. There are few programs designed to treat drug-addicted prisoners while they are incarcerated or newly released. For pregnant drug abusers to engage in treatment, programs need to be accessible, be affordable, include child care (for optimal results), and reflect a nonjudgmental view. For HIV-infected individuals, comprehensive medical care should be linked with the substance-abuse treatment, especially considering the rising incidence of tuberculosis in this group.
6. Rehabilitation and habilitation need to be integrated into substance-abuse treatment programs. Some drug-dependent individuals have the educational background or skills that allow them to gain employment once their drug problem has been treated. Others may require job-seeking skills, job training, or additional schooling prior to seeking employment. A goal of treatment needs to be integration into society, not simply cessation of drug use.

When examining the different modalities of treatment the question is not, "Does treatment work?" but rather, "What works best for a particular individual?" and "What can be done to engage drug abusers in appropriate, well-organized treatment systems?" If these issues are successfully addressed, treatment strategies can be designed for each patient and yet remain affordable. Millions spent on effective treatment will save billions spent elsewhere.

(SEE ALSO: *Abuse Liability of Drugs; Coerced Treatment for Substance Offenders; Comorbidity and Vulnerability; Research; Substance Abuse and AIDS; Treatment; Treatment in the Federal Prison System*)

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Acupuncture The art of acupuncture is an ancient and integral part of the armamentarium used in China for the treatment of medical problems. Acupuncture consists of the insertion of very fine needles into the skin at specific points intended, according to traditional Chinese medicine, to influence specific body functions or body parts. In the traditional Chinese view of the body, life energy, (*chi*), circulates through pathways; blockage of the pathways leads to deficiency of *chi*, or disease. The goal of the traditional acupuncturist is to open up the pathways and stimulate the movement of *chi*. The specific points for needle insertion are based on traditional anatomy maps that depict which pathways affect which body functions.

Following President Richard M. Nixon's historic trip to China in 1972, considerable public interest in acupuncture was generated when the media observed that acupuncture was not only effective in relieving pain, but could also be a substitute for general anesthesia. The following year, Dr. H. L. Wen, a neurosurgeon in Hong Kong, reported a serendipitous observation that acupuncture with electrical stimulation (AES) eliminated withdrawal symptoms in a narcotics addict on whom he had intended to perform brain surgery to treat drug addiction. The discovery occurred the day before the scheduled surgery while Dr. Wen was demonstrating to the patient that AES could relieve pain. Fifteen minutes after the AES had begun, the patient reported a significant reduction of his drug withdrawal symptoms, which disappeared altogether thirty minutes after AES was started. Dr. Wen followed this patient, noting that AES had to be administered every eight hours for the first three days, and gradually the intervals could be increased. Within a week there were no further signs or symptoms of withdrawal. This led Dr. Wen to conduct a study of AES in 40 narcotics addicts experiencing withdrawal. All but one (who re-

The use of acupuncture in addiction treatment is popular, despite the absence of clear evidence that it is an effective treatment for opiate or cocaine dependence. (© Roger Ressmeyer/CORBIS)

quired medication for severe pain and was dropped from the study) were successfully detoxified. It is noteworthy that Dr. Wen's initial observations occurred prior to the discovery, in 1975, of endogenous opioid substances in the brain (also called endorphins).

In a later study, in 1977, Dr. Wen noted that AES increased endorphin levels and relieved abstinence syndromes while simultaneously inhibiting the autonomic nervous system, primarily the parasympathetic nervous system. The findings by Dr. Wen and several other scientific groups that peripheral stimulation could release endogenous opioid substances in the central nervous system (CNS) gave scientific credibility to the possibility that this traditional Chinese therapy could help to deal with a contemporary problem. Chronic or repeated exposure to opioids leads to adaptive changes in the CNS; withdrawal symptoms occur when these drugs are abruptly discontinued. Since the administration of opioid drugs alleviates withdrawal, it was reasonable to believe that one's own endogenous opioids might do the same.

During the mid-1970s, the use of acupuncture became popular in the United States, despite the absence of the kind of rigorous clinical investigation typically required for new pharmacological treatments. There were probably a number of factors that contributed to its popularity. Because it involved no pharmacological agents, it was seen as being more compatible with the approach espoused by SELF-HELP groups, ranging from ALCOHOLICS ANONYMOUS (AA) to THERAPEUTIC COMMUNITIES. Also, acupuncture did not initially require medical personnel, so it was relatively inexpensive compared to either psychotherapy or pharmacotherapy. In addition, its popularity increased at a time when some people objected to using METHADONE for drug detoxification or for maintenance, on the grounds that such use made drug-dependent minority-group members dependent upon the medical establishment. A technique from a non-Western tradition seemed, therefore, to have special appeal for treatment programs that dealt predominantly with minorities.

One such program was the Division of Substance Abuse at Lincoln Hospital in the south Bronx, New York, under the leadership of Dr. Michael O. Smith. Smith was interested in alternatives to methadone for detoxification. Based on Wen's work, Smith first used electrical stimulation along with acupuncture, but he later discarded the use of electrical stimulation. Eventually, a standard protocol was developed which used four or five acupuncture points on each ear. By 1975, the use of acupuncture as a treatment for drug abuse was extended to alcohol patients, then later to cocaine and crack-cocaine patients.

In 1985 Smith founded the National Acupuncture Detoxification Association (NADA) at 3115 Broadway, #51, New York, New York 10027. By 1993, when the second international conference of NADA was held in Budapest, Hungary, there were participants from all over the world.

In the early 1990s, the use of acupuncture in addiction treatment had become popular with many people working in the criminal-justice system. Most of the funding for treatment programs using acupuncture at that time came initially from

the criminal-justice system, rather than from the federal and state agencies that usually fund drug treatment programs. Although the scientific community had been unable to show the efficacy of acupuncture in properly controlled clinical studies, this relatively inexpensive and easily expanded procedure became the mainstay of a number of "drug courts," where judges involved themselves directly in managing the treatment of drug offenders.

At many clinics in the United States, acupuncture treatment is now offered as part of a broad psychosocial program that has elements of self-help and TWELVE-STEP programs, plus traditional medicine and alternative medicine (some clinics, for example, use a "sleep mix" tea brewed from a variety of herbs).

As practiced in the United States, several technical procedures broadly described as acupuncture have been used. *Standard bilateral acupuncture* is the application of five needles to the concha and cartilage ridge of each ear at defined points (*shen men*, lung, sympathetic, kidney, and liver) determined from traditional Chinese anatomy maps. With *unilateral acupuncture*, the needles are applied to one ear. *Acupressure* involves applying pressure by hand or by an object to the same areas. *Electroacupuncture* applies low level electric current to needles placed at the traditional points. With *moxibustion*, herbs are burned near the needles to add heat; and with *neuroelectric stimulation*, low dose electrical current is passed through surface electrodes. Some practitioners advocate the use of surface electrodes and special currents, designating this approach *neuroelectrical therapy* (NET). There is no more evidence for the efficacy of added electrical current in the acupuncture treatment of drug and alcohol problems than there is for acupuncture itself.

Many acupuncture practitioners in the United States belong to and are accredited by the American Association of Acupuncture and Oriental Medicine (AAAOM), founded in 1981. Others may be accredited by the National Acupuncture and Oriental Medicine Alliance (NAOMA), founded in 1992, which accepts a broader range of training for purposes of certification than AAAOM.

In 1991, the NATIONAL INSTITUTE ON DRUG ABUSE (NIDA) sponsored a technical review of the current state of knowledge about the use of acupuncture in the treatment of alcoholism and other drug-dependence problems. One of the participants, Dr. George Ulett, noted that although there is some evidence that electrical stimulation through needles or electrodes placed at certain points on the body can release endogenous opioids and other neuropeptides in the central nervous system, there is little evidence that such release is caused by needles alone. He also asserted that the critical factor is the frequency characteristic of the current, not the specific placement site of needles or electrodes. This group of researchers concluded that part of the difficulty in deciding whether acupuncture is effective was the lack of standard terminology and standard methods. A number of procedures, all called acupuncture, were being applied to a variety of drug and alcohol problems, but in different ways, over varying periods of time, with results measured in differing ways. For example, different numbers of acupuncture needles could be used, at different sites, with or without electrical current. One study of acupuncture for alcohol detoxification, by Bullock and coworkers, which came closest to being scientifically valid, used appropriate controls (placement of needles in non-sites) and staff who were "blinded" as to which group was control and which was receiving acupuncture at specific body sites. This study found a far better outcome for patients in the specific body-site group than for controls—and that the difference persisted even when measured six months later. However, another research group using similar methodology could not replicate the findings and reported no difference between point-specific acupuncture, sham transdermal stimulation, or standard care (no acupuncture control).

Many practitioners who have used acupuncture, even those who are convinced of its efficacy, report that only a small proportion of people who start treatment actually complete the typical series of ten to twenty treatments. Those who have used the technique believe that the minimal amount of treatment required for benefit is at least one twenty-minute session per day of bilateral acupuncture for at least ten days. In general, among both opioid-dependent and cocaine-dependent patients, those with lighter habits seemed to fare best.

The NIDA technical review panel concluded that, at the time of the review (1991), there was no compelling evidence that acupuncture is an effective treatment for opiate or cocaine dependence. Nevertheless, they found no evidence that acupuncture is harmful.

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Approaches Based on Behavior Principles Behavioral treatments are based on a model of drug dependence wherein drug use is considered a learned behavior that is directly influenced by antecedent and consequent events associated with drug use. Within this framework, drug use is deemed the primary target of assessment and treatment. The treatments are generally directed toward a goal of complete abstinence from drug use when dealing with dependent individuals, but moderation is an acceptable goal when dealing with non-dependent individuals who engage in problematic use (e.g., drinking and driving). Many of the treatments also focus on the promotion of prosocial behaviors that are incompatible with continuing the lifestyle of a drug abuser.

Three well-known behavioral treatments are covered in this section (for more comprehensive reviews regarding behavioral treatments for alcohol dependence, illicit drug dependence, and nicotine dependence, see Hester & Miller, 1995; Stitzer & Higgins, 1995; U.S. Department of Health and Human Services, 1996, respectively). Each of these treatments has been demonstrated to be efficacious in controlled studies. Contingency management is another prominent behavioral treatment for drug dependence, but is covered in a separate section of this volume. Other important learning-based treatments, such as brief interventions, motivational interviewing, and relapse prevention therapy are covered in the Cognitive Behavioral Treatments section of this volume.

Behavioral Counseling/Skills Training. Behavioral counseling/skills training emphasizes environmental restructuring and the acquisition of specific skills deemed important to eliminating harmful drug use and avoiding relapse. Whether the treatment goal is abstinence or moderation of harmful use, patients learn how to identify environmental, social and interpersonal antecedents and consequences of their drug use. For example, if drug use or problematic use is more likely when patients are in a particular setting (e.g., bars) or the company of certain individuals (e.g., former high-school buddies), they are counseled to restructure their environment to avoid or minimize contact with those settings or people. Sometimes the goal might be to alter the setting in which the patient socializes with a particular individual (e.g., get together with a particular friend at a sporting event rather than a bar). Regarding consequences, the individual is counseled to make explicit the negative consequences of drug use and to identify healthy alternatives to the positive consequences derived from drug use and intoxication.

Patients often receive coping skills training in areas deemed important to discontinuing drug use and avoiding relapse. To combat the common problem of social pressure to use drugs, for example, patients are systematically instructed in drug-refusal skills through role-playing and other exercises. Other aspects of social skills training and problem solving are also commonly included in behavioral treatments for drug dependence (Monti et al., 1995). When moderation is the goal with problem drinkers, individuals are taught to monitor their drinking, set ingestion limits, and to use specific strategies to limit the amount consumed (e.g., do not drink alcoholic beverages to quench thirst, take small sips, alternate between alcoholic and nonalcoholic drinks) (Hester, 1995).

A relatively extensive scientific literature supports the efficacy of behavioral treatments for various forms of drug dependence and problematic use. For example, a series of clinical trials have demonstrated that social skills training is an efficacious adjunct treatment for alcohol dependence (Miller at al., 1995; Monti et al., 1995). Most of these studies have examined the effectiveness of social skills training as an adjunct to other treatments, and focused on assertiveness and related social skills. In a seminal study on this topic, for example, forty adults hospitalized for alcohol dependence were randomly assigned to either (1) an eight-session skills-training group focused on drinking-related problem-solving or (2) a control group in which similar topics were discussed but no specific training was provided. During a one-year follow-up period, the skills group compared to the control group reported an average of fourfold fewer drinks con-

sumed, sixfold fewer days drunk (eleven versus sixty-four days during the twelve-month follow-up), and a ninefold reduction in duration of drinking episodes (average of five days versus forty-four days).

Although the bulk of the evidence supporting the efficacy of social skills training and other coping skills training has been obtained with alcoholics and problem drinkers, evidence is also available supporting the efficacy of this approach with individuals who abuse or are dependent on illicit drugs like cocaine (Monti et al., 1997).

With regard to teaching non-dependent, problem drinkers to moderate their intake, a series of experimental studies reported over a ten-year period indicated that 20 to 70 percent of clinical samples can learn to drink moderately and that those effects can be sustained for up to two years (Hester, 1995).

Numerous reviews and meta-analyses support the efficacy of behavioral treatments for cessation of cigarette smoking (U.S. Department of Health and Human Services, 1996). The proportion of patients who successfully quit smoking at six- or twelve-month follow-ups generally increases as the intensity of the intervention increases, with 20 percent abstinence rates being common and 40 percent being reported in some early studies with intensive behavioral treatments. Combining behavioral therapy with pharmacological treatments (e.g., nicotine gum or patch) generally increases quit rates above either intervention alone (Hughes, 1995).

Behavioral Marital Therapy. Evidence from studies with alcohol-dependent individuals (O'Farrell, 1995) and with individuals dependent on illicit drugs (Fals-Stewart et al., 1996) indicates that involving spouses who are not themselves drug abusers in treatment and providing them with behavioral marital therapy can improve the quality of the relationship and drug-use outcomes. The evidence is more robust regarding improvements in marital satisfaction than reductions in drug use, but both have been documented in controlled studies. The rationales for involving spouses in treatment is that they may engage in behavior that initiates or reinforces drug use; they can acquire skills that promote abstinence or moderation; and spouses are an important potential source of alternative reinforcement when drug use ceases. Two aspects of behavioral marital therapy particularly merit mention. First, couples receive training in positive communication skills (how to constructively negotiate for changes in each other's behavior that will improve the quality of the relationship. Second, when treatment involves disulfiram therapy for alcohol dependence, spouses are taught how to effectively monitor compliance with the medication regimen (Azrin et al., 1982).

Multimodal Treatments. Treatment packages are sometimes implemented that utilize most of the adjunct behavioral treatments noted above as components in a more comprehensive treatment effort, usually for severely dependent individuals. The Community Reinforcement Approach (CRA) is perhaps the best example of a multimodal-behavioral treatment. CRA includes various forms of social skills and problem-solving training, vocational counseling, marital therapy, social/recreational counseling, and socially monitored disulfiram therapy (see Meyers & Smith, 1995).

In the seminal study examining the efficacy of the CRA treatment for alcohol dependence, sixteen males who had been admitted to a state hospital for alcoholism were divided into matched pairs and randomly assigned to receive CRA plus standard hospital care or standard care alone (Hunt & Azrin, 1973). Following discharge from the hospital, CRA patients received a tapered schedule of counseling sessions across several months. During a six-month follow-up period, patients who received CRA reported approximately six- to fourteen-fold less time drinking, unemployed, away from their families, or institutionalized compared to control patients. Several of the CRA elements noted above were added in subsequent studies conducted by this same group of investigators as the treatment moved from being an adjunct to inpatient treatment to a stand-alone, comprehensive treatment that could be delivered in outpatient settings. Findings from these later studies were at least as impressive as in the seminal study (see Meyers & Smith, 1995). Other groups have effectively extended CRA to the treatment of opiate (Abbott et al., 1998; Bickel et al., 1997) and cocaine dependence (Higgins et al., 1993, 2000). A contingency management element was added in the extension of CRA to the treatment of cocaine dependence (see Budney & Higgins, 1998) as well as one of the studies on opiate dependence (Bickel et al, 1997), and is discussed in the section of this volume on contingency management.

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Aversion Therapy For many years, attempts have been made to condition alcoholics to dislike alcohol. For example, alcoholics are asked to taste or smell alcohol just before a preadministered drug makes them nauseated. Repeated pairing of alcohol and nausea results in a conditioned response—after a while, alcohol alone makes them nauseated. Thereafter, it is hoped, the smell or taste of alcohol will cause nausea and discourage drinking.

Instead of pairing alcohol with nausea, other therapists have associated it with pain, shocking patients just after they drink, or they have associated it with panic from not being able to breathe by giving them a drug that causes very brief respiratory paralysis. Others have trained patients to imagine unpleasant effects from drinking, hoping to set up a conditioned response without causing so much physical distress.

Does it work? Some degree of conditioning is usually established, but it is uncertain how long the conditioning lasts. The largest study that involved conditioning alcoholics was conducted many years ago in Seattle, Washington (Lemere & Voegtlin, 1940). More than 34,000 patients conditioned to feel nauseated when exposed to alcohol were studied ten to fifteen years after treatment. Sixty-six percent were abstinent, an impressive recovery rate compared to other treatments. The patients who did best had had booster sessions—that is, they had come back to the clinic after the initial treatment to repeat the conditioning procedure. Of those who attended booster sessions, 90 percent were abstinent. Based on this study, the nausea treatment for alcoholism would seem an outstanding success. Why hasn't it been universally accepted?

One reason is that the results can be attributed to factors other than the conditioning. The patients in the study were a special group. Generally, they were well educated, had jobs, and were well off financially. They may not have received the treatment otherwise, since the clinic where they were treated was private and expensive. Studies of alcoholics have often shown that certain subject characteristics are more predictive of successful treatment outcome than the type of treatment administered. These factors include job stability, living with a relative, absence of a criminal record, and living in a rural community. In the Seattle study there was no control group that did not receive conditioning therapy. It is possible that this select group of patients, many having characteristics that favor a good outcome, would have done as well without conditioning.

Furthermore, in conditioning treatments, motivation is important. Treatment is voluntary and involves acute physical discomfort; presumably few would consent to undergo the therapy if they were not strongly motivated to stop drinking. The Seattle study makes this point graphically clear. Those who came back for booster sessions did better than those who didn't, but another group did better still: those who *wanted* to come back but couldn't because they lived too far from the hospital. All of these people remained abstinent.

For many years, chemically induced aversive conditioning of alcoholics was virtually ignored in the literature. Then, in 1990, Smith and Frawley published an outcome study of patients who received aversion therapy as part of their inpatient treatment. From a randomly selected sample of 200 patients, 80 percent were located and interviewed by telephone. Between thirteen and twenty-five months had passed since their discharges from the hospital. The overall abstinence rate for the first twelve months was 71 percent; it was 65 percent for the total period.

Follow-up studies of alcoholism treatment rarely report abstinence rates this high. How should these be interpreted?

As in the original Seattle study, in the Smith and Frawley study, the patients, by and large, had good prognostic features. At the time of admission, more than 50 percent were married and had some college education. Nearly 80 percent were employed. They could afford a private hospital. In short, with characteristics that favor a good outcome, they might have done as well without conditioning. Moreover, the inpatient program involved more than aversive conditioning. It included many ingredients found in other treatment programs, including counseling, a family program and aftercare plan, and ALCOHOLICS ANONYMOUS.

One finding in this report was similar to that of the original study—booster sessions are important. One month and three months after discharge, the patients were asked to return for reinforcement treatments. Just as in the original studies, those who returned for the booster sessions had a particularly good outcome. In fact, the most powerful predictor of abstinence was the number of reinforcement treatments utilized by each patient. Those taking two reinforcement treatments had a twelve-month abstinence rate of 70 percent; those who took only one had a 44 percent rate; and those who had no reinforcement had only a 27 percent rate. Seven percent took *more* than two reinforcement treatments and had a phenomenal twelve-month abstinence rate of 92 percent.

The importance of reinforcement sessions may reflect motivation on the part of the patient, actual Pavlovian conditioning, or both. The paper does

not tell whether the patients developed a true conditioned response to alcohol at any time. Information about this would help separate nonspecific motivational factors from actual conditioning.

The study lacked a control group. This was remedied in a report (Smith, Frawley, & Polissar, 1991) that compared 249 alcoholic inpatients who received aversion therapy with patients from a national treatment registry who did not receive aversion therapy. The patients treated with aversion therapy had significantly higher abstinence rates at six and twelve months, suggesting that motivation and good prognostic features may not completely explain the success of this still rather unpopular treatment.

Frawley and Smith (1992) have also reported remarkably high abstinence rates from cocaine (current abstinence of at least six months, 68 percent) among a similar group of patients, with good prognostic features, treated with aversion therapy and follow-up at an average of fifteen months after treatment. Again there was no control group.

Aversion treatment for cigarette smoking has been studied by using appropriate controls. The technique involves encouraging the smoker to keep inhaling at rapid intervals over a period of five to ten minutes until he or she becomes sick, presumably because the nicotine levels exceed the smoker's tolerance levels. This approach has consistently produced higher levels of abstinence from smoking than have control groups.

(SEE ALSO: *Calcium Carbimide; Disulfiram*)

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Cognitive Therapy Cognitive treatment is based on the assumption that the way one thinks is a primary determinant of feelings and behavior. Developed from Beck's research (Beck et al., 1979, 1993), cognitive treatment is approached as a collaborative effort between the client and therapist to examine the client's errors and distortions in thinking that contribute to problematic behavior. This examination is fostered through a combination of verbal techniques and behavioral experiments to test the underlying assumptions the client holds about the problematic behavior.

Cognitive treatment in the substance-abuse field was a direct extension of Beck's work. Beck's catalog of distorted thoughts examined in depression were found to be applicable to cognitive distortions and errors that accompany addictive disorders. Various cognitive treatments for substance abuse focus on these distortions and vary primarily in the techniques used to change these thought processes.

In RELAPSE PREVENTION (Marlatt & Gordon, 1985), cognitive distortions are viewed as instrumental in the process that leads to relapse. By helping the client thoroughly examine the thoughts that accompany substance use, therapy can reduce the likelihood of a lapse (single use), as well as help prevent a lapse from becoming a relapse (return to uncontrolled use). This is accomplished by examining the following cognitive errors:

1. Overgeneralizing—this is one of the most frequently occurring cognitive errors that helps a single lapse become a full-blown relapse. By viewing the single use as a sign of total relapse, the client overgeneralizes the single use of a substance as a symptom of total failure, thereby allowing for increasing use over time and in a variety of situations. This is sometimes referred to as the ABSTINENCE VIOLATION EFFECT (AVE).
2. Selective abstraction—by excessively focusing on the immediate lapse, with an accompanying neglect of all past accomplishments and learning, the client interprets a single slip as equivalent to total failure. The individual measures progress almost exclusively in terms of errors and weaknesses.

3. Excessive responsibility—by attributing the cause of a lapse to personal, internal weaknesses or lack of willpower, the client assumes total responsibility for the slip, which in turn makes reassuming control more difficult than when environmental factors are considered partially responsible for the slip.
4. Assuming temporal causality—here, the client views a slip as the first of many to come, thereby dooming all future attempts at self-control.
5. Self-reference—when the client thinks that a lapse becomes the focus of everyone else's attention, believing that others will attribute blame for the event to the client, this adds to feelings of guilt and shame that may already be present within the person.
6. Catastrophizing—the client believes the worst possible outcome will occur from a single use of the substance instead of thinking about how to cope successfully with the initial lapse.
7. Dichotomous thinking—by viewing events in "black and white," clients view their addictive behavior exclusively in terms of abstinence or relapse and leave no logical room for "gray" areas, where they can get back on track once a slip has occurred.
8. Absolute willpower breakdown—here, the client assumes that once willpower has failed, loss of control is inevitable, never to be regained.
9. Body over mind—the cognitive error here is assuming that once a single lapse has occurred, the physiological process of addiction has exclusive control over subsequent behavior, making continued use inevitable.

These errors in thinking are targeted for change in relapse prevention by helping the client learn how to reattribute the cause of a lapse from internal, stable, personal causes to mistakes or errors in the learning process. To facilitate the client's sense of personal control, lapses are viewed as opportunities for corrective learning, instead of indications of total failure. Congruent with the research in the area (Shiffman, 1991), the therapist presents a lapse as a frequently occurring event in the journey toward recovery. The therapist therefore encourages the client to examine the thoughts and expectancies that surround the lapse closely, with the aim of learning alternative coping skills for similar situations that may arise in the future. By reframing a lapse as a learning opportunity, the client is encouraged to view the event as a chance to hone the skills required for abstinence, thereby countering the cognitive errors of selective abstraction.

To intervene with the errors of overgeneralization and temporal causality, the client is taught to view a lapse as a specific, unique event in time and space, instead of as a symptom with greater significance attached to it (e.g., the beginning of the inevitable end). The errors of self-reference and willpower breakdown can be countered by teaching the client to reattribute a lapse to external, specific, and controllable factors. By examining the difficulty of the high-risk situation, the appropriateness of the coping response employed, and any motivational deficits (fatigue or excessive stress), the client can maintain a sense of control over the event and the process of recovery.

Each of these techniques is aimed at conveying the idea that abstinence is the result of a learning process, requiring an acquisition of skills similar to many other skills one learns. This general metaphor can help the client reverse catastrophizing, by reframing a relapse as a "prolapse," as a fall forward rather than backward. This view, combined with viewing a lapse as a unique event in time, helps the client maintain a sense of personal control, since abstinence or control is framed as just a moment away if use is discontinued.

Several skills are taught to the client in relapse prevention to facilitate these cognitive changes and prevent future lapses. Identifying specific sources of stress that contribute to urges, cravings, or lapses helps isolate the event in time as well as identify other distortions that may be present. For example, clients may identify discussing money with one's spouse as the high-risk situation that preceded a lapse. While discussing the lapse with a therapist, clients can learn to anticipate that discussing money in the marriage may trigger an urge or craving to drink. Teaching clients to use visual imagery, such as viewing the urge as a wave that they can surf, can help manage the feeling that urges will continue to build until they must inevitably be given in to. Self-talk is encouraged if a client believes this will help gain a sense of personal control (such as reciting a phrase to oneself about the goal of abstinence or remembering who can be telephoned when an urge is experienced). In addition, clients are taught to be alert for "apparently irrelevant decisions," which can inadvertently lead to relapse. For example, an abstinent gambler may

decide to take a scenic drive through Reno, only to find a situation that would be extremely difficult for many to ignore, thus in this case causing a relapse.

Other theorists have developed treatments based exclusively on changing irrational thinking. Ellis and colleagues (1988) founded a self-help group network called RATIONAL RECOVERY (RR), based on the principles of rational emotive therapy. Developed as an alternative to the ALCOHOLICS ANONYMOUS network, RR focuses on "addictive thinking" and views abstinence as possible—purely as a result of changing these thought processes. This differs from the relapse prevention model described above, which in its entirety combines cognitive and behavioral techniques. Ellis's RR movement teaches addicts how to identify, their own faulty thinking through a self-help manual (Trimpey, 1989) and the attendance at support groups.

(SEE ALSO: *Alcoholism; Causes of Substance Abuse; Disease Concept of Alcoholism and Drug Abuse*)

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Contingency Management Contingency management (CM) is an intervention that promotes behavior change by providing positive reinforcement when treatment goals are achieved and withholding reinforcement or providing punitive consequences when undesirable behavior occurs. CM has been used effectively in the treatment of a wide variety of forms of drug dependence, including amphetamine (Boudin, 1972), alcohol (Miller, 1975; Petry et al., 2000), cocaine (Higgins et al., 1993, 2000), marijuana (Budney at al., in press), nicotine (Donatelle et al., 2000), and opiates (Hall, et al., 1979; Bickel et al., 1997).

Contingency management involves an agreement or contract that carefully stipulates the desired behavior change, the schedule and methods for monitoring progress, the consequences that will follow success or failure in making the behavior change, and the duration of the contract. Practical details on the development and implementation of CM interventions can be found in several sources (Budney & Higgins, 1998; Higgins & Silverman, 1999; Petry, 2000)

The most common use of CM with drug-dependent individuals is to reinforce abstinence from drug use. Numerous studies have demonstrated that providing incentives contingent on objective evidence of abstinence from recent drug use (e.g., negative urinalysis results) increases future abstinence (see Higgins & Silverman, 1999; Stitzer & Higgins, 1995). Although compelling evidence regarding the efficacy of CM has been available since the 1970s, interest in this treatment approach was bolstered substantially by successes achieved with CM in the treatment of cocaine dependence. In a seminal study on that topic, thirty-eight cocaine-dependent adults were randomly assigned to twenty-four weeks of behavior therapy including CM or to drug abuse counseling (Higgins et al., 1993). In the CM condition, vouchers redeemable for retail items were earned by submitting specimens that tested negative for cocaine use in urine toxicology testing. More than 50 percent of patients in the CM condition remained in treatment for the recommended twenty-four weeks and achieved several months of continuous cocaine abstinence while only 11 percent of patients in the comparison condition did so. Subsequent studies of CM in the treatment of cocaine dependence replicated those findings and also demonstrated benefits during the year after treatment ended (Higgins et al., 2000; Silverman et al., 1996). These positive results with CM were particularly encouraging because so few other treatment approaches have been shown to be efficacious with cocaine dependence.

Most typically, but not always, CM is used as part of a more comprehensive treatment plan. In-

deed, CM can be used to improve compliance with other treatment regimens. Early studies with alcoholics, for example, demonstrated that CM could be used to improve medication compliance among individuals receiving disulfiram (Antabuse) therapy (Liebson et al., 1978). More recent studies have demonstrated CM's efficacy in improving medication compliance among tuberculosis-exposed and HIV-infected drug abusers (Elk, 1999; Rosen et al., 2000). CM can also improve compliance with participation in therapy-related activities among opiate-dependent patients (Bickel et al., 1997; Iguchi et al., 1997). In these applications, patients earned vouchers by completing some minimum number of therapy-related activities weekly. The activities might include attending a job interview if the goal was gaining employment, or attending a self-help meeting if the goal was to increase contact with a social network to support sobriety. Vouchers were provided when patients submitted documentation verifying that they had completed a designated therapeutic activity. Completion of therapeutic activities was associated with greater drug abstinence.

CM is also proving to be capable of improving outcomes with important special populations of drug abusers. Improving adherence to medication regimens among those with infectious diseases was noted above. Another special population is the seriously mentally ill who are also drug-dependent. Results from several preliminary studies indicate that CM may be effective in reducing cigarette smoking (Roll et al., 1998), cocaine use (Shaner et al., 1997), and marijuana use (Sigmon et al., in press) among individuals with schizophrenia. CM is an integral component of a multielement treatment that is efficacious in the treatment of homeless crack and other drug abusers (Milby et al., 2000). Another special group for whom effective treatments are sorely needed is drug-dependent pregnant women. A voucher-based CM intervention has been demonstrated to significantly increase abstinence from cocaine and heroin use while simultaneously increasing vocational skills among pregnant women who were both drug dependent and chronically unemployed (Silverman et al., in press). In another effective CM intervention with pregnant women, vouchers delivered contingent on abstinence from cigarette smoking increased cessation rates during pregnancy and postpartum (Donatelle et al., 2000).

As illustrated in the preceding material, CM is effective in increasing drug abstinence and in improving compliance with treatment regimens for various types of drug dependence and populations. Positive outcomes have been achieved even with some of the most challenging and recalcitrant subgroups of drug abusers. A notable shortcoming associated with CM is a loss of treatment gains when the intervention is terminated. As noted above, beneficial carryover effects have been demonstrated through a year or more posttreatment, and the rates of relapse appear to be comparable to those observed among individuals treated with other interventions. Nevertheless, relapse is an important problem needing improvement. Systematic use of multimodel interventions designed to address the many changes likely to be necessary for longer-term success is one reasonable approach, as is the development of longer-term CM interventions that can be kept in place until the patient gains the requisite skills to sustain abstinence without CM support.

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Group and Family Therapy The illnesses of drug addiction and alcoholism are so severe that they pervade every aspect of an individual's existence. It is rare that so extensive an illness can be reversed by individual therapy alone. Thus therapists are espousing an integration of individual, TWELVE-STEP, group, and family treatment, with specific combinations of treatments tailored to each individual's needs.

Dealing with the family is one more involvement with the patient's ecosystem, which includes working with the treatment team, twelve-step groups, sponsors, employers, EAPs (EMPLOYEE ASSISTANCE PROGRAM counselors), managed-care workers, parole officers, and other members of the legal system. However, family work is most critical to the success of treatment.

Group therapy has frequently been designated as the treatment of choice for addicted patients. This article views group therapy as an essential component of the integrated, individualized approach to addicts and alcoholics.

FAMILY THERAPY

The family treatment of substance abuse begins with developing a system to achieve and maintain abstinence. This system, together with specific family therapeutic techniques and knowledge of patterns commonly seen in families with a substance-abusing member, provides a workable, therapeutic approach to substance abuse.

Family treatment of substance abuse must begin with an assessment of the extent of substance dependence as well as the difficulties it presents for the individual and the family. The quantification of substance-abuse history can take place with the entire family present; substance abusers often will be honest in this setting, and "confession" is a helpful way to begin communication. Moreover, other family members can often provide more accurate information than the substance abusers (also known as the identified patient, IP). However, some IPs will give an accurate history only when interviewed alone.

In taking a drug-abuse history, it is important to know current and past use of every type of abusable drug as well as of ALCOHOL: quantity, quality, duration, expense, how intake was supported and prevented, physical effects, tolerance, withdrawal, and medical complications. At times, other past and present substance abusers within the family are identified; their own use and its consequences should be quantified without putting the family on the defensive. It is also essential to document the family's patterns of reactivity to drug use and abuse. Previous attempts at abstinence and treatment are reviewed to determine components of success and failure. The specific method necessary to achieve abstinence can be decided only after the extent and nature of substance abuse are quantified.

Establishing a System to Achieve a Substance-Free State. It is critical first to establish a system for enabling the substance abuser to become drug-free, so that family therapy can be effective. The specific methods employed to achieve abstinence vary according to the extent of use, abuse, and dependence. Mild-to-moderate abuse in adolescents can often be controlled if both parents agree on clear limits and expectations, and how to enforce them. Older abusers may stop if they are aware of the medical or psychological consequences to themselves or the effects on their family.

If substance abuse is moderately severe or intermittent and without physical dependence, such as intermittent use of HALLUCINOGENS or weekend COCAINE abuse, the family is offered a variety of measures, such as regular attendance at ALCOHOLICS ANONYMOUS (AA), NARCOTICS ANONYMOUS (NA), or COCAINE ANONYMOUS (CA) for the IP and Al-Anon or Naranon for family members.

If these methods fail, short-term hospitalization or treatment in an intensive outpatient program (20 hours or more per week) may be necessary to establish a substance-free state and to begin effective treatment even with nondependent patients. In more severe cases of drug abuse and dependence, more aggressive methods are necessary to establish a substance-free state.

Family Education. A substantial amount of family education is generally very helpful in the early stages of the family's involvement in therapy. In many inpatient addiction treatment programs, the family spends several days or more receiving appropriate education. If this is not available, the therapist should include this education process in early sessions.

Some of the issues covered by this educational emphasis are: (1) the physiological and psychological effects of drugs and alcohol; (2) the disease concept; (3) cross addiction (which helps families learn that a recovering cocaine addict should not drink or vice versa); (4) common family systems—emphasizing the family's roles in addiction and recovery, including enabling, scapegoating, and CODEPENDENCY; (5) the phases of treatment, with an emphasis on the deceptiveness of the "honeymoon" period in early recovery; and (6) the importance of twelve-step family support groups (AL-ANON, ALATEEN).

Working with Families with Continued Drug Abuse. The family therapist is in a unique position with regard to continued substance abuse and other manifestations of the IP's resistance to treatment, including total nonparticipation. The family therapist still has a workable and highly motivated patient(s): the family. One technique that can be used with an absent or highly resistant patient is the intervention, which was developed for use with alcoholics but can be readily adapted to work with drug abusers, particularly those who are middle class, involved with their nuclear families, and employed.

In this technique, the family (excluding the abuser) and significant network members (e.g., employer, fellow employees, friends, and neighbors) are coached to confront the subtance abuser with concern, but without hostility, about the destructiveness of his or her drug abuse and behavior. They agree in advance about what treatment is necessary and then insist on it. As many family members as possible should be included, because the breakthrough for acceptance of treatment may come from an apparently uninvolved family member, such as a grandchild or cousin. The involvement of the employer is crucial, and in some cases may be sufficient in and of itself to motivate the drug abuser to seek treatment. The employer who clearly makes treatment a condition of continued employment, who supports time off for treatment, and who guarantees a job on completion of the initial treatment course is a very valuable ally. The employer's model is also a very helpful one for the family, who need to be able to say "We love you, and because we love you, we will not continue to live with you if you continue to abuse drugs and alcohol. If you accept the treatment being offered to you and continue to stay off drugs, we will renew our lifetime commitment to you."

If substance abusers do not meet the above criteria for an intervention or if the intervention has failed, we are left with the problems of dealing with a substance-abusing family. Berenson (1976) offers a workable, three-step therapeutic strategy for dealing with the spouses or other family members of individuals who continue to abuse substances or who are substance dependent. Step one is to calm down the family by explaining problems, solutions, and coping mechanisms. Step two is to create an external support network for family members so that the emotional intensity is not all in the relationship with the substance abuser or redirected to the therapist. There are two types of support systems available to these spouses. One is a self-help group on the Al-Anon, Naranon, or Coanon model; the other is a significant others (SO) group led by a trained therapist. In the former, the group and sponsor provide emotional support, reinforce detachment, and help calm the family. An SO group may provide more insight and less support for remaining with a substance-abusing spouse.

Step three involves giving the client three choices: (1) keep doing exactly what you are doing; (2) detach or emotionally distance yourself from the drug abuser; or (3) separate or physically distance yourself. When the client does not change, it is labeled an overt choice 1. When a client does not choose 2 or 3, the therapist can point out that he or she is in effect choosing not to change. If not changing becomes a choice, then the SO can be helped to choose to make a change. In choice 2, SOs are helped to avoid overreacting emotionally to drug abuse and related behavior, and they are taught strategies for emotional detachment. Leaving, choice 3, is often difficult when the family is emotionally or financially dependent on the substance abuser.

Each of these choices seems impossible to carry out at first. The problem of choosing may be resolved by experiencing the helplessness and powerlessness in pursuing each choice.

As part of the initial contract with a family, it is suggested that the abuser's partner continue individual treatment, Al-Anon, Coanon, or an SO group even if the abuser drops out. Other family members are also encouraged to continue in family therapy and support groups. It should be reemphasized that whenever therapy is maintained with a family in which serious drug abuse continues, the therapist has the responsibility of not maintaining the illusion that the family is resolving problems, when in fact they are really reinforcing them. Even when the substance abuser does not participate in treatment, however, therapy may be quite helpful to the rest of the family.

The concept of the family as a multigenerational system necessitates that the entire family be involved in treatment. The family members for optimum treatment consist of the entire household and any relatives who maintain regular (approximately weekly) contact with the family. In addition, relatively emancipated family members who have less than weekly contact may be very helpful to these families.

The utilization of a multigenerational approach involving grandparents, parents, spouse, and children at the beginning, as well as certain key points throughout, family therapy is advised. However, the key unit with substance abusers younger than about age 24 is the IP with siblings and parents. The critical unit with married substance abusers older than 24 is the IP and spouse. However, the more dependent the IP is on the parents, the more critical is family work with these parents. The majority of sessions should be held with these family

units; the participation of other family members is essential to more thorough understanding and permanent change in the family.

Family therapy limited to any dyad is most difficult. The mother-addicted-son dyad is almost impossible to treat as a sole entity; some other significant person, such as a lover, grandparent, aunt, or uncle should be brought in if treatment is to succeed. If there is absolutely no one else available from the natural family network, then surrogate family members in multiple-family therapy groups can provide support and leverage to facilitate restructuring maneuvers.

AN INTEGRATED APPROACH TO A WORKABLE SYSTEM OF FAMILY TREATMENT

Family Diagnosis. Accurate diagnosis is as important a cornerstone of family therapy as it is in individual therapy. Family diagnosis looks at family interaction and communication patterns and relationships. In assessing a family, it is helpful to construct a map of the basic alliances and roles, as well as to examine the family rules, boundaries, and adaptability.

Family Treatment Techniques. Each system of family therapy presently in use is briefly summarized below, with an emphasis on the application of these techniques to substance abusers. They are classified into four schools: structural-strategic, psychodynamic, Bowen's systems theory, and behavioral. Any of these types can be applied to substance abusers if their common family patterns are kept in mind and if a method to control substance abuse is implemented.

Structural-Strategic Therapy. These two types are combined because they were developed by many of the same practitioners, and shifts between the two are frequently made by the therapist, depending on the family's needs. The thrust of structural family therapy is to restructure the system by creating interactional change within the session. The therapist actively becomes a part of the family, yet retains sufficient autonomy to restructure it. The techniques of structural therapy have been described in detail by Kaufman (1985). They include the contract, joining, actualization, marking boundaries, assigning tasks, reframing, the paradox, balancing and unbalancing, and creating intensity.

According to strategic therapists, symptoms are maladaptive attempts to deal with difficulties, which develop a homeostatic life of their own and continue to regulate family transactions. The strategic therapist works to substitute new behavior patterns for the destructive repetitive cycles. The techniques used by strategic therapists include the following:

1. Using tasks with the therapist responsible for planning a strategy to solve the family's problems.
2. Putting the problem in solvable form.
3. Placing considerable emphasis on change outside the sessions.
4. Learning to take the path of least resistance, so that the family's existing behaviors are used positively.
5. Using paradox, including restraining change and exaggerating family roles.
6. Allowing the change to occur in stages; the family hierarchy may be shifted to a different, abnormal one before it is reorganized into a new functional hierarchy.
7. Using metaphorical directives in which the family members do not know they have received a directive.

Stanton et al. (1982) successfully utilized an integrated structural-strategic approach with heroin addicts on METHADONE MAINTENANCE treatment.

Psychodynamic Therapy. This approach has rarely been applied to substance abusers because they usually require a more active, limit-setting emphasis on the here and now than is generally associated with psychodynamic techniques. However, if certain basic limitations are kept in mind, psychodynamic principles can be extremely helpful in the family therapy of these patients.

There are two cornerstones for the implementation of psychodynamic techniques: the therapist's self-knowledge and a detailed history of the substance abuser's family.

Important elements of psychodynamic family therapy include the following:

countertransference—The therapist may have a countertransference problem toward the entire family or any individual member of the family, and may get into power struggles or overreact emotionally to af-

fect, content, or personality. The IP's dependency, relationship suction and repulsion, manipulativeness, denial, impulsivity, and family role abandonment may readily provoke countertransference reactions in the therapist. However, family therapists view their emotional reactions to families in a systems framework as well as a countertransference context. Thus they must be aware of how families will replay their problems in therapy by attempting to detour or triangulate their problems onto the therapist. The therapist must be particularly sensitive to the possibility of becoming an enabler who, like the family, protects or rejects the substance abuser.

the role of interpretation—Interpretations can be extremely helpful if they are made in a complementary way, without blaming, guilt induction, or dwelling on the hopelessness of longstanding, fixed patterns. Repetitive patterns and their maladpative aspects for each family member can be pointed out, and tasks can be given to help change these patterns. Some families need interpretations before they can fulfill tasks. An emphasis on mutual responsibility when making any interpretation is an example of a beneficial fusion of structural and psychodynamic therapy.

overcoming resistance—Resistance is defined as behaviors, feelings, patterns, or styles that prevent change. In substance-abusing families, key resistance behaviors that must be dealt with involve the failure to perform functions that enable the abuser to stay "clean."

Every substance-abusing family has characteristic patterns of resistant behavior, in addition to individual resistances. This family style may contribute significantly by resistance; some families may need to deny all conflict and emotion, and are almost totally unable to tolerate any displays of anger or sadness; others may overreact to the slightest disagreement. It is important to recognize, emphasize, and interpret the circumstances that arouse resistance patterns.

Bowen's Systems Family Therapy. In Bowen's (1974) approach, the cognitive is emphasized and the use of affect is minimized. Systems theory focuses on triangulation, which implies that whenever there is emotional distance or conflict between two individuals, tensions will be displaced onto a third party, issue, or substance. Drugs are frequently the subject of triangulation.

Behavioral Family Therapy. This approach is commonly used with substance-abusing ADOLESCENTS. Its popularity may be attributed to the fact that it can be elaborated in clear, easily learned steps.

Noel and McCrady (1984) developed seven steps in the therapy of alcoholic couples that can readily be applied to married adult drug abusers and their families:

1. Functional analysis. Families are taught to understand the interactions that maintain drug abuse.
2. Stimulus control. Drug use is viewed "as a habit triggered by certain antecedents and maintained by certain consequences." The family is taught to avoid or change these triggers.
3. Rearranging contingencies. The family is taught techniques to provide reinforcement for efforts at achieving a drug-free state by frequent reviewing of positive and negative consequences of drug use and self-contracting for goals and specific rewards for achieving these goals.
4. Cognitive restructuring. IPs are taught to modify self-derogatory, retaliatory, or guilt-related thoughts. They question the logic of these "irrational" thoughts and replace them with more "rational" ideation.
5. Planning alternatives to drug use. IPs are taught techniques for refusing drugs through role-playing and covert reinforcement.
6. Problem solving and assertion. The IP and family are helped to decide if a situation calls for an assertive response and then, through role-playing, to develop effective assertive techniques. IPs are to perform these techniques twice daily and to utilize them in situations that would have previously triggered the urge to use drugs.
7. Maintenance planning. The entire course of therapy is reviewed, and the new armamentarium of skills is emphasized. IPs are encouraged to practice these skills regularly as well as to reread handout materials that explain and reinforce these skills.

Families can also be taught through behavioral techniques to become aware of their nonverbal

communication, so as to make the nonverbal message concordant with the verbal and to learn to express interpersonal warmth nonverbally as well as verbally.

FAMILY READJUSTMENT AFTER CESSATION

Once the substance abuse has stopped, the family may enter a honeymoon phase in which major conflicts are denied. They may maintain a superficial harmony based on relief and suppression of negative feelings. When the drug-dependent person stops using drugs, however, other family problems may be uncovered, particularly in the parents' marriage or in other siblings. These problems, which were present all along but obscured by the IP's drug use, will be "resolved" by the IP's return to symptomatic behavior if they are not dealt with in family therapy. In the latter case, the family reunites around their problem person, according to their old, familiar pathological style.

Too many treatment programs in the substance-abuse field focus their efforts on brief, high-impact treatment, neglecting aftercare. Many of these programs include a brief, intensive family educational and therapeutic experience, but have even less focus on the family in aftercare than on the IP. These intensive, short-term programs have great impact on the family system, but only temporarily. The pull of the family homeostatic system will draw the IP and/or other family members back to symptomatic behavior. The family must be worked with for months, and often years, after substance abuse first abates if a drug-free state is to continue. In addition, ongoing family therapy is necessary for the emotional well-being of the IP and other family members.

GROUP THERAPY

Group therapy varies with each of the three phases in the psychotherapy of substance abusers: achieving abstinence, early SOBRIETY, and late sobriety (achieving intimacy).

Early Phase: Achieving Abstinence. In the first phase of psychotherapy, the type of group utilized will depend on the treatment setting: hospital, residential, intensive outpatient (also termed partial hospitalization), or limited outpatient.

In hospital settings, educational groups are an essential part of the early treatment process, and the subjects covered in these groups are quite similar to those in educational family groups (described in the first section of this article). The major difference of emphasis in patient educational groups is on the physiological aspects and risk factors of drugs and alcohol. Other important didactic groups cover in detail issues such as (1) ASSERTIVENESS TRAINING; (2) other compulsive behaviors, such as sexuality, eating, working, and GAMBLING; (3) RELAPSE PREVENTION; (4) the prolonged abstinence syndrome; (5) leisure skills; and (6) cross addiction. All educational groups include appropriate coping strategies, some of which are developed from the experiences of recovering members.

One advantage of 28-day residential programs (now more often 7 to 21 days, followed by an intensive 6-hours-a-day outpatient program) is that group therapy can be started immediately after drinking or drug use stops. In the first few sober days, the addict or alcoholic is so needy that his/her resistance to groups is low. At this stage, the therapist and the group should show the substance abuser how to borrow the confidence that life without alcohol or drugs is possible and better than life with it. This hope is best offered by a therapist or cotherapist who is a recovering substance abuser with solid sobriety. Therapeutic groups in these settings will also deal with appropriate expressions of feelings, relationships with significant others, childhood molestation and abuse, building self-esteem, and development of strategies for self-care.

A critical aspect of early group therapy is for the patient to experience the sharing of a group of individuals struggling against their addiction. This helps to overcome the feelings of isolation and shame that are so common in these patients. The formation of a helping, sober peer group that provides support for a lifetime, in and out of twelve-step groups, is very helpful and dramatic when it occurs.

In outpatient programs there is less of an opportunity to perform uncovering therapy in the early phases because there is less protection and less of a holding environment than in residential settings.

Others, particularly Woody et al. (1986), have developed detailed group therapy techniques for methadone patients. Also, Brown and Yalom (1977) and Vanicelli (1992), with alcoholics, and Khantzian et al. (1990), with cocaine addicts, have

adapted psychodynamic techniques for group work.

Ex-addicts and recovering alcoholics are valuable as cotherapists, or even as primary or sole therapist, particularly in the early stages of groups. Commonality of experience with the client, by itself, does not qualify an individual to be a therapist. Recovering persons should have at least two years of sobriety before they are permitted to function as group therapists. The techniques that help ex-addicts become experienced therapists are best learned gradually and under close supervision, preferably by experienced paraprofessionals and professionals.

Also helpful in cotherapy is male-female pairing, which provides a balance of male and female role models and transference.

During the early sessions of group therapy with substance abusers, the focus is on the shared problem of drinking or drug use, and its meaning to each individual. The therapist should be more active in this phase, which should be instructional and informative as well as therapeutic.

Alcoholics tend toward confessionals and monologues about prior drinking. These can be politely interrupted or minimized by a ground rule of "no drunkalogues." Romanticizing past use of drugs or alcohol is strongly discouraged.

Outpatient Groups. The desire to drink or use drugs and the fear of slipping are pervasive, early concerns in outpatient groups. The patient's attitude is one of resistance and caution, combined with fear of open exploration. Members are encouraged to participate in AA and other relevant twelve-step groups, yet the "high support, low conflict, inspirational style" of AA may inhibit attempts at interactional therapy. Therapists should not be overly protective and prematurely relieve the group's anxiety because this fosters denial of emotions. On the other hand, the members' recognition of emotions and responsibility must proceed slowly because both are particularly threatening to substance abusers. Patients are superficially friendly, but do not show real warmth or tenderness. AA-type hugs are an easy way to begin to show physical support. They are afraid to express anger or to assert themselves. However, sudden irritation, antipathy, and anger toward the leaders and other members inevitably begin to become more overt as the group progresses.

Gradually, tentative overtures of friendship and understanding become manifest. There may be a conspiracy of silence about material that members fear could cause discomfort or lead to drug use or drinking. The therapists can point out to the members that they choose to remain static and within comfortable defenses rather than expose themselves to the discomfort associated with change. Patients usually drop out early if they are still committed to using drugs or drinking. Other patients who drop out early do so because they grow increasingly alarmed as they become aware of the degree of discomfort that any significant change requires.

Middle Phase: Early Sobriety. In the middle phase of group therapy, the emphasis is quite similar to that of individual therapy. Therapists should continue to focus on cognitive behavioral techniques to maintain sobriety. Intensive affects are abreacted toward significant persons outside of the group but are minimized and modulated between group members. In this stage there evolves a beginning awareness of the role of personality and social interactions in the use of drugs and alcohol. Alcoholics are ambivalent about positive feedback. They beg for it, yet reject it when it is given. They repeatedly ask for physical reassurance, such as a warm hug, but may panic when they receive it because of fear of intimacy and a reexperiencing of their unmet past needs. There is a fear of success and a dread of competing in life as well as in the group. Success means destroying the other group members (siblings) and loss of therapist (parent).

Alcoholics are reluctant to explore fantasies because the thought makes them feel as guilty as the act. They view emotions as black or white. This makes them withhold critical comments because they fear their criticism will provoke upset and the resumption of drinking in other members. This withholding may be conscious or unconscious. Rage has been expressed either explosively or not at all. Its expression in the middle phase of group should be encouraged, but gradually and under slowly releasing controls.

The other crucial affect that must be dealt with is depression. There is an initial severe depression, which occurs immediately after detoxification. It appears to be severe but usually remits rapidly, leaving the substance abuser with a chronic, low-grade depression—frequently expressed by silence, lack of energy, and vegetative signs. These patients

should be drawn out slowly and patiently. Ultimately, they are encouraged to cry or mourn, and a distinction is made between helping them deal with despair as opposed to rushing to take it away from them.

The success of the middle phase of group therapy with substance abusers depends on the therapist's and the group's ability to relieve anxiety through support, insight, and the use of more adaptive, concrete ways of dealing with anxiety. Alcohol and drugs must become unacceptable solutions to anxiety. In this vein, it is important not to end a session with members in a state of grossly unresolved conflict. This can be avoided by closure when excessively troubling issues are raised. Closure can be achieved by the group's concrete suggestions for problem solution. When this is not possible, group support, including extragroup contact by members, can be offered. Brown and Yalom (1977) utilize a summary of the content of each group that is mailed to members between sessions and helps provide closure and synthesis.

Final Phase: Late Sobriety. In the final phase of therapy, substance abusers express and work through feelings, responsibility for behavior, interpersonal interactions, and the functions and secondary gain of drugs and alcohol. In this phase, reconstructive group techniques as practiced by well-trained professionals are extremely helpful and essential if significant shifts in ego strength are to be accomplished. Here, the substance abuser will become able to analyze defenses, resistance, and transference. The multiple transferences that develop in the group are recognized as "old tapes" that are not relevant to the present. Problems of sibling rivalry, competition with authority, and separation anxiety become manifest in the group, and their transference aspects are developed and interpreted. Conflicts are analyzed on both the intrapsychic and interpersonal levels. Ventilation and catharsis take place, and may be enhanced by group support. Excessive reliance on fantasy is abandoned.

Alcoholics who survive a high initial dropout rate stay in groups longer than neurotic patients, and thus a substantial number of middle-phase alcoholics will reach this final phase. By the closing phase, the alcoholic has accepted sobriety without resentment and works to free himself or herself from unnecessary neurotic and character problems. He or she has developed a healthy self-concept, combined with empathy for others, and has scaled down inordinate demands on others for superego reassurance. He or she has become effectively assertive rather than destructively aggressive and has developed a reasonable sense of values. More fulfilling relationships with spouse, children, and friends can be achieved.

When members leave the group, the decision to leave should be discussed for several weeks before a final date is set. This permits the group to mourn the lost member and for the member to mourn the group. This is true regardless of the stage of the group, but the most intense work is done in the later phases. In open-ended groups, the leadership qualities of the graduating member are taken over by others, who then may apply these qualities to life outside the group.

By the time substance abusers have reached this phase, they act like patients in highly functioning neurotic groups. Other forms of group treatment combine the principles of group and family work, such as multiple family group treatment and couples groups.

Multiple Family Group Treatment (MFGT). This is a technique that can be used in any treatment setting for substance abusers but is most successful in hospital and residential settings, where family members are usually more available. In a residential setting, the group may be composed of all of the families or separated into several groups of three or four closely matched families. Most MGFTs now include the entire community because this provides a sense of the entire patient group as a supportive family. In residential settings these groups are held weekly for two or three hours. In hospitals, a family week or weekend is often offered as an alternative or adjunct to a weekly group.

Couples Groups. There are two types of couples groups: one for the parents of young substance abusers and one for the significant other and the substance abuser.

Couples often have difficulty dealing with the role of their own issues in family or other couple therapy dysfunction when the children are present. This boundary is generally appropriate, and thus ongoing couples groups should be an integral part of any family-based treatment program.

When the presenting problem of substance abuse is resolved, content shifts to marital problems. It is often at this point that parents want to

leave the MFGT and attend a couples group. In a couples group, procedures are reversed. Couples should not speak about their children but, rather, focus on the relationship between themselves. If material is brought up about the children, it is allowed only if it is relevant to problems that the couples have.

Couples must support each other while learning the basic tools of communication. When one partner gives up substance misuse, the nonusing partner must adjust the way he or she relates to the formerly using partner. There are totally new expectations and demands. Sex may have been used for exploitation and pacification so often that both partners have given up hope of resuming sexual relations and have stopped serious efforts toward mutual satisfaction. In addition, drugs and alcohol may have physiologically diminished the sex drive. Sexual communication must be slowly redeveloped. Difficulties may arise because the recovering abuser has given up the most precious thing in his or her life (drugs or alcohol) and expects immediate rewards. The spouse has been "burned" too many times (and is unwilling to provide rewards when sobriety stabilizes the spouse) to trust one more time; at the same time the recovering abuser is asked to reevaluate expectations for trust.

Couples groups in an adult or an adolescent program provide a natural means for strengthening intimacy. Spouses are encouraged to attend Al-Anon, Naranon, Coanon, and Coda to help diminish their reactivity and enhance their coping and self-esteem.

Couples groups have been used even more widely with alcoholics than with drug abusers, and the techniques are similar to those described above. Spouses of alcoholics are encouraged to attend Al-Anon, which facilitates an attitude of loving detachment.

Many studies have demonstrated that spousal involvement facilitates the alcoholic's participation in treatment and aftercare. It also increases the incidence of sobriety and enhanced function after treatment. Further, the greater the involvement of the spouse in different group modalities (Al-Anon, spouse groups, etc.), the better the prognosis for treatment of the alcoholic.

(SEE ALSO: *Causes of Substance Abuse; Comorbidity and Vulnerability; Contingency Contracts; Families and Drug Use; Sobriety; Toughlove*)

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EDWARD KAUFMAN

Hypnosis Hypnosis is a normal state of attentive, focused concentration with a relative suspension of peripheral awareness, a shift in attention mechanisms in the direction of focus at the expense of the periphery. Being hypnotized is something like looking through a telephoto lens. What is seen, is seen in great detail, but at the expense of context. The use of hypnosis has been associated with inducing a state of relaxation and comfort, with enhanced ability to attend to a therapeutic task, with the capacity to reduce pain and anxiety, and with heightened control over somatic function. For these reasons, hypnosis has been used with some benefit as an adjunct to the treatment of certain kinds of DRUG and ALCOHOL ABUSE and ADDICTION.

Therapeutic approaches involving hypnosis include using it as a substitute for the pleasure-inducing substance, taking a few minutes to induce a self-hypnotic state of relaxation (for example, by imaging oneself floating in a bathtub or a lake, or visualizing pleasant surroundings on an imaginary screen). In this strategy the hypnosis is a safe substitute for the pleasure-inducing effects of the drug. A second approach involves ego-enhancing techniques, providing the subject with encouragement, picturing himself or herself living well without the substance and able to control the desire for it. A third approach involves instructing subjects to reduce or eliminate their craving for the drug. A fourth involves cognitive restructuring, diminishing the importance of the craving for the drug by focusing instead on a commitment to respect and protect the body by eliminating the damaging drug. One widely used technique for smoking control, for example, has people in hypnosis repeat to themselves three points: (1) For my body, smoking is a poison; (2) I need my body to live; (3) I owe my body respect and protection. This approach places an emphasis on a positive commitment to what the person is for, rather than paying attention to being against the drug, thereby keeping attention on protection rather than on abstinence.

Hypnosis has been most widely used in the treatment of NICOTINE dependence, and although the results vary, a number of large-scale studies indicate that even a single session of training in self-hypnosis can result in complete abstinence of six months or more by approximately one out of four smokers.

There are fewer systematic data regarding use of hypnosis with COCAINE, OPIATE, or alcohol addiction. The success of the approach is complicated by the fact that the acute effects of substance intoxication and/or the chronic effects on cognitive function of alcohol and other drug abuse hampers hypnotic responsiveness, thereby diminishing the potential of addicted individuals to enter this state and benefit from it. Nonetheless, there may be occasional individuals who are sufficiently hypnotizable and motivated to use this approach as an adjunct to other treatment, diminishing the dysphoria and discomfort that can accompany WITHDRAWAL and abstinence while enhancing and supporting their commitment to a behavior change. Hypnosis can be used by licensed and trained physicians, psychologists, dentists, and other healthcare professionals who have special training in its use. The treatment is employed in offices and clinics as well as in hospital settings. It should always be used as an adjunct to a broader treatment strategy.

Hypnosis is a naturally occurring mental state that can be tapped in a matter of seconds and mobilized as a means of enhancing control over behavior, as well as the effects of withdrawal and abstinence, in motivated patients supervised by appropriately trained professionals.

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DAVID SPIEGEL

Long-term Versus Brief For many medical and psychiatric disorders that, like substance use disorders, have a chronic course, longer-term treatments are usually found to be much more effective than short interventions. For example, most patients with disorders such as hypertension, elevated cholesterol, diabetes, or schizophrenia have the best clinical course if they maintain lifestyle modifications and remain on their medications for extended periods of time. One would therefore think that individuals with substance use disorders who seek treatment would have better outcomes if they received longer, as opposed to shorter, episodes of care. However, research findings in the addictions have indicated that the relationship between length of treatment and outcome is not particularly straightforward.

There is considerable evidence that patients who stay in treatment longer have better outcomes. That is, when patients with similar demographic characteristics and pretreatment substance-use severity all enter the same treatment program, those who stay in treatment longer will on average have better treatment outcomes that those who leave early. The dividing line that predicts good versus poor outcome has frequently been retention for at least 90 days in treatment. However, it is not clear how much the better outcomes should be attributed to longer stays in treatment or to individual characteristics such as motivation and initial success in treatment. The most direct way to untangle treatment from motivation effects is to conduct studies in which patients are randomized to different lengths or intensities of treatment, and their outcomes examined over time. Studies of this sort have produced very little evidence to indicate that longer or more intense treatments produce better substance-abuse outcomes than shorter or less intense treatments. For example, a recent random assignment study compared 6- and 12-month therapeutic community programs, and 3- and 6-month residential programs with a relapse prevention focus. In both cases, the long and short versions of the same program did not differ in rates or patterns of drug use during six-month posttreatment followup periods. This suggests that the relationship between longer treatments and better outcomes is probably more a function of motivation and other patient characteristics than duration of treatment received.

However, it should also be stressed that many substance abuse treatment programs feature a continuum of care, in which patients spend a certain amount of time in an initial higher intensity treatment and then "step down" to a lower intensity level of care, such as *aftercare*. Perhaps participation in and completion of aftercare following initial treatment has greater prognostic significance than the duration of a single level of care? Surprisingly, research suggests it does not. In the majority of the relatively few studies that have examined this issue, patients who were randomly assigned to active aftercare treatments did not have better substance use outcomes than those who were randomized to either no aftercare or minimal aftercare conditions.

Is it therefore the case that duration of substance use treatment, whether in one level of care or a continuum of care, is not related to substance use outcome? Despite the results from randomized studies described here, duration might still be of some importance. For example, monitoring substance abusers with low-cost, low-intensity interventions over long periods of time and arranging for more intensive treatments if they appear to have resumed use or be at risk might produce better outcomes than simply discharging patients following an initial episode of care and maintaining no contact after that. However, this approach has yet to be evaluated in controlled research studies.

Although the research literature does not strongly support the use of longer-term treatment interventions, there is consensus among clinicians and clinical researchers that sustained recoveries from substance use disorders generally require ongoing efforts by those who have these disorders. Some of the behaviors that have been associated with good long-term outcomes include regular attendance at self-help groups such as Alcoholics Anonymous, treatment for family or marital problems, employment, involvement with religion, and commitment to new interests or hobbies. These findings are consistent with the notion that formal treatment, whether of short or long duration, is useful for beginning a process of change that must be sustained over long periods of time in order to be successful and that ultimately involves many areas of functioning.

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James R. McKay

Minnesota Model Origins of the Minnesota Model of drug abuse treatment are found in three independent Minnesota treatment programs: Pioneer House in 1948, Hazelden in 1949, and Wilmar State Hospital in 1950. The Hazelden Clinics are still in existence and are located in Minnesota and Florida. The original treatment programs recognized Alcoholics Anonymous (AA) as having success in bringing about recovery from Alcoholism. Unique to this early stage of the Minnesota Model was the blending of professional behavioral science understandings with AA's principles. Important in the development of the Minnesota Model is the way treatment procedures emerged from listening to alcoholics, from trial and error, from acknowledgment of the mutual help approach of AA, and from the use of elementary assumptions rather than either a well-developed theoretical position or a generally accepted therapeutic protocol. In many ways, the Minnesota Model may be seen as having come about in a grassroots, pragmatic manner.

Because of its evolutionary, noncentralized development, the Minnesota Model is not a standardized set of procedures but an approach organized around a shared set of assumptions. These assumptions have been articulated by Dan Anderson, the former president of Hazelden Foundation and one of the early professionals working with the Minnesota Model at Wilmar State Hospital. They are the following: (1) Alcoholism exists in a consolidation of symptoms; (2) alcoholism is an illness characterized by an inability to determine time, frequency, or quantity of consumption; (3) alcoholism is nonvolitional—alcoholics should not be blamed for their inability to drink ethanol (alcohol); (4) alcoholism is a physical, psychological, social, and spiritual illness; and (5) alcoholism is a chronic primary illness—meaning, that once manifest, a return to nonproblem drinking is not possible. Although these assumptions are phrased as pertaining to alcoholism, early experience with the Minnesota Model demonstrated that drug abuse other than alcoholism can also be understood and treated within these assumptions. *Chemical dependency* is the term generally used by clients and treatment providers when referring to substance abuse. The Minnesota Model provides treatment for chemical dependency—for both alcohol and other drugs.

A twenty-four to twenty-eight day inpatient treatment stay, or approximately eighty-five hours in outpatient rehabilitation, characterizes the Minnesota Model treatment. Inpatient treatment may occur in hospital settings or free-standing facilities and may be run by for-profit or nonprofit organizations. Different treatment settings have different mixes of staff positions, but the multidisciplinary team of medical and psychological professionals plus clergy and focal counselors are frequently found—either in a close interacting network or a more diffuse working arrangement.

Primary focal counselors have either received specific training in the Minnesota Model approach to treatment or have learned their counseling skills in an apprenticelike placement. Most counselors are neither mental-health-degreed professionals nor holders of medically related degrees, but they are commonly working on their own twelve-step programs because of life experience with chemical dependency or other addictions. As in AA, this shared personal experience of both clients and counselors is important for the client/counselor relationship and the behavior modeling the counselor provides for the client.

Minnesota Model treatment programs vary in the centrality of counseling staff and the programmed autonomy of the treatment experience. Some treatment programs have the counselor facilitating the majority of the groups and visibly directing the treatment experience. Other programs have the treatment groups carrying out the treatment experience where the activity follows a prescribed format, but the group members are the visible actors while the counseling staff maintains a low profile as they seek to empower clients to acquire the insights and resources necessary for their recovery. Treatment also varies in the amount of confrontation, the presence of a family program requirement, the extent of assigned reading, the detail of client record documentation, and other attributes.

What Minnesota Model treatment has without exception is the use of AA principles and understandings (steps and traditions) as primary adjuncts in the treatment experience. Clients are provided with the AA "Big Book" (*Alcoholics Anonymous*) and *The Twelve Steps and Twelve Traditions*. Both of these books are required reading. Spirituality is emphasized as important to recovery, which is consistent with the AA understanding. AA group meetings occur in the schedule of rehabilitation activities, and clients may visit a community AA meeting as part of their treatment experience. Clients will work on AA steps during their treatment experience; some programs focus on the first five steps while others emphasize all twelve steps.

Treatment it not just an intensive exposure to AA. It motivates treatment participants to develop mutual trust and to share and be open about how the use of chemicals has come to control their lives. Clients are told that they have the disease of chemical dependency. Their behavior has been directed by the disease, but they have been unable to see the reality of their behavior and the consequences because of the disease characteristic of denial. Treatment plans are individualized based on assessments by the multidisciplinary staff. Generally, the first goal of treatment is to break the client's denial and the second goal is for the client to accept the disease concept. Because treatment has clients ranging from new admissions to those ready to complete their program, senior peers are very influential in helping clients who are in the early stages of treatment to understand denial and the Disease Concept.

Acceptance and awareness that they are able to change if they take appropriate action to deal with their chronic condition is the message in the final treatment stage. The rehabilitation staff develops an aftercare plan with the client that will continue to support some of the changes that have taken place during treatment and it encourages changes that will promote ongoing recovery. Characteristically, clients comment on their increased awareness of simple pleasures and being with other people without trying to manipulate them. They are told that they must continue to work the AA steps, attend AA meetings, and address other problems of living if they are going to experience recovery because primary treatment is just one part of an ongoing continuum of care. Recovery is hard work made even more difficult by possible bouts of depression, problems of regaining trust from their family, and establishing new friends and activities not tied to alcohol and drug use.

Treatment outcome studies carried out by Hazelden for their treatment clients and for ten treatment programs in the Hazelden Evaluation Consortium are in general agreement with outcome evaluation findings reported by Comprehensive Assessment and Treatment Outcome Research for approximately one hundred hospital and freestanding treatment programs throughout the United States. About 50 percent of all clients treated, including noncompleters, are abstinent for one year following treatment discharge. This percentage is higher for treatment completers and for clients having fewer complications and more stability in their lives. Thirty-three percent of the clients have returned to heavy use patterns within the year, and the remainder have had slips or a period of resumed drinking/use but also have sustained periods of abstinence.

Abstinent clients have fewer legal, health, interpersonal, and job-related problems, and about 75 percent attend AA and/or continuing care.

The Minnesota Model is a label that is applied to a broad range of programming. Nevertheless, it represents a highly visible treatment modality serving a large number of clients throughout the United States, although it is more dominant in certain regions. It has a counterpart known as the Icelandic Model, and both of these treatment models have influenced treatment in SWEDEN and other parts of Scandinavia. International interest in adopting the Minnesota Model appears to be growing, with scattered treatment programs appearing in many countries. Little research has been done on the diffusion of this treatment model to other cultures.

(SEE ALSO: *Alcoholism; Treatment, History of*)

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Non-Medical Detoxification The term 'detoxification' is used to refer to the management of two distinct types of problem resulting from excessive alcohol or other drug use. These are the symptoms and behavioral changes associated with extreme *intoxication* on the one hand and of *withdrawal* following extended use on the other. Although both involve recovering from the toxic effects of a drug while refraining from further use, the problems associated with each are quite different and require different methods to tackle them. In relation to Western society's favorite drug, alcohol, these problems are so common that the challenge is to develop methods which can be widely used without excessive cost. This requirement tends to rule out an exclusive reliance on expensive medical settings, medical personnel and medication—even though both problems carry with them a small but significant risk of death or serious injury. Despite this restriction, human ingenuity has devised a number of relatively safe and cost-effective alternatives to hospital care and which are frequently preferred by the clients in need of 'sobering up' or 'drying out'. These innovative services have usually been developed for people who run into problems with their use of alcohol and have later been emulated by services for people who use other dependence-inducing drugs.

The most visible problems associated with extreme intoxication concern public order, particularly in relation to the use of alcohol. Drunkenness is associated with violence, both to the self and to others as well as with 'public nuisance' offenses. The habitual drunken offender, who may otherwise be quite harmless, and the potentially dangerous disorderly 'drunk' present themselves in huge numbers to police forces the world over and, typically, then clog up already overburdened court and penal systems. In the past two decades several countries have experimented with having drunkenness 'decriminalized' i.e. made no longer a criminal offense. The aim of this has been to free up the courts and the police so that they can concentrate on more serious crimes. Another impetus for decriminalization of drunkenness has been a growing awareness that locking up drunk people in police cells puts them at risk of serious harm. In Australia, for example, the tragic deaths of many Aboriginal people while in police custody are thought to have been caused by the combined effects of alcohol and confinement.

Historically, the setting up of non-medical detoxification services occurred hand-in-hand with the decriminalization of drunkenness. Among the first experiments in the 1970s were by the Addiction Research Foundation in the Canadian province of Ontario and St. Vincents' Hospital in New South Wales, Australia. In both cases, services were set-up with the principal aim of diverting drunkenness offenders from the criminal justice system to a

more humane setting where they might be also be counseled to seek help for their drinking problems. Both utilized a residential social setting staffed by non-medical personnel and provided no medical care or medication. To this day they successfully supervise thousands of problem drinkers, mainly self-referred, through sobering-up and/or alcohol withdrawal with an impressive record of safety. For example, in its first ten years of operation, the New South Wales facility has dealt with nearly 14,000 admissions and recorded only two fatalities among this high-risk population. Only 1 percent have required transfer to a nearby hospital for specialized medical care, often for reasons unrelated to alcohol withdrawal. These facilities have not been successful, however, in terms of attracting referrals from the police. In New South Wales, for example, the police have accounted for only 0.2 percent of referrals. It is possible that these facilities are diverting some potential offenders before they come to police attention, although this does not appear to be to a very significant extent.

SOBERING-UP SHELTERS

In an excellent review of detoxification services worldwide, Orford and Wawman (1986) suggest that the design of the above services confused the problems of intoxication and withdrawal. They should be seen as highly successful and cost-effective alternatives to hospital care for alcohol withdrawal but not the solution for what society should do with the habitual drunken offender. Australia's continuing concern to prevent Aboriginal deaths in custody has also prompted an increasing use of what have come to be called 'sobering up shelters'. These provide supportive non-medical settings where people can stay a few hours or, if necessary, overnight until, literally, they have sobered up. They have been found to provide an inexpensive alternative to prison and have succeeded in gaining the necessary support of the local police. Experience to date suggests that close liaison between shelter staff and police officers is necessary so that all concerned are clear about the specific aims of the project and how each can help the other. It is important that specialist treatment facilities are available to the sobering-up shelters so that people requiring urgent medical attention or longer-term help with a drinking problem can be referred on.

It should be noted that there are also potentially serious medical emergencies associated with extreme levels of drug intoxication. Poisoning through overdose, accidental or otherwise, is a common cause of admission to hospital emergency rooms the world over and all too frequently this may result in death. The most common of such instances are deliberate acts of self-poisoning, usually with prescribed medication, closely followed by cases of accidental alcohol poisoning. Over-dosing on heroin can also be quite common where that drug is widely used—especially as a result of users having lost tolerance to the drug's effects after a period of abstinence, if used with other CNS depressant drugs such as alcohol or benzodiazepines and/or if the heroin is unusually pure. It is for this reason that the staff of sobering up shelters, or of any facility which also caters for drug users, should be trained to identify the warning signs of overdose so that the sufferer may be taken to hospital with as little delay as possible. In some countries the opiate-antagonist drug Narcan is used in a variety of non-medical settings including by drug using peers at the scene of an overdose (Lenton and Hargreaves, in press). Similarly, there is a great educational need among the general drug-using and drinking public who all too often abandon their friends to 'sleep it off' and later find them asphyxiated.

DEALING WITH ALCOHOL AND OTHER DRUG WITHDRAWAL

Since the pioneering Canadian and Australian development of 'social setting' detoxification services to assist people safely through alcohol withdrawal, a variety of other non-medical approaches have been developed. Really, detoxification services should be seen as being on a continuum ranging from supervision by an informed 'lay person'—a relative, a recovered problem drinker or user or non-medical professionals—all the way to 24 hour nursing and medical care in a specialist hospital unit. Even in the latter case substantial variations exist regarding the amount of medication used during withdrawal—or even whether any medication is used at all. Detoxification services designed to minimize discomfort and the possibility of actual harm occurring during withdrawal may be 'non-medical' in several senses: by, variously, using non-medical settings (e.g. hostels, the client's home),

non-medical personnel (e.g. relatives, ex-problem drinkers) or non-medical procedures. There is wide consensus that medical assistance needs to be available if required but the responsibility for accessing this need not be left only with medical personnel.

The Ontario model of non-medical detoxification was created following the results of a study reported in 1970. It found in the relative safety of an alcoholism treatment unit that only 5 percent of admissions required any form of medical assistance. In addition to the residential 'social setting' model of detoxification, 'ambulatory' or outpatient detoxification procedures were developed which relied on the drinker calling in daily to a clinic to collect their medication and receive a brief check-up. Evaluations of these types of service conducted in several countries have demonstrated that their success rate in terms of both safety and effectiveness is at least the equal of inpatient care—and is considerably cheaper.

A variation of this approach is 'home detoxification', an approach developed initially in the UK with problem drinkers and now widely used in many other countries. This usually involves a community alcohol worker (e.g. nurse, counselor or psychologist) assisting a family practitioner to assess a drinker who wishes to stop drinking alcohol but who may experience severe withdrawal symptoms in the process. Providing the home environment is deemed to be supportive and the client sufficiently motivated to stop drinking the detoxification then occurs in the patient's home with supportive visits from the alcohol worker. The family doctor's telephone number is provided to the client and any close relative or partner in case of emergency. A particular effort is made to screen out drinkers with a history of withdrawal fits, delerium tremens or Korsakoff's Psychosis. In order to reduce the real risk of overdose with some types of medication (notably chlormethiazole) either the alcohol worker or a relative holds the medication. An important reason for developing this service in the UK was the discovery that many family doctors were already prescribing chlormethiazole to cover alcohol withdrawal but in the absence of any supervision and frequently longer than the recommended maximum period—sometimes even indefinitely. It was found that this was the single most common method of managing alcohol withdrawal among a group of patients who, for many reasons, were loathe to attend a psychiatric hospital or specialized treatment unit. Later studies have found evidence that home detoxification is more acceptable to groups that are frequently under-represented in traditional settings such as the young, the elderly and women. Home detoxification therefore offered a safe alternative to completely unsupervised withdrawal on the one hand and a cost-effective alternative to inpatient hospital care. The cost of Home Detoxification per client has been estimated to be approximately a quarter that of inpatient hospital care. Formal evaluations of the UK service suggest that not only is there no loss in terms of either safety or efficacy but that the clients prefer to be treated at home and that many would refuse to attend a hospital facility.

CONCLUSIONS

Non-medical detoxification services have been developed to cope with the problems associated with alcohol withdrawal in chronic heavy drinkers and also with episodes of alcohol-induced intoxication. While such services are being developed for users of other mood-altering drugs, there is, as yet, only limited published research concerning their efficacy. Non-medical detoxification services need clear aims and objectives and should be part of a comprehensive range services for people with alcohol problems. Both intoxication and alcohol withdrawal are so common in Western society that, although they carry a small but significant risk of serious injury or death, it is too costly to attempt to provide specialist medical care in every instance. Safe and inexpensive alternatives have been developed in a number of countries, which are to be recommended over a laissez-faire or punitive approach to these major social problems. There is encouraging evidence that community-based detoxification services attract problem drinkers who are usually under-estimated in treatment services, such as women, young people and the elderly.

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Outpatient Versus Inpatient With the rising cost of drug treatment and the growth of managed care, outpatient treatment is becoming a much more common form of treatment for substance abuse than inpatient treatment. Recent reviews of the scientific literature have supported this trend by showing that there is no strong evidence for the superiority of inpatient over less costly outpatient treatment. In fact, more recent investigations have focused on comparing various levels of intensities of outpatient treatment.

ALCOHOL TREATMENT

Finney et al. (1996) reviewed fourteen studies of ALCOHOL abuse and found that seven showed no significant differences in drinking outcomes between inpatient and outpatient treatment, five showed inpatient treatment to be superior, and in two studies a day hospital outpatient treatment was more effective. In the studies that found inpatient treatment to be more effective, patients in the comparative outpatient programs were less likely to receive an initial period of inpatient DETOXIFICATION and these studies were slightly less likely than those finding no treatment differences to randomly assign patients to treatment. Unless subjects are randomly assigned to each of the treatments, no way exists of knowing whether the findings were due to different kinds of patients volunteering for the different types of treatment. On the other hand, it could be argued that random assignment is an artificial selection process that makes it difficult to generalize findings to "real life" situation. Among the studies that compared costs, treatment in outpatient settings was less expensive than treatment in inpatient settings. Overall, the investigators concluded that there were no differences between inpatient and outpatient treatments. However, particular types of patients (e.g., those with medical/psychiatric impairments) may benefit more from inpatient treatment.

COCAINE TREATMENT

Alterman et al. (1994) found that a twenty-seven hour per week day hospital treatment was just as effective as more costly inpatient treatment for low SES male veterans. Both groups showed significant improvements in functioning at the seven-month follow-up evaluation. Although a greater proportion of subject assigned to inpatient treatment completed treatment, the day hospital treatment costs were 40 to 60 percent of inpatient treatment. Another randomized clinical trial comparing day and residential treatment programs for drug abuse (mostly COCAINE) found no overall differences in substance use problems between the two treatment conditions (Guydish et al.,1998).

Comparing Outpatient Treatment Intensities. As a result of finding no superior effect of inpatient treatment and given the limited availability of inpatient care, researchers are now comparing various intensities of outpatient treatment. Coviello et al. (in press) found no differences between male veterans randomly assigned to either a 12 hour per week day hospital program or a six hour per week outpatient program for cocaine dependence. Both treatments were similar in therapeutic structure and only differed in level of treat-

ment intensity. McLellan et al. (1997) found no differences between intensive outpatient programs of at least three sessions per week and traditional outpatient programs of one or two sessions weekly. In addition, Avants and colleagues (1999) have demonstrated that providing enhanced standard care for OPIATE-dependent patients enrolled in METHADONE maintenance treatment may be just as effective and less costly than intensive day treatment.

CONCLUSIONS

Research suggests that there are few differences between inpatient and outpatient treatment for substance abuse. Both treatments result in improvements in patient functioning. While inpatient treatment is more effective in retaining patients in treatment, it is much more costly than outpatient treatment. However, initial short-term inpatient treatment in the form of detoxification may be necessary to increase positive outcomes of later outpatient care. Recently, much more attention is being directed toward studying various levels of intensities of outpatient programs. Preliminary findings suggest that lower intensity outpatients treatments may be just as effective as similar higher intensity treatments. What seems to be more important is the content of the intervention rather than the setting in which the treatment is provided.

It should be noted that inpatient treatment is clearly indicated for patients with acute medical and psychiatric problems that can only be handled in an inpatient setting. Inpatient treatment may also be necessary for patients who continually fail in outpatient treatment, have few social sources, or whose recovery would be jeopardized in an outpatient program due to exposure to a social environment where substance use is prevalent. As a final cautionary note, much of the research in this area has been conducted with adult male clients. More research is needed with women and adolescent populations.

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Pharmacotherapy, An Overview Pharmacological agents may be used for several purposes in the treatment of drug and alcohol addiction. These include the alleviation of acute withdrawal symptoms, the prevention of relapse to drug or alcohol use, and the blocking of the euphorigenic effects of drugs of abuse. The various medications are used in the treatment of addiction to alcohol, opiates, cocaine, tobacco, and sedatives.

ALCOHOLISM

Detoxification. The use and abuse of ALCOHOL has been known to humankind for centuries, and alcohol is currently one of the most widely used of the mood-altering substances. Habitual alcohol use is associated with the development of TOLER-

ance and physiological (Physical) Dependence. Tolerance refers to a decrease in susceptibility to the effects of alcohol following chronic alcohol use, which results in the user consuming increasing amounts of alcohol over time. Physical dependence may be conceptualized as a physiological state in which the recurrent administration of alcohol is required to prevent the onset of withdrawal symptoms. Symptoms of alcohol withdrawal include irritability, tremulousness, anxiety, sweating, chills, fluctuations in pulse and blood pressure, diarrhea, and, in severe cases, seizure. These symptoms generally begin within twenty-four hours following the last use of alcohol, peak within forty-eight hours, and subside over several days.

Pharmacotherapy for alcohol withdrawal includes the use of agents, such as Benzodiazepines and Barbiturates, that are cross-tolerant with alcohol. These agents attenuate the symptoms of withdrawal and result in decreased arousal, agitation, and potential for seizure development. Medication is provided in doses that are sufficient to produce mild sedation and physiological stabilization early in the withdrawal period; this is followed by a gradual dose reduction and then discontinuation over the next one to two weeks. Currently, benzodiazepines are the agents of choice for the treatment of alcohol withdrawal, because of the relatively high therapeutic safety index of these medications, their ability to be administered both orally and intravenously, and because of their anticonvulsant properties. Barbiturates may be used in a similar fashion, but they have a lower therapeutic index of safety than do benzodiazepines.

Recent additions to the pharmacotherapy of alcohol withdrawal include clonidine and carbamazepine. Clonidine is an antihypertensive agent (i.e., it lowers blood pressure) that has recently been used in the treatment of drug withdrawal states and chronic pain. This medication decreses autonomic hyperactivity (i.e., it lowers an increased pulse and blood pressure), but it does not have the anticonvulsant properties of the benzodiazepines or barbiturates. Carbamazepine has also been employed in the treatment of alcohol withdrawal and does have anticonvulsant properties. Neither medication is habit forming and thus may have potential in the treatment of alcohol withdrawal.

Maintenance Lithium. Lithium is primarily employed in the treatment of bipolar mood disorder (previously termed manic-depressive disorder), but it may be beneficial in the treatment of other psychiatric disorders. It has received much attention in the investigation of pharmacologic agents for the treatment of alcohol dependence, and several studies have reported that its use had favorable effects on alcohol consumption. For example, after receiving doses of lithium comparable to those administered to human beings, laboratory animals demonstrated a significant reduction in alcohol consumption. In recovering alcoholics, lithium treatment has been associated with a decreased desire to continue drinking after alcohol use and, in several studies, with a higher rate of abstinence for those alcoholic patients who were compliant with therapy. Although these small studies on the efficacy of lithium for alcohol dependence appeared promising, a recent large placebo-controlled study failed to demonstrate a beneficial effect of lithium. At the present time, although lithium certainly has a place in the treatment of alcoholic patients with bipolar disorder, the indications for its use in other patients with alcohol dependence are less clear.

Antidepressants. Depressive symptoms are noted in many alcoholics at the time that they enter treatment. Because of the frequent co-occurrence of depression and alcoholism, the use of antidepressants would appear to be potentially useful in this population. Several studies have demonstrated favorable effects of antidepressants on alcohol consumption. Tricyclic antidepressants such as imipramine and desipramine inhibit the re-uptake of norepinephrine and serotonin in nerve terminals. These medications have been associated with decreased ethanol consumption in laboratory animals and in human alcoholic subjects. The serotonin reuptake inhibitors (blockers) zimelidine, viqualine, fluvoxamine, and fluoxetine (Prozac) have also demonstrated favorable short-term results in the treatment of alcohol dependence. Although these medications are not routinely administered to all recovering alcoholics, many physicians consider the use of antidepressants in alcoholic patients if depressive symptoms do not resolve after several weeks of abstinence, or if a mood disorder was present prior to the onset of ethanol abuse.

Anxiolytics. Used to decrease anxiety, anxiolyrics include benzodiazepines, such as chlordiazepoxide (Librium) and diazepam (Valium), and azaspirodecadiones, such as buspirone. Both classes of medication have been investigated

for use in alcohol dependence. Early studies supported the use of benzodiazepines in recovering alcoholics with claims of decreased alcohol craving and consumption after chlordiazepoxide administration. Other controlled trials refuted this, however, and many physicians would question the use of benzodiazepines in this population. The azaspirodecadiones such as buspirone are nonaddictive medications that have been marketed for the treatment of anxiety. Although few controlled trials have been conducted that evaluated the effect of buspirone on human alcohol use, animal studies have demonstrated decreased alcohol consumption after treatment with this agent. Unlike benzodiazepines, buspirone is not known to be habit forming and thus may be a promising agent for additional controlled studies in human subjects.

Dopaminergic Agents. The effects of dopaminergic agents on the consumption of alcohol in animal studies have been conflicting, since both agents that augment dopaminergic activity and those that diminish it have been noted to decrease alcohol consumption. In humans, controlled studies with apomorphine and bromocriptine, both of which increase dopaminergic activity, have revealed decreases in alcohol craving, anxiety, and depression, and increased abstinence among alcoholic depressed patients.

Opioid Antagonists. Opioid antagonists are competitive antagonists of OPIODS at opiate receptors. They include NALOXONE, which may be used intramuscularly or intravenously to rapidly reverse opiate intoxication, and NALTREXONE, which is prescribed orally to prevent or reverse intoxication from opioids. Unlike opioids, these medications are not habit forming and may have a place in the treatment of alcohol-dependent patients. A variety of studies have demonstrated a reduction of alcohol consumption or self-administration by experimental animals treated with these agents. In human subjects, naltrexone administered as an adjunct to substance-abuse treatment has resulted in a decreased rate of alcohol consumption. In addition, those patients who did experience a "slip" were less likely than those who were not treated with naltrexone to suffer a complete relapse to alcohol use.

Antidipsotropics. Antidipsotropics are medications that are used to decrease alcohol consumption by creating an adverse reaction following alcohol use. They include DISULFIRAM, CALCIUM CARBIMIDE, and Flagyl. Disulfiram use results in an accumulation of acetaldehyde following the consumption of alcohol. Acetaldehyde levels accumulate if patients who are receiving disulfiram ingest alcohol, with the result that the patients may experience symptoms of acetaldehyde toxicity. These include sweating, chest pain, palpitations, flushing, thirst, nausea, vomiting, headache, difficulty breathing, hypotension, dizziness, weakness, blurred vision, and confusion. Symptoms may begin within five to fifteen minutes following alcohol ingestion and may last from thirty minutes to several hours. The use of disulfiram is based upon the premise that the fear or actual experience of this adverse event may serve as a deterrent to alcohol use. Despite its toxicity, disulfiram has been used safely by thousands of recovering alcoholics since its introduction in 1948. Supervised voluntary use of the medication as an adjunct to other rehabilitative therapy has resulted in reduced alcohol consumption and decreased alcohol-related criminal behavior among alcohol-dependent patients.

Compliance is the key to successful use of disulfiram in alcohol dependence, since patients need only discontinue using disulfiram if they wish to resume drinking. Indeed, in an unsupervised setting, disulfiram administration shows no superiority over placebo on outcome measures related to alcohol use. Methods that have been investigated to improve compliance include surgical implants of disulfiram, reinforcement by providing a reward for compliance, and contingency management techniques. Although surgical implants have met with little success, the other two methods have demonstrated various degrees of efficacy.

OPIOID DEPENDENCE

The opioids include opiates, drugs derived from the opium poppy (*Papaver somniferum*), as well as those synthesized to produce similar narcotic effects. Opium has been used as a medicinal substance for at least 6,000 years. Widespread abuse of opiates was noted by the eighteenth century, with the smoking of opium in Asia; currently, HEROIN is a major opiate of abuse in the United States. Pharmacotherapy for opiate dependence may be employed both during the acute withdrawal syndrome and later to maintain abstinence from illicit opioids (e.g., heroin).

Acute Opioid Withdrawal. The syndrome of acute withdrawal from opiates varies in regard to

the opiate of abuse. The time of onset, intensity, and duration of withdrawal symptoms depend on several factors, including the half-life of the drug, the dose, and the chronicity of use. Heroin is a relatively short-acting agent; symptoms of withdrawal often begin within eight to twelve hours after the last use. Early symptoms include craving, anxiety, yawning, tearing, runny nose, restlessness, and poor sleep. Symptoms may progress to include pupil dilation, irritability, muscle and bone aches, piloerection (the goose bumps—thus the term *cold turkey*), and hot and cold flashes. Peak severity occurs 48 to 72 hours after the last dose and includes nausea and vomiting, diarrhea, low-grade fever, increased blood pressure, pulse, and respiration, muscle twitching, and occasional jerking of the lower extremities (which explains the term *kicking the habit*). The opiate withdrawal syndrome following chronic heroin use may last seven to ten days, but with longer-acting agents such as METHADONE, a similar constellation of symptoms may occur; they begin later, peak on the third to eighth day, and persist for several weeks.

A variety of medications may be used in the treatment of acute opiate withdrawal. The most common method is to use opiates alone. A dose high enough to stabilize the patient is administered on the first day and then gradually tapered over one to two weeks. Generally, long-acting opiates such as methadone are employed, but any opiate may be used.

Other medications used for opiate withdrawal are CLONIDINE and BUPRENORPHINE. Clonidine is an alpha-2 adrenergic agonist that is commonly employed as an antihypertensive medication. It is active on central nervous system (CNS) locus coeruleus neurons in the same areas at which opiates exert their effects. Clonidine appears most effective in decreasing symptoms such as elevation of pulse and blood pressure and may be less effective in relieving other symptoms of withdrawal. The major side effects of clonidine are orthostatic hypotension and sedation. A recent development in the pharmacotherapy of opiate withdrawal is rapid detoxification through the combined use of clonidine with opiate antagonists such as naltrexone. This treatment may decrease the time required for the detoxification process to two to three days. Opiate addicts may be stabilized on buprenorphine, a mixed opioid agonist/antagonist, with minimal discomfort and then withdrawn over five to seven days with less severe withdrawal symptoms than those associated with methadone withdrawal.

Antagonists. Opiate antagonists such as naloxone and naltrexone compete with opiates for CNS opioid receptors. Naloxone has a short half-life (two to three hours) and is generally employed on a short-term basis to reverse acute opiate intoxication. Naltrexone has a longer duration of action (approximately twenty-four hours) and is used as a long-term maintenance medication to inhibit euphoria in opioid addicts. Both medications have been used with relative safety for several years, and maltrexone has been successfully employed as an adjunct to other therapies in the treatment of opioid addicts. Clinically, side effects of naltrexone may include mild dysphoria and elevation in cortisol and beta-endorphin levels; no withdrawal syndrome has been noted following its discontinuation. Naltrexone is generally administered three to four times a week at an average dose of 50 milligrams per day. Despite its advantages, many opioid addicts resist therapy with this medication, and even in the most successful of programs, six-month retention rates may range from only 20 to 30 percent. The addition of psychosocial interventions such as counseling and contingency-management programs is helpful. When these interventions are added, naltrexone has been noted to be particularly effective in selected groups, such as those made up of health care professionals, business people, and prisoners on work-release programs.

Methadone Maintenance. Methadone has been used as a safe and effective treatment for opioid dependence for over twenty years. Heroin addicts easily adapt to using this long-acting opiate that possesses all of the physiological characteristics of heroin. When taken orally, methadone may have less abuse potential than heroin, but the onset of its CNS effects are slower and its tendency to induce euphoria is generally less than that of intravenous or inhaled heroin. In addition, it has a longer half-life than heroin and if it is administered daily, tissue levels accumulate, thereby decreasing interdose withdrawal symptoms that may lead to repeated opiate use. Methadone maintenance may be helpful for addicts who have difficulty adjusting to a drug-free lifestyle or for those who have been unsuccessful with other forms of treatment.

During maintenance therapy, methadone is initiated at a low dose and then gradually increased to higher doses, which are associated with decreased

opiate craving and secondary illicit opiate use. With methadone maintenance treatment, many patients show significant decreases in illicit drug use, depression, and criminal activity, and they demonstrate increased employment. Therapy that is provided for extended periods of time and in the context of other psychosocial services has been associated with the highest success rates.

Another maintenance medication currently under investigation is levo-alpha-acetylmethadol (LAAM). LAAM is a long-acting form of methadone that requires administration three times per week instead of daily as with methadone. Although LAAM has been associated with a reduction in illicit opioid use, its slower onset of action may lead to decreases in treatment retention compared to the use of methadone. The initiation of treatment with methadone and subsequent conversion to LAAM therapy may improve compliance with this medication. LAAM is not yet routinely used in the treatment of opioid dependence, and additional studies will be necessary to determine the appropriate use of this agent.

Buprenorphine. Buprenorphine is a mixed opioid agonist/antagonist that has been used for several years as a possible maintenance medication for opioid dependence. Although it has only recently been available within the United States, preliminary studies indicate that it may be a promising agent for the treatment of opioid dependence. As with methadone, maintenance treatment consists of daily administration of buprenorphine, but the optimal daily dose of medication remains under investigation. At low doses, buprenorphine has agonist effects at opioid receptors, but at higher doses antagonistic effects may occur. Buprenorphine maintenance has been associated with good treatment retention, decreased illicit opiate use, and a relatively mild withdrawal syndrome. On the basis of early studies, buprenorphine was thought to be a promising agent in the treatment of both cocaine and opioid dependence, but significant benefits have not been confirmed by better-controlled studies.

COCAINE DEPENDENCE

Cocaine abuse has increased markedly since the 1970s, and by 1984, more than 20 million Americans reported that they had tried cocaine. In addition to psychotherapy and other traditional approaches to substance-abuse treatment, a variety of pharmacotherapeutic interventions may be of benefit to cocaine abusers.

Pharmacotherapy for cocaine abuse may be employed to address specific symptoms that occur during the cocaine-withdrawal syndrome. Gawin and Kleber identified three phases in the cocaine abstinence syndrome. The crash phase generally begins soon after cocaine use ends and may last up to four days. Symptoms experienced at this time may include depression, suicidal ideation, irritability, anxiety, and intense cocaine craving. Sedatives such as alcohol and heroin may be used by addicts to alleviate these symptoms. The second or withdrawal phase may last two to ten weeks and is characterized by anxiety, depression, inability to experience pleasure, and increased cocaine craving. The third or extinction phase may last three to twelve months; during this phase, cocaine craving may continue as well as increased susceptibility to relapse in response to environmental cues.

Pharmacotherapy for cocaine dependence may be used to alleviate symptoms experienced during the cocaine abstinence syndrome. During the crash period, early symptoms such as anxiety and insomnia may be relieved by benzodiazepines such as CHLORDIAZEPOXIDE. Neuroleptics (ANTIPSYCHOTICS) may also be helpful during this period to alleviate psychotic symptoms such as paranoia.

Other agents that may be used on a short-term basis include dopaminergic agents such as bromocriptine and AMANTADINE. Some investigators postulate that CNS dopamine may be depleted by chronic cocaine use. Dopaminergic agents may be used to augment CNS dopaminergic function, and various dopaminergic agents such as amantadine, bromocriptine, and L-dopa have been employed for this purpose. Although few long-term, double-blind, placebo-controlled studies have been conducted, several studies have supported the use of dopaminergic agents such as amantadine as anticraving medications during withdrawal.

Antidepressants may be helpful during the withdrawal and extinction stages of cocaine abstinence. One controlled and several uncontrolled studies in recovering cocaine addicts suggested that the tricyclic antidepressant desipramine might decrease cocaine use and craving. Other antidepressants investigated in pilot studies include fluoxetine, imipramine, doxepin, and trazodone. Antidepressants may take several weeks to begin to alleviate symp-

toms of depression or craving, however, and some cocaine addicts may drop out of treatment during this period. These patients may benefit from initiation of treatment with a short-term agent (such as a dopaminergic agent) followed by long-term treatment with an antidepressant. As with every treatment, however, no firm conclusions are warranted about any agent until it has been tested in a controlled clinical trial that has been replicated at least once.

Pharmacotherapy may also be helpful for patients with psychiatric diagnoses other than cocaine dependence. In some patients, cocaine abuse may be an attempt at self-medication to address the discomfort of depression or other psychiatric disorders. Patients with major depressive disorder and bipolar disorder may respond to therapy with antidepressants or lithium, and those with attention deficit disorder may benefit from the cautious use of low doses of stimulant medication.

In summary, antipsychotics and benzodiazepines may be used to alleviate symptoms of acute cocaine withdrawal, whereas tricyclic antidepressants and dopaminergic agents may be helpful in the long-term treatment of cocaine withdrawal. Pharmacotherapy should be considered an adjunct to other forms of rehabilitative therapy during the long-term treatment of the cocaine-dependent patient.

TOBACCO DEPENDENCE

One commonly used pharmacological treatment for tobacco dependence is a nicotine-containing gum called Nicorette. The main reason to quit smoking cigarettes is its powerful association with lung cancer, emphysema, and other medical problems. Yet nicotine, the active ingredient in cigarettes, is another drug that is associated with pleasant effects and with withdrawal discomfort, thereby making it an extremely addicting drug. Providing cigarette smokers with nicotine replacement in the form of a gum will help them avoid the health risks associated with smoking cigarettes. One problem with Nicorette is that it is difficult to chew correctly and therefore people need to be trained in how to chew it in order to derive the therapeutic effect. Recently, a patch has been developed that is placed on the arm and automatically releases nicotine. A method that shows good potential as a treatment, the patch was made available in the early 1990s. Detoxification from nicotine may also be facilitated with the medication clonidine, the same agent used to help alleviate opiate withdrawal symptoms.

SEDATIVE DEPENDENCE

Current treatments for sedative dependence include detoxification agents rather than anticraving agents. Detoxification is accomplished by tapering the dosage of benzodiazepines over two to three weeks. More recently, carbamazepine, an antiseizure medication, was shown to relieve alcohol and sedative withdrawal symptoms, including seizures and delirium tremens. Future work with agents that block the actions of benzodiazepines may hold promise as a maintenance or anticraving agent used to help the sedative abuser abstain from drug abuse.

CONCLUSIONS

Medications must be accompanied by psychological and social treatments and support; they do not work on their own. Moreover, medications to block illicit-drug effects in the brain may be of little use if the patient does not take them. More research in many fields is needed to identify potential medications, but this research must recognize the psychosocial as well as the neurobiological areas of therapy. Without this integration, the work to develop more effective treatments for the difficult problem of drug abuse and dependence cannot begin.

(SEE ALSO: *Causes of Substance Abuse; Complications; Disease Concept of Alcoholism and Drug Abuse; Nicotine Delivery Systems for Smoking Cessation*)

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Psychological Approaches Psychological treatments of drug dependence assume that drug abuse is a learned behavior. As such, it is not different from other less controversial and more healthful behaviors in its development. That is, a psychological perspective suggests that drug abuse is, for the most part, learned in many of the same ways as behaviors such as reading or driving a car. This perspective also suggests that drug abuse can be changed in the ways that other behaviors are changed. Forces for change include rewards (reinforcers) and unpleasant events (punishments); cues that signal the need for specific actions (discriminative stimuli); and training in new ways of thinking about oneself and the world that lead to ways of living that do not involve drugs.

Operant Learning Models. Psychological treatments for drug abuse can be grouped into three categories, based on the models of behavior that they represent. The first are those that draw from operant learning models. These models suggest that many important behaviors, including those many behaviors that end with the use of an illegal drug are controlled by environmental events, rather than events inside the individual. Internal events may come into play but, ultimately, these are caused by external events. These models suggest that the important factor in determining drug use is the balance between the rewards and the punishments of use. CONTINGENCY MANAGEMENT, a system of rewards for abstinence and punishment for drug use, is an example of an operant-based treatment.

Classical Conditioning. A second model used is classical conditioning. A neutral event is paired repeatedly with another important event, one that usually evokes a response for the organism. A man who has experienced heroin withdrawal many times may eventually find that certain rooms of his apartment itself have come to cause him to crave drugs, because the apartment itself has become associated with withdrawal. A treatment based on classical conditioning, for example, is an attempt to remove the craving induced by the sight of drug paraphernalia, by repeatedly presenting pictures of those paraphernalia with no drugs, and therefore with lack of a reinforcing response.

Social Learning Models. Other treatments draw from social learning models. These assume that behaviors, such as drug abuse, are learned in many ways, including operant conditioning, classical conditioning, imitation (learning by watching someone else), and learning certain ways of thinking. These models also usually assume that imitation and learning new ways of thinking are more important for humans than other ways of learning. An example of a treatment based on a social learning model is cognitive behavioral psychotherapy, where the drug abuser is taught new ways of viewing old situations, as well as new social skills, in the hope that these new thoughts and skills will lead to a less troubled life, which does not demand drug abuse to make it tolerable.

OPERANT MODELS: CONTINGENCY MANAGEMENT

Contingency management has been incorporated into many drug-treatment programs as a way of assisting people in reducing drug use. In contingency management, reinforcers or punishers are applied depending on the patient's behavior. Often, contingencies are formalized in a contract. In contingency contracting, a treatment plan is developed and agreed to by treatment staff and patient. As part of the contract, both agree that certain consequences will occur as a result of certain behaviors on the part of the patient.

Early work indicating the usefulness of contingencies was completed largely at Johns Hopkins University. Working in a methadone-maintenance program, investigators at Johns Hopkins found that money and the opportunity to raise dose levels all served to decrease drug abuse. Work at the University of California in detoxification treatment programs also indicated that payment for drug abstinence was an effective adjunct to short-term detoxification treatments, where methadone is used for only about three weeks, to help drug abusers in their transition from heroin use to a drug-free state. Both of these experimental programs focused on rewards for desired behavior, rather than punishments for drug use. Contingencies also have been used to help clients conform to other treatment demands, including attending counseling sessions (Stitzer & Kirby, 1991).

Even though early work focused on providing positive reinforcers for desired behavior, the adaptations of this work in most clinics around the country has involved negative consequences. For reasons not clear, most clinical sites that have adopted the contingency contracting procedures use punishers, not reinforcers. A common example is the use of a detoxification contract in methadone-maintenance treatment. Frequently, patients who are using illegal drugs sign a contract with treatment staff indicating that if they do not terminate all unapproved drug use within a certain period of time, their methadone dose will be reduced. If they continue to use drugs, their dose is incrementally reduced until they are no longer receiving methadone. At any point in the sequence, however, that the patient shows evidence of discontinuing drug use, the methadone dose can be raised and the person continued on the treatment program. Usually, the contract indicates that patients are given a certain amount of time to decrease the number of drug-positive urines or they are gradually detoxified from the program.

Contingency management has been used with practically every addiction, both by itself and in conjunction with other treatments. The evidence is now convincing that contingencies, especially positive contingencies, are effective in decreasing drug abuse. Work is needed to train clinic staff in using contingency programs, especially those employing positive contingencies (Stitzer & Kirby, 1991).

CLASSICAL CONDITIONING: AVERSIVE CONDITIONING

A form of behavioral therapy once widely used is AVERSION THERAPY. Here, the drug or the cues that remind drug users of it are paired with unpleasant events. The notion is that by pairing this very desirable substance with an unpleasant event, the association with the substance will become negative. The most successful of these has been rapid smoking, a treatment for tobacco dependence. In rapid smoking, the smoker smokes and inhales at a rate about 6 times that of normal. During this process, the therapist points out negative things about smoking, including the smell of the smoke, burning eyes, racing heart, and pounding head. Over time, the poisonous elements of the smoke itself (usually an amount of NICOTINE that exceeds the smoker's tolerance) may make the smoker nauseated. Thus, the cues associated with a cigarette (its appearance and smell) rather than calling forth pleasant reactions in the smoker, come to call forth unpleasant ones. Aversive-conditioning treatments have been attempted with other drugs, most notably ALCOHOL and COCAINE. Usually, for example, a chemical that induces vomiting is given so that nausea and vomiting occur at about the same time the patient is drinking in a controlled setting. However, aversion treatments for drug abuse other than TOBACCO abuse have had limited success or, at least, limited popularity. There are at least two reasons for this. First, with other drugs, the dose of the problem drug needed to produce unpleasant reactions may be physiologically dangerous. Second, rapid smoking is unique in that it is the actual drug, tobacco smoke, that is used to form the aversion. There is evidence in the psychological literature that such aversions are especially potent.

Aversive smoking has been evaluated in several well-controlled studies. It appears that when it is done correctly, abstinence rates can be as high as 60 percent after one year—a very high abstinence rate indeed—since the average abstinence rate after treatment for cigarette smoking is about 20 percent. The data for aversion for alcoholics using chemicals is not so clear. There are few comparisons with other treatments or with no treatment. Individuals who choose aversion treatment may be especially motivated to change, and they might have achieved high abstinence rates even without treatment.

One variant of aversion conditioning is covert conditioning. In covert conditioning, the drug abuser, with the help of a therapist, imagines both the drug use and the unpleasant consequences of it. For example, alcoholics might picture a cold beer, prepare to savor it, took at it, sip it, then slowly feel increasingly nauseated until they become violently ill. Thus, both the aversive events and the unpleasant consequences are imagined, rather than real. This has advantages if the drug of choice is illegal or quite dangerous, because it avoids drug use at all. Also, patients who might refuse to participate in actual aversive conditioning may feel able to do so when the aversion experienced is imagined. Unfortunately, however, there is not a great deal of evidence to support the usefulness of this approach (Council on Scientific Affairs, 1987).

The use of aversion conditioning has decreased recently, except in a limited number of private psychiatric hospitals. There are several reasons contributing to its demise. The first is the lack of demonstrated efficacy in controlled clinical trials with drugs other than tobacco. The second is its expense when compared with other treatments. Last, because of its intrinsically unpleasant nature, it has low acceptability.

SOCIAL LEARNING MODELS

Skill Training. In skill training, drug abusers, and others at risk for drug abuse, are taught skills that will help them not to use drugs. These can be simple and direct; for example, teaching junior high school students effective ways to refuse a cigarette. The skills learned may also be complex. Consider, for example, a smoker who knows the temptation to smoke when angry, because in the past anger-provoking situations have resulted in relapse. A therapist working with such a person in skill training would first review the situations that produce anger. These might be as diverse as incorrect charges on a credit card bill to a fight with the boss. After identifying the situations, the smoker and therapist would then discuss the details of the situation. For example, they might imagine what the boss would say to smokers to elicit anger. They would attempt to find ways of handling the situation that would leave the smokers feeling satisfied after it was over. They would discuss the usual response that would culminate in smoking. They would then identify alternative responses. Finally, they would role-play the alternative responses. The therapist would play the role of both the boss and the smoker, to give the smoker a model of different ways to handle the situation. In this way, the smokers would learn to handle anger in a better way, would be satisfied with the new responses, and be less likely to smoke. The smoker would also have ready responses other than smoking. Skill-training programs have been studied with smokers, alcoholics, cocaine abusers, and abusers of multiple drugs. Skill training is closely related to the recovery training and self-help that is discussed below. Recent data indicate skill training may be an especially useful treatment for heroin and/or cocaine abusers and alcoholics when used in the context of a large therapy program (Carroll, Rounsaville, & Gawin, 1991).

Skill training has been shown to be especially useful as an ancillary to other treatments. For example, one program developed a workshop to train drug-treatment patients in job-finding skills. There was a great deal of practice in new ways to interview for jobs. Patients were taught how to fill out a job application to maximize their strengths—also how to handle the existence of prison records or long lapses in employment. They practiced their interviews and saw themselves in practice interviews on videotape. The rationale was that if drug abusers could be taught to present themselves positively in a job interview, they would be more likely to get jobs. And, were they to become employed, they would be less likely to use drugs, for several reasons. These reasons include increased general life satisfaction and making new friends and social contacts who are not drug abusers. Studies using this technique found that it was helpful in increasing employment rates in both METHADONE-MAINTENANCE clients and former addicts recruited from the criminal-justice system. These studies did not address the length of time the job was held, however. It may be that a separate set of skills is needed to maintain employment. This set should be the object of further study (Hall et al., 1981).

Some programs have attempted to combine several approaches, so that abstinence is supported in multiple ways. Among the most successful of these is the community-reinforcement approach to alcoholism treatment developed by Azrin (1976). The original community-reinforcement approach incorporated (1) placement in jobs that interfered with drinking; (2) marriage and family counseling;

(3) a self-governing social club; and (4) encouragement to engage in hobbies and recreational activities that could substitute for drinking. This procedure was found to decrease time spent drinking alcohol, increase rates of employment, increase time spent with families, and decrease the time spent in the hospital being treated for alcoholism. A later revision of the program also encouraged patients to take DISULFIRAM, a drug which produces unpleasant reactions if one drinks after taking it; taught alcoholics how to identify and handle danger signals so that they did not lead to drinking; provided patients with a "buddy" in the client's neighborhood; and switched from individual to group counseling. This procedure produced even more strikingly positive results than the original program. It can be argued that subjects in these studies had resources available to them that many drug abusers and alcoholics do not have, including the opportunity to receive inpatient treatment, a local economy that provides a choice of job opportunities, and supportive families. Recent work with cocaine abusers has replicated these positive results. The finding is especially impressive because the cocaine abusers were treated on an outpatient basis, and they traditionally have fewer resources than alcoholics.

Psychotherapy. Psychotherapy has also been useful in treating drug addicts, especially those with social and psychological problems that complicate their drug abuse. The assumption behind providing psychotherapy to drug abusers is that drug abuse is motivated by the problems that abusers have with other people, as well as their feelings about themselves. Early workers in the field attempted to provide psychotherapy as the sole treatment for drug abuse. Most found that it was not successful; they assumed that this was because the personality characteristics of addicts were not those that allowed people to succeed in psychotherapy—that is, addicts are often distrustful of nonaddicts and may not easily reveal their feelings to professionals. Also, they may not be especially reliable and often appear to have shaky to no motivation to change. Nevertheless, a large-scale study at the University of Pennsylvania—using clients who were already in methadone maintenance—found that, in the context of a larger treatment program, drug-treatment clients with other or extensive psychological problems do benefit from the addition of psychotherapy. The forms of psychotherapy available included one focusing on feelings and emotions (supportive—expressive) and one focusing on thought and behaviors (cognitive—behavioral). These researchers found that the type of therapy was not important, just participating in therapy was important (Woody et al., 1983).

The Recovery Training and Self-Help Model. Researchers at Harvard University studied a model that combined skill training in Relapse Prevention with Self-Help Groups. In their study, opiate addicts attended a recovery-training session once a week and a self-help group led by a former addict. Members also met informally outside the treatment meetings and in group-sponsored recreation and community activities. In the professionally led recovery meetings, leaders addressed a variety of topics, including high-risk situations, friendships, physical illness, and relations with family; they developed new ways of handling these situations that would be less likely to lead to drug use. The self-help groups supported these changes and further reinforced them. In two studies, one in the United States and one in Hong Kong, this treatment led to higher rates of abstinence or infrequent use than was found in a control condition, to increases in employment, and to fewer reports of criminal behavior. These differences were quite long-lasting—occurring six months to one year after entrance into treatment (McAuliffe & Ch'ien, 1986).

Twelve-Step Programs. The most well-known TWELVE-STEP program for helping substance abusers is ALCOHOLICS ANONYMOUS (AA). AA, founded in 1935 by a group of recovering alcoholics, is a fellowship of men and women who are committed to helping other alcoholics. NARCOTICS ANONYMOUS (NA), founded in 1953, was adapted from AA principles to include all substance abusers, not only alcoholics.

AA and NA programs focus on alcoholism and substance abuse as a disease for which there is no cure—therefore recovery becomes a lifetime commitment. These programs emphasize the personal powerlessness of individuals in combating their illness and get individuals to recognize that they must give themselves to a greater power so that they may be saved.

The guiding tenets of AA and NA programs are called the Twelve Steps. Each step is a passage through recovery, combining self-discovery with spiritual guidance. They involve five psychological

tasks: (1) recognition and admission of powerlessness over alcohol; (2) acceptance of a high power as a source of strength and guidance during recovery; (3) self-help appraisal and self-disclosure in the service of personal change; (4) making amends for past wrongs; and (5) carrying the AA message to others (Anderson & Gilbert, 1989).

One can argue that aspects of AA parallel psychological approaches. For example, similar to psychotherapy, AA and NA members are encouraged to "work through" problems and to change the attitudes and actions associated with an alcohol- or drug-using lifestyle. These programs also use principles common to other self-help groups. Members are encouraged to attend meetings on a daily or weekly basis, at which the steps are discussed and made relevant, speakers recount their lives, and connections with support networks and role models are made.

Nevertheless, despite the facility with which psychological models might explain such approaches, they are not psychological approaches. They were developed from a spiritual approach, not from psychological principles.

SUMMARY

There are many psychological treatments that appear to be useful in aiding drug abusers to stop using drugs, no matter whether the drug be an illegal one, or alcohol or nicotine. Positive results come from contingency-contracting programs and multifaceted-reinforcement programs that are offered in the context of complex treatment programs or from skill-training programs that address several facets of the drug abuser's life. Also, there is evidence for the usefulness of different forms of psychotherapy for drug abusers, especially for those who have psychological and social problems. Drug abuse is increasingly becoming identified as a complicated problem that involves both biological and psychological factors. Because of this and the clear usefulness of psychological intervention, we can expect to see the development of new psychological treatments for drug abuse.

(SEE ALSO: *Addiction: Concepts and Definitions; Adjunctive Drug Taking; Causes of Substance Abuse; Disease Concept of Alcoholism and Drug Abuse; Prevention; Vulnerability; Wikler's Pharmacologic Theory of Drug Addiction*)

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Self-Help and Anonymous Groups Self-help groups for drug and alcohol abuse, often called mutual-help groups, are of two basic types. First are the long-standing anonymous groups closely patterned after ALCOHOLICS ANONYMOUS (AA). An alternative type also has a group context,

but rejects the spiritual aspects (such as reliance on "higher power") of AA and urges members instead to take personal responsibility for gaining sobriety. The AA-like anonymous groups embrace the TWELVE STEPS, applying them to their own particular disorder. In some instances, they also adapt the AA Twelve Traditions. NARCOTICS ANONYMOUS, Emotions Anonymous, Overeaters Anonymous, Gamblers Anonymous, AL-ANON, COCAINE ANONYMOUS, and Nicotine Anonymous are prominent examples. Examples of the alternatives to AA are RATIONAL RECOVERY (RR), SECULAR ORGANIZATION FOR SOBRIETY (SOS), and WOMEN FOR SOBRIETY (WFS). Numerous members of these groups have been dropouts from AA.

In embracing AA's Twelve Steps, the first type of organization teaches powerlessness over their malady, reliance on the group or on some entity as a "higher power," catharsis via self-inventory, confession and amends, and a commitment to search out and tell others suffering from the same disorder about their programs for recovery. The rationale is that members have deep-seated denials that must be blunted by admitting helplessness and invoking the group and a higher power to help them. Moreover, this powerlessness is seen as a lifetime condition and the Twelve Steps are seen as providing a mechanism for ensuring a lifetime cessation of the compulsive behavior. The steps were devised in the late 1930s by Bill W., the major cofounder of AA, in conjunction with a small group of his earlier followers.

The Twelve Steps of Alcoholics Anonymous.

1. We admitted we were powerless over alcohol—that our lives had become unmanageable.
2. Came to believe that a Power greater than ourselves could restore us to sanity.
3. Made a decision to turn our will and our lives over to the care of God *as we understood Him*.
4. Made a searching and fearless moral inventory of ourselves.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. Were entirely ready to have God remove all these defects of character.
7. Humbly asked Him to remove our shortcomings.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.
10. Continued to take personal inventory, and when we were wrong, promptly admitted it.
11. Sought through prayer and meditation to improve our conscious contact with God *as we understood Him*, praying only for knowledge of His will for us and the power to carry that out.
12. Having had a spiritual awakening as the result of these steps, we tried to carry this message to others, and to practice these principles in all our affairs.

SOURCE: The Twelve Steps are reprinted with permission of Alcoholics Anonymous Word Services, Inc. Permission to reprint this material does not mean that AA has reviewed or approved the contents of this publication, nor that AA agrees with the views expressed herein. AA is a program of recovery from alcoholism *only*—use of the Twelve Steps in connection with programs and activities patterned after AA, but which address other problems, does not imply otherwise.

The second type of organization emphasizes that individuals, as individuals, must use their own resources and, in effect, "Save Our Selves" (SOS). The founder of WFS has written Thirteen Statements of Acceptance around which meetings are anchored: For example, number 5 is "I am what I think," and number 13 is "I am responsible for myself and my actions." The other statements encourage in women alcoholics a strong feeling of self-worth even though they have symptoms of a serious disease (Kirkpatrick, 1989).

The two types of organizations differ on basic treatment strategies. One difference is their divergent views of the permanency of their obsessive behavior. AA, and the many AA-like groups, view their problems as lifetime conditions over which they are powerless. In short, they will never recover; they are permanently "recovering" from a disease. In contrast, RR, for example, plays down the disease concept, and the higher-power notion that goes with it, and appeals to forces within a member's own intellect and willpower. Self-reliance is taught. WFS targets the development of self-value, self-esteem, and self-confidence as a way to meet the emotional needs of modern

women, thereby, members believe, reducing significantly the basic roots of alcohol abuse for them.

The success rates of the AA fellowship have been assessed at two points in time. Of those initially attracted to AA, a large proportion drop out—somewhere between 35 and 65 percent. Of those who become active members, 65 to 70 percent "improve to some extent, drinking less or not at all during A.A. participation" (Emrick, 1989:45). Membership in AA seems to be associated with relatively high abstinence rates, but with fairly typical improvement rates (Emrick, Lassen, & Edwards, 1977). It appears that AA is effective only with some 25 to 30 percent of the population with alcohol-related problems. AA, then, is a highly selective treatment source—attracting and holding those alcohol-troubled persons with severe alcohol problems who have high affiliative needs, conformist tendencies, proneness to guilt, and need for external controls (Trice & Roman, 1970; Ogborne & Glaser, 1981).

Unfortunately, the alternative type of organization has yet to be scrutinized by objective researchers. But subjective estimates of the number of groups and members have been put forward. SOS claims 1,000 groups with 2,000 members (Christopher, 1992); Hall (1990:1,46) has estimated that RR has meetings in 100 cities, "with perhaps two thousand members at any one time," and Hall (1990) estimated 5,000 members in 32 groups for WFS. Assuming that, like AA, there are dropouts and misfits for each type of group, these numbers must be sharply discounted. Nevertheless all three have demonstrated some staying power. SOS even publicizes itself as a demonstrated and proven alternative to AA. As yet no reliable data support this contention, but the fact that sizable numbers have been attracted to it suggests that it, or groups like it, are realistic contenders for some of AA's approximately 1 million members.

(SEE ALSO: *Alcoholism; Disease Concept of Alcoholism and Drug Addiction; Ethnic Issues and Cultural Relevance in Treatment; Women and Substance Abuse*)

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Therapeutic Communities Therapeutic communities (TCs) are drug-free residential treatment facilities for drug and/or alcohol addiction. TCs emerged in the 1960s as a self-help alternative to the conventional medical and psychiatric approaches being used at that time.

Most traditional TCs have similar features, including their organizational structure, staffing patterns, perspectives, rehabilitative regimes, and a twelve- to eighteen-month duration of stay. They differ greatly, however, in size (30-600 beds) and client demography. Most people entering TCs have used multiple drugs-including TOBACCO, MARIJUANA, ALCOHOL, OPIODS, pills, and, recently, COCAINE and CRACK-cocaine. In addition to their substance abuse, most TC clients also have a considerable degree of psychosocial dysfunction (Jainchill, 1994). In traditional TCs, 70 to 75 percent of clients are men, but admission for women is increasing. Most community-based TCs are integrated across gender, race/ethnicity, and age. Primary clinical staff are usually former substance abusers who were rehabilitated and trained. Other staff are the professionals who provide medical, mental health, vocational, educational, family-

More than 500 women from Synanon communities throughout California shaved their heads to symbolize acceptance of equal responsibility—with Synanon men—for the management and operation of the therapeutic communities. Oakland, February 27, 1975. (© Bettmann/CORBIS)

counseling, fiscal, administrative, and legal services.

Traditional TCs share a defining view of substance abuse as a deviant behavior, which may be attributed to psychological factors, poor family effectiveness, and, frequently, to socioeconomic disadvantage. Drug abuse is thus seen as a disorder of the whole person and recovery as a change in lifestyle and personal identity. As part of the recovery process, TCs seek to eliminate antisocial attitudes and activity, develop employable skills, and inculcate prosocial attitudes and values. This TC view of recovery is based upon several broad assumptions: the client's motivation to change, the client's main contribution to the change process (*self-help*), the mediation of this recovery through peer confrontation and sharing in groups (*mutual self-help*), the affirmation of socially responsible roles through a positive social network, and the understanding that treatment is a necessarily intense "episode" in a drug user's life.

Diverse elements and activities within the TC foster rehabilitative change. Junior, intermediate, and senior peer levels stratify the *community*, or the family. The TC's basic program elements, consisting of individual counseling and various group processes, make up the therapeutic and educative elements of the change process. The daily activities, including morning meetings, seminars, house meetings, and general meetings facilitate assimilation into the community as a *context for social learning*. Clients are oriented into the program during the *orientation-induction* stage. They progress through the *primary treatment* stage of the program by achieving plateaus of stable behavioral change. Client development reflects their changing relationship with the community, characterized as *compliance*, *conformity*, and *commitment*. Finally, *reentry* represents the final program stage where the skills needed in the greater social environment are fostered through increased self-management and decision making.

The effectiveness of the traditional long-term residential TC, as described here, has been well-documented (De Leon, 1997, 2000). Today, TCs include a wide range of programs serving diverse clients who use a variety of drugs and present complex social/psychological problems. Client differences, clinical requirements, and funding realities have all encouraged the development of modified residential TCs with shorter stays (3, 6 and 12 months) as well as TC-oriented day treatment and outpatient models. Most traditional TCs have expanded their social services or incorporated new interventions to address the needs of special populations such as adolescents, mothers and children, homeless, mentally ill chemical abusers, and prison inmates. In these modifications the cross-fertilization of personnel and methods from the traditional TC, mental health, and human services portends the evolution of a new therapeutic community.

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George De Leon

Traditional Dynamic Psychotherapy *Dynamic psychotherapy* is the term for the various psychological treatments, primarily talking treatments, intended to modify and ameliorate behaviors based on inner conflicts (e.g., "Should I study for the test or cheat?") and/or interpersonal conflicts (difficulties with others). These techniques range from those intended primarily to support individuals, lending them the therapist's strength or understanding ("If you do that you'll get in trouble. Have you thought of handling it this way?"), to helping patients reach their own understanding of the origins and implications of their behaviors. The application of these techniques to the treatment of alcoholics and substance abusers is supported by the high incidence of cooccurrence of psychiatric illness—in several studies, 70 percent—some of which may play a role in initiating or maintaining the behavior. It has been suggested that for some substance abusers, the use of illicit compounds is a misguided attempt at self-medication. Often, psychotherapy must be provided in conjunction with other treatments—pharmacologic, such as DISULFIRAM for alcoholics or METHADONE for HEROIN abusers; SELF-HELP groups, such as ALCOHOLICS ANONYMOUS; or family or group psychotherapy.

Psychotherapy is based on the assumption that the patient will think and talk about ideas and feelings rather than acting upon them. This may prove particularly difficult for substance abusers who often have little sense of what they feel, other than generalized pain, and who are used to action and immediate gratification. Therefore, treatment, particularly at the beginning, must take place within a structure that both supports and helps control impulsive behavior. Sometimes, treatment starts in a hospital or other residential setting; often, it is accompanied by regular drug testing. After the agreement to start therapy and setting goals, therapist and patient meet once to several times a week. As trust is developed between patient and therapist, the therapist can expect less lying and less denial of difficulties; treatment can, if indicated, begin to move from support toward expression of feelings—toward identification of conflicts and the understanding of their origins. Initially the therapist listens, struggling to understand the patient's inner experience and its meaning. The therapist then attempts to help patients to understand what they have presented, with appropriate changes and qualifications based on further information provided by the patient. Important issues to be explored in treatment include current relationships (with spouse, children, friends, coworkers), past relationships (with parents and other family), and the relationship within the treatment between the patient and the therapist. Often, the difficulties and distortions within this relationship mirror past and current relationships and may be used to help the patient see the nature and impact of the past on current behaviors.

Treating substance abusers can be frustrating for therapists; there are many slips with return to drug use, and patient behavior is often calculated to make the therapist angry and to give up. It is essential that therapists who make the attempt carefully monitor their own feelings so that they do not interfere with the treatment itself. It is also important to remember that when properly done, treatment can make the difference between suffering with chronic problems and successful adaptation. This is particularly true when substance abuse is accompanied by other psychiatric disease and/or disability.

(SEE ALSO: *Causes of Substance Abuse: Psychological (Psychoanalytic) Perspective; Disease Concept of Alcoholism and Drug Abuse; Epidemiology*)

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Twelve Steps, The The heart of the ALCOHOLICS ANONYMOUS (AA) is a program called the Twelve Steps set forth by cofounder Bill W. and his early followers. The Twelve Steps establish a suggested, unfolding process for becoming, and remaining, sober. The process begins with an admission of powerlessness over alcohol, along with unmanageable lives, and builds momentum gradually into a commitment to carry the AA program via the Twelve Steps to active alcoholics. Newcomers are not pressed to follow all the steps if they feel unwilling or unable to do so. This suggested policy seems to be followed. Thus, Madsen (1974) found that 41

of the 100 AA members he studied had gone through all the Twelve Steps. And Rudy (1986:10) reports that "in Mideast City, A.A. members talk about and emphasize steps 1, 2, 3, 4, and 12 more than others." This pragmatic view of the Twelve Steps can be heard in an AA saying—"Take the best and leave the rest." The steps are:

1. We admitted we were powerless over alcohol—that our lives had become unmanageable.
2. Came to believe that a Power greater than ourselves could restore us to sanity.
3. Made a decision to turn our will and our lives over to the care of God *as we understood Him*.
4. Made a searching and fearless moral inventory of ourselves.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. Were entirely ready to have God remove all these defects of character.
7. Humbly asked Him to remove our shortcomings.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.
10. Continued to take personal inventory, and when we were wrong, promptly admitted it.
11. Sought through prayer and meditation to improve our conscious contact with God *as we understood Him*, praying only for knowledge of His will for us and the power to carry that out.
12. Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics and to practice these principles in all our affairs [Alcoholics Anonymous World Services, 1976:59].

Step one meant for Bill W., the founder of AA, "the destruction of self centeredness" (Alcoholics Anonymous, 1939:16). In informal talk, AA members often urge everyone "to leave their egos at the door." Trice (1957:45) found that affiliation with AA was initially encouraged among those newcomers who reported that they had no willpower models among their friends or relatives for quitting alcohol abuse. Many observers have noted the strong tendency among alcoholics toward an "exaggerated belief in the ability to control their impulses, especially the impulse to use alcohol . . . that they are in charge of themselves, that they are autonomous and able to govern themselves" (Khantzian & Mack, 1989:74). AA teaches that until alcoholics accept the first step they will continue to believe a fiction—that they are clever enough and strong enough to control their drinking. In any event, by taking the first step, newcomers to AA dramatically change their conception of self from believing they can control their drinking to believing they cannot ever do so.

In step one, AA taps into the repentant role in U.S. tradition. Redemptive religions emphasize that one can correct a moral lapse, even one of long duration, by public admission of guilt and repentance. AA members can assume this repentant role, beginning with step one, and it becomes, along with the other steps, a social vehicle whereby they can reenter the community (Trice & Roman, 1970).

This role is strengthened by step two and step three, wherein alcoholics agree there is a power greater than themselves who will help and agree to turn their destiny over to this higher power as they conceive of it. In essence, members believe that one does not have to stand alone against alcohol abuse and the strains of life; AA offers the group itself and its collective notion of a higher power to help the powerless.

By accepting and executing step four and step five, AA members believe they are engaging in a realistic self-examination of the factors of fear, guilt, and resentment that cause their drinking. In step four, new members list all people they now resent or have resented in the past. Along with this list, newcomers note what they believe to be the substance of the resentment. Following this exercise, new members work out ways to try to alter conceptions of these resented persons. They also attempt an inventory of their own behaviors that have contributed to their fears, guilts, and resentments. In step five, alcoholics acknowledge these inventories to a higher power and confess them to some other individual. for example, a friend, pastor, therapist, or sponsor. Members believe that this moral inventory and its reduction in resentments enable them to live through emotional experiences that in the past were managed by the abuse of alcohol.

Steps six and seven are reinforcements of the changes produced by acting out steps four and five. In step six, members indicate and reaffirm a readiness to respond to help from a higher power. In step

The meeting room at the Wilson House in East Dorset, Vermont. The birthplace of Alcoholics Anonymous co-founder Bill Wilson serves as an inn and a gathering place for AA participants. (AP Photo/Craig Line)

seven, with as much humility as possible, members actually request that the higher power help them eliminate the inventory of "shortcomings" assembled by the member. In steps eight and nine, members seek to make further changes and reinforce past changes by providing restitution to those they have hurt in the past. Members list those actually harmed by their past behaviors and then do as much as they can to make amends and try to cancel out the harm caused. Most members agree that some amends might actually do harm to either themselves or others and caution against them. For example, the member might grievously damage a spouse by confessing in detail sexual infidelities. Step ten is a repetition and a reinforcement of steps four and five. In this step, members continue to "take my moral inventory" and admit their wrongs to themselves, others, and the Greater Power. Step eleven also acts as an implementer, but this time for step three, in which through meditation and prayer they again decide to turn over their willpower and their lives to a higher power.

Step twelve is the culmination of all these steps. Members are urged to carry their experiences and stories to active alcoholics in treatment centers, hospitals, even homes—in effect, to offer the redemptive model of AA sobriety to them. AA participants argue that, by becoming helpers, they help themselves at the same time and that they derive new commitments to the truths believed to be manifest in the other eleven steps. Furthermore, in twelfth-step work, there is a one-on-one, often a two-on-one (two AA members and one active alcoholic) meeting that often results in a sponsor-sponsoree relationship between a newcomer and older (in AA "birthdays") members. The group wisdom of AA teaches that new members are more likely to join during a crisis. Consequently, twelfth-step workers do not press for an admission of alcoholism during initial contacts. Rather, they try to be nonjudgmental, accepting, and reassuring, while nevertheless trying to help the prospect define the problem and what he or she will do about it. Members do, however, briefly describe their recovery via AA and invite the prospect to come to their meetings. If there is a positive response, they will promise to take the prospective member. According to Bales (1962:575), the sponsor-sponsoree relationship, along with the actual twelfth-step work itself, is "the heart of the therapeutic process" in AA.

The use of these steps is supported by basic assumptions: that intense self-examination and confession are cathartic; that alcoholics cannot control even moderate drinking and therefore are incapable of drinking at all. In other words, "once an alcoholic, always an alcoholic." According to the first step, "We admitted we were powerless over alcohol." The assumption of being powerless has been the focus of considerable controversy outside AA. The controversy centers around a follow-up study of 11,000 alcoholics whose drinking patterns were obtained 6 months and 18 months after experiencing one of a variety of treatment programs. The study, which contained numerous flaws (e.g., short follow-up time), showed that the majority of former alcoholics (who drank, on average, more than 8 ounces a day of ethanol [alcohol]) who had experienced a treatment program could drink moderately (2.5 ounces per day) at levels that many believe to be no problem (Armor, Polich, & Stambul, 1976).

A competing assumption is that ALCOHOLISM is a disease—that alcoholics suffer from an "allergy." This belief has also been controversial. An alternative has been the concept of the "problem drinker," the heavy drinker who gets into trouble, directly or indirectly, because of drinking alcohol. This bypasses the debate about alcoholism being a disease and about the amount drunk; it focuses instead on the "problem" correlates of drinking, that is, a role-impairment definition—financial problems and problems with family, police, friends, and neighbors. For example, Trice (1966:29) suggests that role impairment—such as job impairment—

would be one of the performance criteria for the definition of alcoholism: alcoholics differ from those around them because the performace of their adult roles becomes clearly impaired by their recurrent use of alcohol. In the United States, most alcoholics are very poor husbands and fathers or wives and mothers; on the job, they falter and disappoint their coworkers. In addition, their unreliable behavior makes for doubts and confusion in intimate friendships. In sum, drinking behavior that significantly damages the performance of basic roles is the phenomenon, and it is not necessarily a disease as AA claims. Calahan and Room (1974) reported significant correlations between heavy drinking and impairments in the performance of these elementary roles. Such a definition opens the door for other therapies that assume that moderate drinking is possible. It even assumes that there may be "spontaneous recovery," that no therapy of any kind may be involved in some recoveries.

Finally, it should be noted that the Twelve Steps of AA are, in many members' minds, inevitably associated with AA's Twelve Traditions, which are aphorisms for the maintenance and continuity of AA itself at the group level. Examples are: Tradition 1—Our common welfare should come first; personal recovery depends upon AA unity. Tradition 10—We need always maintain personal anonymity at level of press, radio, and films (Alcoholics Anonymous World Services, 1965).

(SEE ALSO: *Alcoholism; Disease Concept of Alcoholism and Drug Abuse; Rational Recovery; Sobriety; Treatment, History of; Vulnerability As Cause of Substance Abuse*)

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HARRISON M. TRICE

TRIPLICATE PRESCRIPTION An estimated hundreds of millions prescribed medication doses are diverted to the street each year. Triplicate-prescription programs were developed as an effort to decrease the diversion of prescription medications to illicit markets at a reduced cost of government investigation. States with such laws require physicians to write prescriptions on special triplicate forms for all Schedule II drugs, including narcotic analgesics, *Barbiturates*, and stimulants. In 1989 New York State passed legislation requiring triplicate prescribing for the *Benzodiazepines* (Schedule IV substances).

In triplicate prescribing, the physician keeps one copy of the prescription for five years and sends two copies with the patient to the pharmacist. The pharmacist keeps one copy and forwards the third to a specified state agency. Here the prescription is used to track the physician's prescribing practices and the patient's use of the controlled substances. With some exceptions, refills are not permitted for medications prescribed under this system.

Opponents of the triplicate-prescription system claim that although it is effective in decreasing diversion, it does so at the expense of some patients who are unjustly denied analgesics, anxiolytics, or sedative-hypnotics. The New York experience with

triplicate prescribing of benzodiazepines is often considered an example of this. Although benzodiazepine prescriptions were reduced by up to 60 percent, the number of prescriptions for the older and potentially more hazardous sedatives (such as MEPROBAMATE, methyprylon, ETHCHLORVYNOL, butalbital, and CHLORAL HYDRATE) increased markedly—in contrast to continued decreases in prescribing them in the rest of the United States. New York also required that any physician who prescribed an applicable drug for a long term period was required to report the patient as a drug "addict" or "habitual user," a notion the doctors found unsettling, especially when the drug was prescribed for maladies like cancer. The American Medical Association called the practice of triplicate prescriptions no less than "intimidation by regulatory and law enforcement agencies" (Report 4). It was viewed as so intimidating by New York doctors that 82 percent of the doctors surveyed in 1998 did not use the drug deemed most appropriate because of the observation of regulators.

In 1990 an attempt to federally legislate triplicate prescriptions for Schedule II medications for all states was unsuccessful in the House of Representatives, but efforts in some states, like Texas, to develop an electronic method of gathering the information may, and is likely to phase out the triplicate prescription for a tighter method of control there. In the State of New York, some effort is being made to remove the triplicate prescription system for a single official system that is intended to be less intimidating, although there is no evidence to how successful it will be.

(SEE ALSO: *Controls: Scheduled Drugs/Drug Schedules, U.S.; Iatrogenic Addiction; Legal Regulation of Drugs and Alcohol; Multidoctoring*)

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TWELVE STEPS, THE *See* Treatment: Twelve Step Facilitation

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UKAT: U. K. ALCOHOL TREATMENT TRIAL Everyone has a view about the nature and remedy of ADDICTION disorders, most likely because so many of these behaviors are visible in the public domain. Moreover they are common, so everyone knows someone who has one. As a result, things done in the name of treatment are sometimes based in science and sometimes they have more to do with folklore.

BACKGROUND

Many treatments of drinking problems have been presented over the years, some have endured due to the scientific evidence for their efficacy, but many have endured because of their popularity and in spite of the paucity of evidence for their effectiveness. A handbook of treatments shown to be effective, with ratings of their effectiveness from clinical trials as well as clinical descriptions of the method of their delivery was published during the nineties (Miller and Ilester, 1995): following on front this a large study was conducted in the U.S., which aimed to answer the question of whether one treatment was better than another for certain sorts of people (for example those who were socially stable, mentally ill, committed to entering treatment). Three treatments were compared in the attempt to answer this question and one of these involved encouraging clients to enter TWELVE STEP recovery programs in the form of ALCOHOLICS ANONYMOUS. The other two treatments were individually based cognitive and behavioral programs, the one focusing on behavior change and the other focusing on motivational change. All were found to be equally good at helping people with ALCOHOL dependence and problems to give up or reduce their drinking (Project MATC11, 1997).

In the U.K., treatment for problem drinking and dependence has taken a somewhat different course: the twelve step approach to recovery, while practiced in Alcoholics Anonymous, is not the most common form of or basis for treatment. Most treatment agencies in the U.K. are provided by the state and based in the cognitive behavioral approach. Moreover, the pursuit of moderation drinking goals for those with mild to moderate levels of alcohol dependence and an absence of alcohol related physical harm is common. Controlled drinking practice is prescribed for a minority of patients in most treatment agencies. A further consideration leading up to the present study was the growing recognition of the central role of the social network in supporting change in people with alcohol and drug problems. It has increasingly become common practice in the U.K. to recruit family members and significant others in the process of treatment (Orford, 1994).

In light of these considerations, the Medical Research Council in Britain agreed to fund a multicenter study of treatments for drinking problems. The Principal Investigators, a mixture of National Health Service and University based clinicians and researchers have collaboratively designed and im-

plemented the study. Results will be available in the year 2002.

DESIGN

The UKATT study compares two treatments to determine their relative effectiveness: Motivational Enhancement Therapy, adapted from the treatment studied in Project MATCH (Miller et al. 1992), is treatment which targets the motivation of the individual for drinking and for stopping or reducing drinking. Using feedback of objectives tests which are run as part of the assessment procedure, the therapist uses specific techniques which have been shown to enhance client motivation for change. The content of sessions is discussion of the negative consequences of continuing to drink in a harmful fashion and of the benefits of change. The treatment with which MET is compared is Social Behavior and Network Therapy whose focus is network support for change. Treatment sessions concentrate on the recruitment of social network whose members are than encouraged to modify their coping responses, improve lines of communication with the client, assist in the development of a relapse prevention program including identification of alternative activities and further sources of support. This treatment is adapted from a number of sources, primarily the Community Reinforcement Approach (Hunt and Azrin 1973) and Network Therapy (Galanter 1993). Both treatment protocols are specified in manual form and supervision of therapy, conducted by telephone and simultaneous viewing of videos, is designed to ensure manual adherence.

Clients for the study are recruited at the participating clinical centers, which are a combination of National Health Service and counseling agencies for the treatment of alcohol dependence. The clinical sites are in three different parts of the country: Yorkshire, South Wales, and the Midlands. The goal is to include as many as possible of the clients normally treated in these agencies and therefore the exclusion criteria have been kept to a minimum. People with active mental health problems or with addiction to a different treatment. Those younger than sixteen are not included: they have to be seen with a responsible adult other than the therapist and this would interfere with the individual nature of one of the treatments. Homeless people are not excluded provided that they can demonstrate that they have contact with someone in the community and are deemed possible to trace after treatment is complete, at three months and at one year. This requirement tends to exclude only those who are rootless and not in regular contact with any other agency. Also excluded are those who have already been treated as part of the study, the goal is to identify the effects of a single dose of the treatment rather than repeated doses.

Once they have been accepted for the study, clients are given a battery of tests and questionnaires designed to measure their drinking, related psychological and physical health, their use of health and other social services, their social networks, the extent to which there is drinking in these, their daily activities and whether these involve, their motivational stage of change and readiness for treatment. Clients are than randomly assigned to one of the two treatments which commences forthwith. Where there is a preliminary requirement for medically supervised withdrawal from alcohol or the need for another physical or social intervention, the above assessment will be deferred until this has been achieved.

An important goal of the study is to be pragmatic in order that the findings are relevant to the average treatment agency in the U.K. Relevance would mean that the treatments could be offered as the standard treatments for alcohol dependence and problem drinking by those staff normally recruited to work in such agencies. Therapists for the study are therefore existing employees at the clinical sites participating in the study. They are invited to express an interest in becoming a study therapist and to submit a resume and video recording of their practice for selection. If deemed suitable they are also randomly allocated to be trained in one or the other treatment. They are unable to select the treatment that they will be delivering in the study. The purpose of this procedure is to address the question of whether it is the case that any therapist with the above qualifications can be taught to deliver these treatments.

The therapists normally have professional qualifications in nursing, medicine, social work, occupational therapy or counseling and at least two years experience working with clients with drinking problems. They attend a three-day introduction to the therapy to which they have been assigned and this takes place at the national training center in Leeds in Yorkshire. Thereafter they are required to

practice and demonstrate competence by objective pre-determined criteria with at least two cases before proceeding to offer treatments in the study.

All therapy sessions are video recorded for the purpose of supervision, standardization of the delivery of treatment and evaluation of the extent to which these things have occurred.

OUTCOMES

The effectiveness of the two treatments is judged on the basis of the amount and frequency of drinking, the level of dependence and alcohol related problems in the study clients at three months and at twelve months. Measures of quality of life, economic activity, psychiatric morbidity and adjustment are also used to assess the value of the treatments.

Qualitative data on the process of therapy and the perceptions of the client and therapist of the active ingredients of the treatments are collected through a number of instruments administered at the end of the therapy sessions and the quality of the deliver of the treatment is separately assessed through independent ratings of therapist performance as demonstrated in the video recordings or practice. Integrity of the treatments as well as individual variations between therapists are identified through this method off evaluation.

CLINICAL IMPLICATIONS

There is an increasing demand for time limited treatments of alcohol dependence, for standardization and transparency of practice. While it is well recognized that there are therapist behaviors which are associated with improved outcomes in clients and these behaviors are often expressed in rather individual ways, it is also recognized that too often the question of the duration and nature of treatment is based upon the personal preference of the therapist and therefore subject to a variety of overt and convert influences. That therapists with a wide variety of backgrounds and different working practices can be taught to adhere to a manual and to deliver treatments in line with protocols has been demonstrated during this trial. How effective their interventions will be revealed in the results.

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U.S. DRUG POLICY *See* Anslinger, Harry G., and U.S. Drug policy; U.S. Government/U.S. Government Agencies

U.S. DRUG UNDERCOVER OPERATIONS *See* Drug Interdiction

UNITED NATIONS CONVENTION AGAINST ILLICIT TRAFFIC IN NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES, 1988 This international treaty was intended to extend and augment the agreements among the signatories that were contained in the 1961 Single Convention on Narcotic Drugs and the 1971 Convention on Psychotropic Substances. The 1988 Convention came into force in November 1990. By November 1994, 103 governments and the European Economic Community had been parties to the Convention. Included among the provisions are arrangements and agreements to legalize seizure of drug-related assets;

criminalize MONEY LAUNDERING; relax bank-secrecy rules; permit extradition of individuals charged with drug-law violations; control shipments of precursor and essential chemicals; continue to support CROP CONTROL and eradication; and share evidence with law enforcement and prosecuting agencies of governments who are party to the conventions.

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U.S. GOVERNMENT The following articles appear in this section:

Agencies in Drug Law Enforcement and Supply Control;
Agencies Supporting Substance Abuse Prevention and Treatment;
Agencies Supporting Substance Abuse Research;
Drug Policy Offices in the Executive Office of the President;
The Organization of U.S. Drug Policy

Agencies in Drug Law Enforcement and Supply Control So many agencies are involved in drug law-enforcement and supply-control activities that none are discussed here in detail. Except for the Drug Enforcement Agency (DEA), the order in which these descriptions appear is not necessarily related to the importance of an agency's role in the overall supply-control effort: Their functions frequently fit together like parts of an intricate puzzle.

The DEA was created in 1973 as a result of a reorganization that merged the activities and personnel from four federal drug law-enforcement programs into one agency within the Department of Justice (DOJ). John Bartels, Jr., was the first director. The offices and programs merged into DEA were the Bureau of Narcotics and Dangerous Drugs (BNDD), the Office for Drug Abuse Law Enforcement (ODALE), the Office for National Narcotic Intelligence, and U.S. Customs Service activities primarily directed to drug law enforcement. Since that time, DEA has been the lead federal agency for enforcement of drug laws.

DEA operates domestically and in foreign countries with the agreement of the government in each country. Its legal authority stems primarily from the CONTROLLED SUBSTANCES ACT and other laws directed at control of essential chemicals and precursors. DEA's efforts are directed against illicit drug production and high level drug-smuggling and drug-trafficking organizations operating within the United States or abroad. This agency is responsible for working with foreign governments to identify and disrupt the cultivation, processing, smuggling, and distribution of illicit substances, and the diversion of legally manufactured pharmaceuticals to illicit traffic in the United States. It maintains formal relationships with INTERPOL and the United Nations and works with them on international narcotics-control programs. The U.S. Department of State also has major responsibilities in working with foreign governments in this aspect of drug-traffic control. In carrying out these activities, DEA works closely with the state department, the Coast Guard, the Internal Revenue Service, and the U.S. Customs Service, and also with state and local law-enforcement agencies.

One of DEA's major domestic responsibilities is the enforcement of regulations concerning importation, manufacture, storage, and dispensing of all drugs scheduled under the Controlled Substances Act. Related to this function is the oversight, authorized by the Drug Treatment Act of 1974, of drug treatment programs using such drugs as LAAM or METHADONE (in METHADONE MAINTENANCE). DEA employs approximately 400 administration compliance officers to enforce regulations dealing with production and distribution of PRESCRIPTION DRUGS and supports a training program for narcotics officers at state and local levels. Virtually all state legislatures have passed a version of a prototype law, the Uniform Controlled Substances Act, which places legal CONTROLS on drugs at the state level similar to those at the federal level and establishes penalties under state law for violation of those laws. The Uniform Controlled Substances Act promotes uniformity in the way drugs are regulated, but individual states may schedule drugs not

included in federal schedules and may place any drug at a different level of scheduling.

Because of similar laws at the federal and state levels, and overlapping responsibilities among federal agencies, several law-enforcement agencies may have jurisdiction with respect to any single drug offense or group of offenders. The decision about which of the cooperating agencies takes the lead and under which law a case will be tried depends on mutual assessment among enforcement agencies and prosecutors of their capabilities and procedures, and of which jurisdiction is most likely to obtain a conviction, since rules of evidence and procedures differ between federal and local courts. Generally, federal agencies will focus on high level drug traffickers and networks. Local police are empowered only to enforce state and local drug laws and are not permitted to arrest people for breaking a federal drug law. Federal agents may not enforce state and local drug laws unless specifically authorized to do so. The DEA also has enforcement responsibilities under the Chemical Diversion and Trafficking Act of 1988. This law was designed to control the availability of chemicals and precursors used by clandestine laboratories to produce DESIGNER DRUGS or to further process plant products such as COCA leaf into pure COCAINE. Since at least thirty-seven states have passed similar laws, this is another area where federal and local enforcement agencies may have concurrent jurisdiction.

Other major responsibilities of DEA include investigation of major drug traffickers operating at interstate and international levels; personnel training; scientific research related to control or prevention of illicit trafficking; management of a narcotics intelligence system; seizure and forfeiture of assets derived from or traceable to illicit drug trafficking.

Forfeiture is the loss of ownership of property used in connection with drug-related criminal activity or property derived from its income. Such forfeiture was authorized in the Comprehensive Drug Prevention Control Act of 1970 and the Racketeering Influenced and Corrupt Organization (RICO) Statute also passed in 1970. In 1990, DEA seized assets valued at more than one billion dollars, although not all of this property was ultimately forfeited. Forfeited property is usually sold at public auction and the proceeds are used for government activities and shared with cooperating state governments. States have used these funds for drug treatment and education programs as well as for drug law enforcement. Some goes into a special forfeiture fund within the Office of National Drug Control Policy (ONDCP), which in turn transfers it to other federal agencies. For example, significant amounts were transferred to the Center for Substance Abuse Treatment (CSAT) to support treatment programs for pregnant addicts.

In addition to DEA, several other organizations within the DOJ and other Cabinet departments have responsibility in areas concerning drug laws and related matters. The Office of Justice Programs (OJP) in the DOJ, established by the Justice Assistance Act of 1984, contains several bureaus involved with these issues. Three having significant roles at the present time are the Bureau of Justice Assistance (BJA), the Bureau of Justice Statistics (BJS), and the National Institute of Justice (NIJ). The BJA provides technical and financial assistance to state and local government for controlling drug trafficking and violent crime. Under the terms of the Anti-Drug Abuse Act of 1988, states may apply for grants to assist them in enforcing local and state laws against offenses comparable to those included in the Controlled Substances Act. Part of the application for these "formula grant" funds requires devising a statewide anti-drug and -violent crime strategy. The BJS collects, analyzes, and disseminates information on crime, its victims, and its perpetrators. Its 1992 report, *Drugs, Crime, and the Justice System*, the source for much of the material in this article, may be the best written and most comprehensive summary on the topic ever produced by the federal government. BJS also manages the Drugs and Crime Data Center and Clearinghouse (tel. 1-800-666-3332), which gathers and evaluates existing data on drugs and the justice system. The NIJ is the major research and development entity within the DOJ. Among its other activities, NIJ evaluates the effectiveness of programs supported by BJA, such as community anti-drug initiatives, and SHOCK INCARCERATION AND BOOT-CAMP PRISONS.

Other drug law-enforcement entities within the DOJ include the Federal Bureau of Investigation (FBI); the U.S. Attorneys, who are the chief federal law-enforcement officers in their districts and are responsible for prosecuting cases in federal court; the Immigration and Naturalization Services (INS); and the U.S. Marshals Service, which manages the Asset Forfeiture Fund. The FBI became more prominently involved in antidrug activities when its

resources were significantly expanded in 1982 under President Ronald W. Reagan's reinvigoration of the "war on drugs." At that time it was given concurrent jurisdiction with DEA to investigate drug offenses, with the FBI concentrating primarily on drug trafficking by organized crime, electronic surveillance techniques, and drug-related financial activities such as investigations of international MONEY LAUNDERING.

Treasury Department agencies that play a role in controlling illicit drugs include the U.S. Customs Service, which stops and seizes illegal drugs as well as other contraband being smuggled into the United States; The Bureau of Alcohol, Tobacco, and Firearms (BATF), which investigates violations of laws dealing with weapons, particularly federal drug offenses invoking weapons; and the Internal Revenue Service (IRS), which assists in financial investigations, particularly money laundering.

Two agencies in the Department of Transportation, the Federal Aviation Administration (FAA) and the U.S. Coast Guard, are significantly involved in drug-control activities. The FAA uses its radar systems to assist in detecting smuggling by air; the Coast Guard is involved in interdiction of drugs being smuggled into the U.S. by water.

The Postal Inspection Service of the U.S. Postal Service is also involved in the antidrug effort. This agency enforces laws against using the mail to transport drug paraphernalia and illegal drugs.

The Department of State's role in international drug policy is to coordinate drug-control efforts with foreign governments. Within State, the Bureau of International Narcotics Matters (INM) is responsible for international antidrug policy. This bureau provides technical assistance, money, and equipment to foreign governments for local law enforcement, transportation of personnel, and equipment for crop eradication. It also monitors worldwide drug production. Each U.S. Embassy abroad has a designated narcotics coordinator. In countries where there is considerable drug-related activity, there may be an entire narcotics-assistance section at the embassy. The state department also helps selected foreign governments with demand-reduction activities. Helping countries adversely affected economically by drug CROP CONTROL and eradication is a responsibility of the Agency for International Development. The U.S. Information Agency provides information about drug policy and relevant laws to U.S. officials serving in foreign countries.

The Department of Defense (DOD) is involved in detecting and monitoring aircraft and ships that might be involved in smuggling drugs into the United States. Until the 1980s, the military was prohibited from exercising police power over U.S. civilians by the Possae Comitatus Act of 1876. Changes in the act allow the military to share resources with civilian law-enforcement agencies, although military personnel are still not permitted to arrest civilians. The National Guard also assists federal agencies in border surveillance and in marijuana eradication.

Eleven agencies are involved in the Intelligence Center at El Paso, Texas (EPIC), operated by the DEA. EPIC is designed to target, track, and interdict drugs, aliens, and weapons moving across U.S. borders. The participating agencies, in addition to the DEA, are the Federal Bureau of Investigation (FBI); the Immigration and Naturalization Service (INS); the Customs Service; the U.S. Marshals Service; the U.S. Coast Guard; the Federal Aviation Administration (FAA); the Secret Service; the Department of State Diplomatic Service; the Bureau of Alcohol, Tobacco and Firearms (BATF); and the Internal Revenue Service (IRS). There is also a Counternarcotics Center developed by the Central Intelligence Agency (CIA) that coordinates international intelligence on narcotics trafficking. This effort involves personnel from the National Security Agency (NSA), the Customs Service, the DEA, and the Coast Guard.

(SEE ALSO: *Crime and Drugs; Drug Interdiction; International Drug Supply Systems; Terrorism and Drugs*)

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Agencies Supporting Substance Abuse Prevention and Treatment Within the U.S. Department of Health and Human Services (DHHS), originally established in 1953 as the Department of Health, Education, and Welfare (DHEW), a number of Public Health Service (PHS) agencies have been involved in reducing drug abuse. From 1974 to 1992, many demand-reduction activities have related to increasing, through research, the scientific foundations for a better understanding of how drugs of abuse interact with individuals, so as to prevent drug abuse and effectively treat those who do abuse drugs. Included among these agencies are the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA), both components of the National Institutes of Health (NIH), as well as the Center for Substance Abuse Prevention (CSAP) and the Center for Substance Abuse Treatment (CSAT), components of the Substance Abuse and Mental Health Services Administration (SAMHSA). In addition, the Health Resources and Services Administration (HRSA) and the National Institute of Child Health and Human Development (NICHD), another NIH component, play a role in the department's anti-drug abuse mission. Although not all inclusive, the chart below shows the organizational hierarchy of these agencies within the department.

From its creation in 1974 by statute, the National Institute on Drug Abuse has conducted RESEARCH on drugs of abuse and their effects on individuals. In its early days, NIDA supported PREVENTION and TREATMENT programs and conducted clinical training programs for professional health-care workers (particularly in schools of medicine, nursing, and social work) and counselor and other paraprofessional training. With the advent of the Alcohol and Drug Abuse and Mental Health Services block grant, enacted into statute in 1981, the direct provision of treatment and prevention services became a state responsibility. Enactment of the block grant that is currently administered within SAMHSA served to refocus NIDA's role on the generation of knowledge through scientific research, so that more could be learned about strategies and programs to help prevent and treat drug abuse.

The National Institute on Alcohol Abuse and Alcoholism (NIAAA) conducts research on alcohol abuse and alcoholism. Because a comprehensive approach to prevention and treatment of drug abuse requires attention to alcohol as well as to illicit drugs, and because individuals who abuse illicit drugs often abuse alcohol as well, the research programs of NIDA and NIAAA are symbiotic. Furthermore, the genetic, environmental, and social influences important to the initiation of drug and alcohol use are similar, and research in one area suggests researchable hypotheses in the other.

The Center for Substance Abuse Prevention (CSAP), established in 1986 as the Office for Substance Abuse Prevention (OSAP), has led the nation's efforts to prevent alcohol and other drug use, with a special emphasis on youth and FAMILIES at particularly high risk for drug abuse. Youth considered to be at high risk include school DROPOUTS, economically disadvantaged youth, or children of parents who abuse drugs or alcohol or who are at high risk of becoming drug or alcohol abusers. CSAP administers a variety of programs, including Prevention demonstration grants targeting youth at high risk and projects for pregnant and postpartum women and their infants.

The Center for Substance Abuse Treatment (CSAT), formerly the Office of Treatment Improvement (OTI), was established administratively in 1990 with a focus on improving treatment services and expanding the capacity for delilvering treatment services. In addition to administering the Alcohol and Drug Abuse block grant, CSAT administers a number of demonstration grant programs such as the Target Cities, Critical Populations, and Criminal Justice treatment programs.

Drug and alcohol abuse are complex behaviors that often result in a multitude of adverse consequences. Thus, to understand them necessitates multifaceted, often crosscutting areas of research. Because many individuals who suffer from alcohol or drug abuse also suffer from mental illness, NIAAA and NIDA, as well as the National Institute of Mental Health (NIMH) of the NIH, are engaged in initiatives to learn more about individuals who are dually diagnosed.

Acquired immunodeficiency syndrome (AIDS) has become a growing health program among intravenous drug users, and an increased risk of human immunodeficiency virus (HIV) infection in those who share drug paraphernalia with other drug users has been clearly demonstrated (Chaisson et al., 1987; Schoenbaum et al., 1989). Accordingly, NIDA collaborates with the Centers for Disease Control (CDC) on AIDS prevention programs and with the National Institute of Allergy and Infectious Diseases (NIAID) to provide HIV therapeutics to intravenous drug abusers with HIV.

The study of maternal and fetal effects of drug abuse is another high-priority focus within the department. Research and demonstration programs have been undertaken by NIDA and CSAP, and the NICHD is also conducting studies in this area.

Recent research has shown that the most effective treatment for drug abusers is a comprehensive array of services that address not only their drug-abuse problems but also other health problems and their potential need for education and vocational rehabilitation, as well as a host of ancillary services. Accordingly, NIDA, the centers within SAMHSA, and HRSA are exploring the effectiveness of providing a comprehensive range of drug-abuse and other primary-care services, both in drug-abuse settings and primary-care settings.

Besides the DHHS, there are many other agencies involved in prevention and treatment efforts. For example, the Food and Drug Administration (FDA), plays a determining role in deciding when new pharmacological treatment agents can be marketed for clinical use, and it is one of the key agencies setting policies and standards for the use of OPIOID drugs in the treatment of opioid dependence. Both the Department of Education and the Department of Justice (through the Drug Enforcement Agency [DEA]) have significant programs aimed at prevention; the Department of Veterans Affairs and the Department of Defense (U.S. MILITARY) have also made major commitments to treatment.

(SEE ALSO: *Education and Prevention; Prevention Movement; Research; Substance Abuse and HIV/AIDS*)

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Agencies Supporting Substance Abuse Research In the United States, federal support of drug-abuse research began in the 1920s with the work of Lawrence Kolb. It became more formalized with the establishment of the Addiction Research Center in 1935. A small research unit was formed with only fifteen employees in a U.S. Public Health Service Hospital in Lexington, Kentucky, by 1944. The Addiction Research Center was designed for federal prisoners who were narcotics addicts. This research group became part of the National Institute of Mental Health (NIMH) in 1948, the year the institute was established. In 1979, the Addiction Research Center moved to Baltimore, Maryland, and became the in-house (intramural) research program of the National Institute on Drug Abuse (NIDA), which was itself established by Congress in 1974.

In the early 1990s, it was estimated that NIDA funded 88 percent of the drug-abuse research in the world. In 1992, the NIDA budget for the almost 1,000 research grants awarded to universities and other research institutions (i.e., extramural research) totaled 338 million dollars. NIDA's 1992 intramural research budget for the Addiction Research Center was 24 million dollars. The research thus funded includes studies in practically every basic and clinical science, both biomedical and social. The National Institute on Alcohol Abuse and Alcoholism (NIAAA), established in 1970, conducts parallel efforts in the area of alcohol-abuse research. In 1992, its budget for extramural research was 155 million dollars for over 600 research projects. NIAAA's intramural research arm, located in Bethesda, Maryland, had a budget of nearly 20 million dollars.

Both NIDA and NIAAA became part of the National Institutes of Health (NIH) in October 1992.

They had previously been part of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA), which included both research and services components. By separating these two components, the Congress indicated its intention to give proper emphasis to both. Now treatment and prevention services for alcohol and drug abuse are under the direction of the Substance Abuse and Mental Health Services Administration (SAMHSA).

NIDA and NIAAA are the two largest federal research institutes dedicated to drug abuse and alcohol research, but there are many other agencies that have a stake in these areas. They include other institutes in the National Institutes of Health; for example, the National Institute of Child Health and Development centers its research on the effects of drugs and alcohol on fetal development and on the consequences for the neonate of exposure to drugs and alcohol during pregnancy. The National Institute of Mental Health conducts research on the high coincidence of mental illness and substance-abuse disorders. Some of the other institutes have similarly targeted interests, as, for example, the National Cancer Institute, which played an important role in support of research on tobacco dependence and the adverse health effects of tobacco.

Other parts of the Public Health Service also play a role in substance abuse research. The Centers for Disease Control (CDC) use their epidemiological expertise to resolve certain questions about the nature and extent of the abuse of drugs and alcohol. The Agency for Health Care Policy and Research conducts research on the costs associated with medical care and health insurance for drug and alcohol abusers seeking treatment.

Beyond the Public Health Service and the Department of Health and Human Services, many other federal agencies and departments are concerned with and conduct research on the social problems caused by drug and alcohol abuse: the departments of education, labor, transportation, treasury, justice, state, veterans affairs and even defense—each has a stake in drug-abuse research. The Department of Education is concerned primarily with drug and alcohol prevention; the departments of labor and transportation with workplace performance impaired by drugs and alcohol.

The Department of Veterans Affairs has played an important role in both basic and clinical research. Some of the most important work on the treatment of opioid dependence and on alcoholism and the toxic effects of alcohol have been conducted by researchers based at Veterans Administration (VA) hospitals and funded in part by research funds from the Department of Veterans Affairs. Other federal agencies have a regulatory role in certain types of drug-abuse research. Many of the drugs that are studied in animals and volunteer human subjects are included under the CONTROLLED SUBSTANCES ACT of 1970. In order to obtain and store the drugs, researchers must be properly registered with the Drug Enforcement Agency (DEA). The DEA is also responsible for ensuring that the drugs are properly stored and the records of their use are properly kept by the researchers. In addition, researchers who are interested in studying any drug not yet approved for clinical use, or studying an approved drug for a new use (such as using the antihypertensive agent, CLONIDINE, to control alcohol, tobacco, or opioid withdrawal), must obtain permission obtaining an Investigational New Drug (IND) authorization from the Food and Drug Administration (FDA). Further, when a new agent seems promising, a sponsor (usually a pharmaceutical company) must submit the data supporting its safety and effectiveness to the FDA before it can be approved for marketing and general use.

Both the Department of Justice and the Department of the Treasury are concerned with law enforcement issues surrounding drug and alcohol use, and they have funded research on detection of clandestine laboratories and the nature of DESIGNER DRUGS. The 1994 National Strategy showed that of the entire federal drug-abuse research budget, some 500 million dollars, approximately 67 million was allocated to domestic law-enforcement research.

The Department of State and the Department of Defense are involved in matters relating to international narcotics control. The U.S. Information Agency (USIA) and the Agency for International Development sponsor small drug-abuse research programs, mostly epidemiological in nature, in various countries. The Office of National Drug Control Policy (ONDCP) was given the mandate by Congress in 1988 to coordinate the federal antidrug-abuse effort. It does this through its budgetary oversight and through the Research, Data, and Evaluation Committee. The ONDCP for several years has had a Science and Technology subcommittee, which oversees the Counter-Drug Technol-

ogy Assessment Center (CTAC). CTAC is involved in both medical research and supply-related counter-drug technology development. The latter includes activities such as the use of satellites for wide area surveillance, non-intrusive inspections, and development of information systems to permit sharing of data among criminal justice data bases. All of these policy-related organizations rely on facts based on the biomedical, epidemiological, and behavioral research funded by NIDA, NIAAA, and NIMH.

(SEE ALSO: *Addiction Research Unit (U.K.); Education and Prevention; Prevention Movement; Wikler's Pharmacologic Theory of Drug Addiction*)

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Drug Policy Offices in the Executive Office of the President The Executive Office of the President (EOP) is an administrative group of key advisors and agencies supporting the president and the White House staff. Changes to the organization and functions of the EOP reflect the priorities and interests of each president. The organization of the EOP can be modified by executive order, by reorganization plan (when authorized), or by legislation.

Since 1970, several drug-policy activities have been established in the EOP. The list includes three separate EOP agencies, authorized and funded by statute, and three drug-policy offices, authorized by the president and located within a larger EOP agency. The drug-policy offices are listed immediately below, followed by a general description of each's activity.

Separate Agencies. Special Action Office for Drug Abuse Prevention (SAODAP), 1971–1975. Office of Drug Abuse Policy (ODAP), 1977–1978. Office of National Drug Control Policy (ONDCP), 1989–present.

Offices. Federal Drug Management (Office of Management & Budget), 1973–1977. Drug Policy Office (Domestic Policy Staff), 1978–1980. Drug Abuse Policy Office (Office of Policy Development), 1981–1989.

SPECIAL ACTION OFFICE FOR DRUG ABUSE PREVENTION (SAODAP)

A separate agency in the EOP from 1971 to 1975, SAODAP was responsible for providing leadership and coordination of all federal drug-abuse prevention activities (demand related) and to coordinate the demand-related activities with the supply-related efforts of law enforcement agencies.

Directors. Jerome H. Jaffe, 1971–1973 (also Consultant to the President for Narcotics and Dangerous Drugs) Robert L. Dupont 1973–1975.

Authorization and Role. Established by President Richard M. Nixon (E. O. 11599, June 17, 1971). Legislative authorization: Public Law 92-255, March 21, 1972; the "Drug Abuse Office and Treatment Act of 1972." The director reported to the president, working through the Domestic Council and the White House staff. SAODAP had a staff of over 100 and an annual budget of approximately $50 million. About 50 percent of the budget was in a "Special Fund for Drug Abuse" to be transferred to other federal agencies as an incentive to develop more effective prevention programs.

SAODAP provided oversight of all categories of "Demand Reduction" functions and made recommendations to the Office of Management and Budget (OMB) on funding for drug-abuse programs. SAODAP published three federal strategies under the auspices of the relatively inactive Strategy Council on Drug Abuse.

When the authorizing statute expired on June 30, 1975, SAODAP's treatment, rehabilitation, and prevention functions were moved from the EOP to the National Institute on Drug Abuse in the Department of Health, Education, and Welfare.

FEDERAL DRUG MANAGEMENT, OFFICE OF MANAGEMENT AND BUDGET

Opened in 1973 as a unique office within OMB, Federal Drug Management (FDM) was designed to manage federal activities directed at illegal drugs during a time of rapid expansion and major reorga-

nization. FDM continued in operation until early 1977.

FDM Chiefs. Walter C. Minnick, 1973–1974 Edward E. Johnson, 1974–1977.

Authorization and Role. Established by OMB memorandum, the authority of the staff office and the budget for operating expenses were derived from OMB. Initially, FDM was responsible for coordinating the implementation of drug policy, resolving interagency disputes, assisting drug agencies with reorganization and management, and working closely with other inter-agency drug-coordinating structures. In August 1974, FDM's budget and management responsibilities reverted to the normal OMB divisions and FDM continued to provide Executive Office oversight of the domestic and international drug abuse programs, interdepartmental coordination, and staff support to the cabinet councils on drug abuse.

Located in the Old Executive Office Building, FDM's five-person staff functioned with little public visibility. Working with other OMB staff, FDM guided the implementation of Reorganization Plan No. 2 of 1973, including union negotiations. FDM continued through the Ford Administration, providing staff assistance and policy advice to OMB, the Domestic Council, and the National Security Council. FDM was eliminated in early 1977 during the transition to the Carter Administration.

OFFICE OF DRUG ABUSE POLICY (ODAP)

In March 1976, Congress authorized the Office of Drug Abuse Policy, located in the EOP and intended to be the successor agency to SAODAP. President Gerald R. Ford did not activate the new agency, choosing instead to continue with the existing FDM staff. President Jimmy Carter opened ODAP in March of 1977 and abolished it one year later. The director's office was located in the West Wing of the White House and the staff offices were in the Old Executive Office Building.

Director. Dr. Peter G. Bourne, 1977–1978 (also Special Assistant to the President for Health Issues).

Authorization and Role. Congress established ODAP in Public Law 94-237 and provided an annual budget of $1.2 million. The director was the principal advisor to the president on policies, objectives, and priorities for federal drug-abuse functions. The director coordinated the performance of drug-abuse functions by federal departments and agencies.

ODAP, with a staff of approximately fifteen, conducted a comprehensive set of drug-policy reviews using interagency study teams. The director and staff sought a close cooperative relationship with Congress and testified when requested before various congressional committes. The director was required to prepare an annual report on the activities of ODAP and to oversee the preparation of a drug-abuse strategy.

In mid-1977, the President's Reorganization Project prepared a reorganization of the EOP that included abolishing ODAP. Congress objected to the loss of ODAP. After spirited congressional hearings emphasizing the continuing need for executive coordination of the drug program, ODAP was abolished in March 1978 and its responsibilities transferred to the Domestic Policy Staff.

Bibliography of Associated Major Policy Publications (ODAP):

- U.S. Executive Office of the President. Office of Drug Abuse Policy. *Border Management and Interdiction—An Interagency Review, September 1977.*
- U.S. Executive Office of the President. Office of Drug Abuse Policy. *Supply Control: Drug Law Enforcement—An Interagency Review*, December 1977.
- U.S. Executive Office of the President. Office of Drug Abuse Policy. *International Narcotics Control Policy, March 1978.*
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- U.S. Executive Office of the President. Office of Drug Abuse Policy. *Drug Abuse Assessment in the Department of Defense: A Policy Review, November 1977.*
- U.S. Executive Office of the President. Office of Drug Abuse Policy. *1978 Annual Report*. Washington, DC: Government Printing Office, 1978.

DRUG POLICY OFFICE (DPO), DOMESTIC POLICY STAFF

The Drug Policy Office (DPO) opened March 26, 1978, as an integral part of the White House Domestic Policy Staff. Six people were transferred from ODAP, and the DPO provided direction and oversight of federal drug-program activities through 1980.

Director. Lee I. Dogoloff, 1978–1980 (Associate Director for Drug Policy in the Domestic Policy Staff).

Authorization and Role. Reorganization Plan No. 1 of 1977 transferred the ODAP responsibilities to the Domestic Policy Staff in the EOP. President Carter signed Executive Order No. 12133 on May 9, 1979, formally designating the associate director for Drug Policy in the Domestic Policy Staff as

Primarily responsible for assisting the President in the performance of all those functions transferred from the Office of Drug Abuse Policy and its Director . . . in formulating policy for and in coordinating and overseeing, international as well as domestic drug abuse functions by all Executive Agencies.

DPO continued to report to Dr. Bourne as special assistant to the president for health issues. On numerous occasions, the associate director testified before Congress on drug-policy matters.

DPO published a 1979 federal strategy under the auspices of the Strategy Council on Drug Abuse, an annual report in 1980, and an annual budget crosscut of all drug-abuse prevention and control activities. Both the Domestic Policy Staff and DPO were eliminated during the transition to the Reagan Administration.

DRUG ABUSE POLICY OFFICE (DAPO), OFFICE OF POLICY DEVELOPMENT

Similar in organization and responsibilities to the preceding DPO, the Drug Abuse Policy Office (DAPO) was the principal EOP drug-abuse staff during the eight years of President Ronald W. Reagan's administration. In 1981, DAPO was established within the White House Office of Policy Development.

Directors. Carlton E. Turner, 1981–1986 (also Special Assistant to the President; promoted in March 1985 to Deputy assistant to the President).

Dr. Donald Ian MacDonald, 1987–1989, (Special Assistant to the President; promoted in August 1988 to Deputy Assistant to the President).

Authorization and Role. The statutory basis for the office (21 USC 1111 & 1112) required the president to establish a system to assist with drug abuse policy functions and to designate a single officer to direct the drug functions. Presidential Executive Order 12368, signed on June 24, 1982, assigned the Office of Policy Development (OPD) to assist the president with drug-abuse policy functions, including international and domestic drug-abuse functions by all executive agencies. The director of ODAP was responsible for advising the president on drug-abuse matters and assisting Nancy D. Reagan and her staff in developing the First Lady's drug-abuse prevention program.

The director and staff developed policies regarding all aspects of drug abuse, including drug law enforcement, international control, and health-related prevention and treatment activities for both government and the private sector. DAPO coordinated the development and publication of 1982 and 1984 drug-abuse strategies.

In October 1984, Public Law 98-473, which created the National Drug Enforcement Policy Board to oversee drug law enforcement, also included a new statutory duty for DAPO; "to insure coordination between the National Drug Enforcement Policy Board and the health issues associated with drug abuse."

In March 1987, Executive Order 12590 established a National Drug Policy Board (NDPB) to assist the president in formulating all drug-abuse policy, replacing the director of DAPO in that role. The new executive order made the director a member of the NDPB and assigned DAPO to assist both the president and the NDPB in the performance of drug-policy functions. The DAPO director assisted in developing the health-related aspects of the national drug strategy published in the board's 1988 report *Toward a Drug-Free America—The National Drug Strategy and Implementation Plans*.

DAPO was terminated early in the administration of President George H. Bush by Public Law 100-690, which created the Office of National Drug Control Policy.

OFFICE OF NATIONAL DRUG CONTROL POLICY (ONDCP)

In January 1989, the Office of National Drug Control Policy (ONDCP) was established as an agency in the EOP to oversee all national drug-control functions and to advise the president on drug-control matters. Functioning as the so-called drug czar, the director of ONDCP had the broadest combination of staff, funding, and authority of any previous EOP drug agency or office.

Directors. William J. Bennett, 1989–1990. Bob Martinez, 1991–1992. Lee P. Brown 1993–1996. General Barry R. McCaffrey 1996–present.

Authorization and Role. Established by Public Law 100-690 (21 USC 1504) with a five-year authorization, ONDCP had a staff of approximately 130 and a Fiscal Year 1993 budget of $59 million for salaries, expenses, and support for High Intensity Drug Trafficking Areas. The fiscal year 1994 budget request reduced the ONDCP staff to 25 positions. In 1996, with the appointment of retired Army General Barry R. McCaffrey, President Clinton planned to increase the ONDCP staff to 150 positions. The director controls a Special Forfeiture Fund with over $75 million appropriated in Fiscal Year 1993 to provide added funding for high-priority drug-control programs.

ONDCP was responsible for national drug control policies, objectives and priorities, and annual strategy, and a consolidated budget. ONDCP was also required to make recommendations to the president regarding changes in the organization, management, personnel, and budgets of the federal departments and agencies engaged in the antidrug effort.

ONDCP was required to promulgate an annual national drug control strategy and to coordinate and oversee the implementation of the strategy. The director had to consult with and assist state and local governments regarding drug-control matters.

More recently, the ONDCP has set its agenda, at least in part, toward international drug control policies. The current director, Gen. Barry McCaffrey, has expended significant effort working with the Mexican government to thwart drug trafficking in Mexico. According to an article in *Insight on the News*, 70 percent of all the cocaine that enters the United States comes via Mexico (Dettmer, 1997). Additionally, McCaffrey has pushed the U.S. Congress to approve an anti-drug supplemental package of more than a billion dollars to help aid the Colombian government in its drug interdiction efforts. According to McCaffrey, as quoted in a Press Release from the ONDCP, "Now ninety percent of the cocain on our streets and two-thirds of the heroin seized in the U.S. originates in or passes through Colombia." That package was passed by the House of Representatives in March, 2000. (ONDCP, Press Release, 2000).

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy*)

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The Organization of U.S. Drug Policy

Reducing drug abuse has been a priority for the U.S. government since the late 1960s, with continuing expansion of management attention and federal budgets. In 1969, eight agencies and four cabinet departments received drug-program funding; in 1975, seventeen agencies in seven cabinet departments were included; the federal drug control program for 1993 involves forty-five agencies and twelve cabinet departments. In 1969, the total budtet for federal drug-abuse programs was $81 million; for 2000, the budget was approximately $17.8 billion.

WHY IS IT DIFFICULT TO ORGANIZE DRUG POLICY?

Drug-policy issues are complex. The organization for drug-policy development must be able to handle the complexity of the drug problem and of the government's response.

Illegal drugs come from both international and domestic sources; they include a wide variety of substances; they involve many different forms of transportation, geographical areas, criminal activities, use patterns, and social effects. All these elements are dynamic—constantly adjusting to changes in supply and demand. Drug traffickers and continuing users immediately react to drug law enforcement pressures by shifting to areas or techniques that have less risk. Federal managers and policymakers must recognize the complex changes (and the probable causes) and be capable of adjusting the federal effort promptly and effectively.

National leadership, including an accepted strategy and a process to ensure implementation, is essential to real progress in eliminating illegal drugs and their use. The president must have congressional cooperation in authorizing and funding the strategy. The cabinet departments and agencies must be willing participants, with an effective procedure for resolving interdepartmental differences of opinion.

The complex drug issue, however, does not fit the usual organization of the federal government: There is no cabinet department with line authority over all drug-program resources; and only a few federal agencies are organized around a single drug-related function (e.g., the Drug Enforcement Agency and the National Institute on Drug Abuse). Most of the drug control agencies and all the departments have various other important roles, so they must balance their drug and nondrug responsibilities.

Every step in the policy-determination and -implementation process is complex and subject to bureaucratic, political, and technical differences of opinion. Two of the most difficult aspects of the drug problem are (1) seeking agreement on the extent and nature of the problem, and (2) attempting to assess the impact of the federal effort on the ever changing situation.

During the past two decades, the federal organization for determining drug policy and implementing drug programs has expanded to involve a significant portion of the federal government. The following list of cabinet departments and agencies that execute drug policy reflects the breadth of implementation activities.

NATIONAL DRUG CONTROL AGENCIES

The 1992 National Drug Control Strategy lists over forty-five agencies and several activities in twelve cabinet departments involved in drug-control efforts:

- ACTION
- Agency for International Development
- Department of Agriculture
  - Agricultural Research Service
  - U.S. Forest Service
- Central Intelligence Agency
- Department of Defense
- Department of Education
- Department of Health and Human Services
  - Administration for Children and Families
  - Alcohol, Drug Abuse, and Mental Health Administration (includes the National Institute of Mental Health, the National Institute on Drug Abuse, the Na-

- tional Institute on Alcohol Abuse and Alcoholism, the Office for Substance Abuse Prevention and the Office for Treatment Improvement)
  - Centers for Disease Control
  - Food and Drug Administration
  - Health Care Financing Administration
  - Indian Health Service
- Department of Housing and Urban Development
- Department of the Interior
  - Bureau of Indian Affairs
  - Bureau of Land Management
  - Fish and Wildlife Service
  - National Park Service
  - Office of Territorial and International Affairs
- The Judiciary
- Department of Justice
  - Assets Forfeiture Fund
  - U.S. Attorneys
  - Bureau of Prisons
  - Criminal Division
  - Drug Enforcement Administration
  - Federal Bureau of Investigation
  - Immigration and Naturalization Service
  - INTERPOL/U.S. National Central Bureau
  - U.S. Marshals Service
  - Office of Justice Programs
  - Organized Crime Drug Enforcement Task Forces
  - Support of U.S. Prisoners
  - Tax Division
- Department of Labor
- Office of National Drug Control Policy
  - Counter-Narcotics Technology Assessment Center
  - High Intensity Drug Trafficking Areas
  - Special Forfeiture Fund
- Small Business Administration
- Department of State
  - Bureau of International Narcotics Matters
  - Bureau of Politico/Military Affairs
  - Diplomatic and Consular Service
- Department of Transportation
  - U.S. Coast Guard
  - Federal Aviation Administration
  - National Highway Traffic Safety Administration
- Department of the Treasury
  - Bureau of Alcohol, Tobacco, and Firearms
  - U.S. Customs Service
  - Federal Law Enforcement Training Center
  - Financial Crimes Enforcement Network
  - Internal Revenue Service
  - U.S. Secret Service
- U.S. Information Agency
- Department of Veterans Affairs
- Weed and Seed Program

COORDINATING MECHANISM FOR DRUG POLICY

In reviewing historical drug-policy coordinating systems since the late 1960s, each system reflects a complex set of considerations. Two elements seem to differentiate between the various approaches: Either a drug-policy adviser and supporting drug staff is fully integrated into the regular policy processes at the White House, or a high-priority cabinet-level activity or agency is established with its own special policy process but with less participation in White House internal staff activity.

Each president selects his own White House staff and establishes a policy-development process to meet his needs. Therefore, any policy-coordinating mechanism that is closely related to a president must be expected to change with each new administration.

Congress has repeatedly attempted to establish a "drug czar" in the Executive Office of the President (EOP)—one person to oversee drug policy and to advise both the president and Congress.

HISTORY

A chronological summary of drug-policy coordinating mechanism is presented here, beginning with 1971—first from the perspective of the Executive Branch, then from the perspective of Congress.

Executive Drug Policy 1971–1976. On the demand side, President Richard M. Nixon created the Special Action Office for Drug Abuse Prevention (SAODAP) in the EOP in June 1971—to lead and coordinate all federal drug-abuse prevention activities. The first director, Dr. Jerome H. Jaffe, was given the added title of Consultant to the President for Narcotics and Dangerous Drugs. SAODAP then monitored the annual budget process and prepared budget analyses of all federal drug-abuse programs, by agency and by activity.

Also in 1971, President Nixon called for "an all out global war on the international drug traffic" (1973 Federal Strategy, p. 112), and his organization for policy reflected the international perspective. International efforts were coordinated by the Cabinet Committee on International Narcotics Control (CCINC), chaired by the secretary of state. Established in August 1971, CCINC was responsible for developing a strategy to stop the flow of illegal narcotics into the United States and to coordinate federal efforts to implement that strategy. Domestic drug-law enforcement had a high priority within the normal cabinet-management system.

In January 1972, President Nixon created the Office of Drug Abuse Law Enforcement (ODALE) in the Department of Justice and gave the ODALE director, Myles J. Ambrose, the added title of Consultant to the President for Drug Abuse Law Enforcement. The directors of both SAODAP and ODALE had a policyoversight role in advising the president.

The 1972 legislation authorizing SAODAP also created the Strategy Council on Drug Abuse (known as "The Strategy Council") and directed the "development and promulgation of a comprehensive, coordinated, long-term Federal strategy for all drug abuse prevention and drug traffic functions conducted, sponsored, or supported by the Federal government." The cabinet-level strategy council, with the directors of SAODAP and ODALE as co-chairmen, prepared the 1973 Federal Strategy for Prevention of Drug Abuse and Drug Trafficking, the first explicit strategy document.

During 1973, the drug program and drug-policy organizations underwent major change. The Office of Management and Budget (OMB) established a special management office called Federal Drug Management (FDM), which supported OMB's senior officials, the CCINC, and the White House Domestic Council. Given unusually wide latitude in providing direct management assistance to the drug-related operating agencies, FDM assisted in implementation of President Nixon's Reorganization Plan No. 2 of 1973. Also in 1973, Dr. Jaffe was succeeded at SAODAP by Dr. Robert Dupont who in 1975 became the first director of the newly established National Institute on Drug Abuse. FDM also assumed oversight of the demand-related drug activities as SAODAP was phased out of the EOP. Before terminating in mid-1975, SAODAP published the 1974 and 1975 federal strategies, under the auspices of a relatively inactive Strategy Council.

In early 1975, President Gerald R. Ford directed the White House Domestic Council to review the federal drug effort. Vice-President Nelson A. Rockefeller chaired an interagency task force called the Domestic Council Drug Abuse Task Force, with the chief of FDM as study director. The task force, with advice from community organizations, prepared a comprehensive White Paper on Drug Abuse. The 1975 white paper recommended assigning responsibility for overall policy guidance to the Strategy Council on Drug Abuse; creating an EOP Cabinet Committee to coordinate prevention and treatment activities; and continuing a small staff in OMB to assist the Strategy Council and the EOP. In April 1976, President Ford announced two new cabinet committees, the Cabinet Committee on Drug Law Enforcement and the Cabinet Committee on Drug Abuse Prevention "to ensure the coordination of all government resources which bear on the problem of drug abuse" (1976 Strategy, p. 26). The cabinet committee structure, supported by the FDM staff, worked to the satisfaction of President Ford but did not satisfy Congress.

Congress enacted legislation establishing an Office of Drug Abuse Policy (ODAP) in March 1976, seeking a single individual in the EOP who had responsibility for the overall drug program. President Ford did not activate the new agency but continued with the three cabinet committees, supported by the FDM staff.

Executive Drug Policy 1977–1980. In March 1977, President Jimmy Carter revised the drug-policy structure, activating ODAP and abolishing the three drug-related cabinet committees. Also, he revitalized the strategy council, with the director of ODAP as executive director, to serve as the governmentwide advisory committee for all drug-abuse matters. ODAP worked particularly well with the

White House staff, partially because Director Peter Bourne was also special assistant to the president for health issues and had an excellent relationship with President Carter and the White House staff. ODAP aggressively pursued a wide range of policy and coordination activities, including a major review of all federal drug programs.

The President's Reorganization Project reviewed the organization of the Executive Branch and recommended abolishing ODAP in mid-1977. Within the EOP, ODAP was an unusual federal agency, with a strong presence and authority for a single issue, somewhat contrary to the normal EOP structure. Thus, ODAP was a logical target in efforts to streamline the EOP. Congress disagreed strongly with the elimination of ODAP, however. After congressional hearings and negotiations, the Carter Administration compromised by continuing part of the ODAP staff and all the ODAP functions as part of the White House Domestic Policy Staff (DPS).

In March 1978, six members of ODAP's staff were transferred to DPS and became the Drug Policy Office (DPO). DPO continued to perform the ODAP functions, including responding to congressional interests and reporting directly to Peter Bourne. After Bourne departed the White House staff in 1978, the drug staff worked through the director of the DPS. In May 1979, the president affirmed the head of DPO (Lee Dogoloff, the associate director for drug policy)—as the individual primarily responsible for the federal government's drug-abuse prevention and control programs. DPO published the 1979 Federal Strategy and a 1980 Annual Report. A major policy-coordinating mechanism was the monthly meetings held by DPO with the heads of the major operating agencies (called the Principals Group). DPO also supported another policy-coordinating mechanism called the National Narcotics Intelligence Consumers Committee, established in April 1978. DPO also initiated efforts to increase military support for drug-interdiction activities. During the transition to the Reagan Administration in early 1981, most of President Carter's DPO staff departed.

Executive Drug Policy 1981–1988. In 1981, President Ronald W. Reagan's Office of Policy Development (OPD) included a Drug Abuse Policy Office (DAPO) similar in organization and role to the preceding DPO. President Reagan charged DAPO with (1) a full range of policy-development and -coordination activities, (2) international negotiations, and (3) assisting First Lady Nancy Reagan's drug-abuse prevention efforts. In addition to overseeing the efforts of the federal drug agencies, DAPO emphasized the use of all opportunities for the federal government to encourage a wide range of nongovernment antidrug activities. DAPO was directed by Carlton Turner, a pharmacologist, who was succeeded in 1987 by Dr. Donald Ian Macdonald, a pediatrician. DAPO published the 1982 Federal Strategy and, reflecting the broader policy direction, published the first "National" Strategy in 1984.

DAPO continued the coordination meetings with the agency heads (the previous Principals Group, renamed the Oversight Working Group) and assisted in the design and implementation of the National Narcotics Border Interdiction System (NNBIS), headed by Vice-President George H. Bush. DPO assisted the Cabinet Council on Legal Policy and the Cabinet Council on Human Resources with drug matters until the cabinet councils were replaced by the Domestic Policy Council in April 1985. The Domestic Policy Council Working Group on Drug Abuse Policy prepared a major presidential drug initiative in 1986, with assistance from DAPO.

During this period, the oversight of drug law enforcement moved away from the White House.

In 1984, Congress had established a federal drug law-enforcement czar to "facilitate coordination of U.S. operations and policy on illegal drug law enforcement." The attorney general was chairman of the new cabinet-level National Drug Enforcement Policy Board (NDEPB) with staff offices in the Department of Justice. DAPO was charged with ensuring "coordination between the NDEPB and the health issues associated with drug abuse," in addition to supporting the president and the White House staff. In January 1987, the NDEPB published the *National and International Drug Law Enforcement Strategy*, which expanded on the sections of the 1984 National Strategy involving drug law enforcement and international controls. DAPO continued to provide Executive Office oversight of the entire drug program.

In 1987, President Reagan replaced the NDEPB by creating a National Drug Policy Board (NDPB) to coordinate all drug-abuse policy functions. The director of the White House DAPO was a member and assisted the NDPB in developing the health-related drug policy. The NDPB published *Toward*

a Drug-Free America—The National Drug Strategy and Implementation Plans in 1988.

The White House Conference for a Drug Free America was opened in 1987 with DAPO assistance; it was charged with reviewing a wide range of drug programs, policies, and informational activities—including focusing "public attention on the importance of fostering a widespread attitude of intolerance for illegal drugs and their use throughout all segments of our society" (Executive Order No. 12595, Section 1(c)). The conference, chaired by Lois Haight Herrington, published a final report in 1988 with 107 wide-ranging recommendations, including a "Cabinet-rank position of National Drug Director."

In late 1988, Congress again passed drug czar legislation, authorizing a new agency named the Office of National Drug Control Policy (ONDCP) in the EOP.

Executive Drug Policy 1989–1990s. ONDCP began operation in the EOP in early 1989, absorbing the NDPB, and terminating the two existing White House drug activities, DAPO and NNBIS. Although never actually a member of the cabinet, the first two cabinet-level directors were given broad responsibilities for developing and guiding a National Drug Control Program, including developing an annual strategy and overseeing its implementation. The first director, William Bennett, had been secretary of education in the Reagan administration; he was succeeded by Bob Martinez, a former governor of Florida. ONDCP had oversight of organization, management, budget, and personnel allocations of all departments and agencies engaged in drugcontrol activities. ONDCP used a complex set of interagency coordinating committees under a Supply Reduction Working Group, a Demand Reduction Working Group, and a Research and Development Committee. The director chaired the NSC's Policy Coordinating Committee for Narcotics which ensured coordination between drug law enforcement and national security activities. The director also provided administrative support to the President's Drug Advisory Council, which in turn assisted ONDCP in supporting national drug-control objectives through private sector initiatives. ONDCP was also required to establish realistic and attainable goals for the following two years and the following ten years and to monitor progress toward the goals. Following the election of President Bill Clinton, Lee Brown, a criminologist and former New York police commissioner, was appointed director of ONDCP and was also given membership in the cabinet. The fourth director, retired Army General Barry R. McCaffrey, was appointed in 1996.

CONGRESSIONAL DRUG-POLICY OVERSIGHT

Various legislative committees and subcommittees oversee the drug-control activities of the Executive Branch departments and agencies. In addition to the various standing committees, Congress had special drug-oversight activities, including the Senate Caucus for International Narcotics Control and the House Select Committee on Narcotics Abuse and Control. Special audits and evaluations by the General Accounting Office and support from the Congressional Research Service also assisted Congress in its oversight role.

The continuing congressional interest in establishing an effective drug-policy oversight mechanism reflected the difficulties of the various committees in attempting to address the drug activities of a single agency within the context of the overall federal effort. The frustration was reflected in the repeated legislative efforts to establish a drug czar in the EOP to oversee federal drug policy and to advise both the president and Congress.

For example, the Senate Committee on Government Operations had a long-term interest in drug-program oversight. Senator Charles H. Percy, responding to the plan to abolish ODAP in 1977, summarized the congressional view. Reiterating the programmatic needs for a single, high-level coordinating body with broad statutory authority over federal drug-abuse policy and its implementation, Senator Percy stated:

My concerns are not limited to the question of whether the Federal drug abuse effort can function effectively under this proposal (to abolish ODAP). Indeed, my greatest opposition . . . is that Congressional participation in the formulation and execution of Federal drug policy will be seriously impaired with the demise of ODAP. . . . Although Congress has jurisdiction over the individual offices and agencies, this authority is meaningless without corresponding jurisdiction over those responsible for coordinating the line agencies' programs—the point where policy differences must be reconciled.

[Congressional Record, September 30, 1977; S-16071–16072].

In the House of Representatives, the Select Committee on Narcotics Abuse and Control, headed by Representative Charles Rangel, played an important role in Congressional oversight of drug programs and policy. The select committee was formed in July 1976 "to oversee all facets of the Federal narcotics effort and coordinate the response of the seven legislative committees in the House which have jurisdiction over some aspect of the narcotics problem." Without legislative jurisdiction, the select committee was primarily a fact-finding activity to support the seven standing committees in the House of Representatives. The select committee also was a focal point for congressional pressure for a legislatively based federal drug czar. In early 1993, the select committee on Narcotics Abuse and Control was discontinued.

DRUG-POLICY LEGISLATION

In 1972, Congress passed legislation authorizing the Special Action Office for Drug Abuse Prevention, as requested by President Nixon. After SAODAP expired in 1975, Congress authorized a replacement drug-policy agency (ODAP), in early 1976, and was critical of President Ford's decision to not open the new agency.

When President Carter decided to activate ODAP in early 1977, Congress applauded the decision and confirmed the director and deputy director; but ODAP was abolished in early 1978 despite congressional objections, ending their successful relationship with ODAP. The resulting executive/congressional negotiations required the Drug Policy Office of the DPS to carry out the functions previously assigned to ODAP and to allow congressional access to the drug-policy staff.

In late 1979, Congress followed up with legislation requiring the president to establish a drug-abuse policy coordination system and to designate a single officer to direct the activities (21 USC 1111 & 1112). A system was established by President Carter (Executive Order 12133, 1979-Drug Policy Office) and by President Reagan (Executive Order 12368, 1982-Drug Abuse Policy Office).

In late 1982, Congress enacted a strong drug czar, in an Office of National and International Drug Operations and Policy, with a cabinet-level director. The director was granted broad powers to develop, review, implement, and enforce government policy and to direct departments and agencies involved. The explicit power to direct other departments and agencies was seen as too strong and in conflict with the principles of cabinet government. President Reagan did not accept the legislation.

In 1984, the Congress and the administration agreed to establish a cabinet-level NDEPB with a limited charter to coordinate drug law enforcement. The legislation designated the attorney general as chairman and primary adviser to the president and to Congress—on both national and international law enforcement.

In 1987, President Reagan signed Executive Order 12590, broadened the charter of the attorney general and the NDEPB to include the entire federal drug program and named the new activity the National Drug Policy Board.

In late 1988, Congress passed new drug czar legislation, creating the Office of National Drug Control Policy in the EOP, with a cabinet-level director and funding provisions for both operating expenses and program activities. President Bush accepted the new agency and appointed a cabinet-level director, but he did not include the first director or his successor in his immediate cabinet.

Thus, Congress achieved the drug czar objectives that it pursued for two decades—a cabinet-level drug-policy manager with broad oversight of policy and budgets, responsible both to Congress and the president.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy International Drug Supply Systems; Opioids and Opioid Control, History; Prevention Movement; Treatment, History of*)

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RICHARD L. WILLIAMS

U.S. GOVERNMENT AGENCIES The following articles appear in this section:

Bureau of Narcotics and Dangerous Drugs (BNDD);
Center for Substance Abuse Prevention (CSAP);
Center for Substance Abuse Treatment (CSAT);
National Institute on Alcoholism and Alcohol Abuse (NIAAA);
National Institute on Drug Abuse (NIDA);
Office of Drug Abuse Law Enforcement (ODALE);
Office of Drug Abuse Policy (ODAP);
Office of National Drug Control Policy (ONDCP);
Special Action Office for Drug Abuse Prevention (SAODAP);
Substance Abuse and Mental Health Services Administration (SAMHSA);
U.S. Customs Service;
U.S. Public Health Service Hospitals

Bureau of Narcotics and Dangerous Drugs Presidential Reorganization Plan No. 1 of 1968 created the Bureau of Narcotics and Dangerous Drugs (BNDD) in the U.S. Department of Justice. The new agency combined the drug law enforcement functions of two predecessor organizations—the Federal Bureau of Narcotics (FBN) in the Department of the Treasury and the Bureau of Drug Abuse Control in the Food and Drug Administration, Department of Health and Human Services. Long-standing conflicts between two Department of the Treasury agencies that shared drug-enforcement responsibilities—the Federal Bureau of Narcotics and the Bureau of Customs—led to the decision to move the FBN functions into a new agency (BNDD) in a different cabinet department (Justice).

MISSION AND EXPERIENCE

BNDD's role was to suppress illicit narcotics trafficking and to control the diversion of legally manufactured drugs. BNDD was responsible for working with foreign governments to halt international drug traffic, immobilizing domestic illegal drug-distribution networks, providing a wide range of technical assistance and training to state and local officers, and preparing drug cases for prosecution.

BNDD emphasized investigations of high-level drug trafficking to identify and target major national and international violators. Director John E. Ingersoll described the success of BNDD as being "able to apprehend scores of illicit drug traffickers who were previously immune to the feeble efforts which law enforcement was formerly able to mount." In 1968 and 1969, BNDD contributed to major international success in stopping heroin traffic originating in Turkey.

The Bureau of Customs continued interdiction of drug smuggling at the borders and ports of entry. Customs special agents investigated drug cases based on seizures made by Customs inspectors and on antismuggling intelligence. Conflict between BNDD and Customs continued, with allegations of lack of cooperation and failure to share intelligence with each other.

The White House and Office of Management and Budget (OMB) tried to resolve the conflict and, in early 1970, President Richard M. Nixon directed BNDD and Customs to work out a set of operating guidelines. After considerable interagency discussion, formal guidelines were prepared to give to BNDD full jurisdiction over drug-enforcement operations both within the United States and overseas. Customs was to be limited to border operations. The president approved the guidelines, but the conflicts continued. Neither Congress nor the White House was satisfied. Senator Abraham Ribicoff described the detailed guidelines as "more reminiscent of a cease-fire agreement between combatants than a working agreement between supposedly cooperative agencies."

ADDITIONAL DRUG ENFORCEMENT COMPLICATIONS

The "war against drugs" continued to expand. In 1972, President Nixon established two new drug agencies in the Department of Justice—the Office of Drug Abuse Law Enforcement (ODALE) and the Office of National Narcotics Intelligence (ONNI). ODALE's operational involvement with state and local law enforcement against local drug dealers was intended to complement BNDD's focus on high level traffickers. ODALE, however, depended on existing federal agencies for agents and attorneys, and BNDD was required to lend over 200 narcotics agents to ODALE. The additional antidrug agencies, combined with sensational reporting of conflicts between special agents from BNDD and Customs, added to the public perception of fragmentation and disorder in federal drug law enforcement.

In early 1973, another presidential reorganization plan was designed to eliminate the overlap and duplication of effort in drug enforcement. A factual assessment of the BNDD/Customs situation, provided to the Congress by the chief of OMB's Federal Drug Management Division, Walter C. Minnick, reported "Having attempted formal guidelines, informal cooperation and specific Cabinet-level mediation, all without success, the President concluded in March of 1972 that merging the drug investigative and intelligence responsibilities of Customs and BNDD into a single new agency was the only way to put a permanent end to the problem." Under Reorganization Plan No. 2 of 1973, BNDD, ODALE, and ONNI were eliminated; their functions and resources, along with 500 Customs special agents (those previously involved in drug investigations), were consolidated in the new Drug Enforcement Administration (DEA) in the Department of Justice.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy*)

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RICHARD L. WILLIAMS

Center for Substance Abuse Prevention (CSAP) This agency was originally established as the Office for Substance Abuse Prevention (OSAP). It was created by the Anti-Drug Abuse Act of 1986 for the prevention of alcohol and other drug (AOD) problems among U.S. citizens, with special emphasis on youth and families living in high-risk environments. Dr. Elaine Johnson was appointed as the first director of the office. From 1986 to 1992, OSAP operated as a unit of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA), one of the eight Public Health Service agencies within the U.S. Department of Health and Human Services.

In 1992, Public Law 102–321 reorganized ADAMHA and renamed it the Substance Abuse and Mental Health Services Administration (SAMHSA); it also created CSAP to replace OSAP.

The goal of CSAP is to promote the concepts of no use of any illicit drug and no illegal or high-risk use of alcohol or other legal drugs. (High-risk alcohol use includes drinking and driving; drinking while pregnant; drinking while recovering from alcoholism and/or when using certain medications; having more than two drinks a day for men and more than one for women, or to intoxication).

These are the principles that guide the prevention work of CSAP:

1. The earlier PREVENTION is started in a person's life, the more likely it is to succeed.
2. PREVENTION PROGRAMS should be knowledge based and should incorporate state-of-the-art findings and practices drawn from scientific research and field expertise.
3. Prevention programs should be comprehensive.
4. Programs should include both process and outcome evaluations.
5. The most successful programs are likely to be those initiated and conducted at the community level.

To utilize these principles and achieve its goals, CSAP performs the following functions:

1. Carries out demonstration projects targeting specific groups and individuals in high-risk environments.
2. Assists communities in developing long-term, comprehensive AOD-use prevention programs and early intervention programs.
3. Operates a national clearinghouse for publications on prevention and treatment and other materials and services, including the operation of the Electronic Communication System and the Regional Alcohol and Drug Awareness Resource (RADAR) Network.
4. Supports the National Training System, which develops new drug-use prevention materials and delivers training.
5. Supports field development.
6. Conducts an evaluation strategy consisting of individual grantee evaluations, contractual program-wide evaluations, and the National Evaluation Project.
7. Provides technical assistance for capacity building and promotes collaborations to help states, communities, and organizations develop and implement communications, drug-use prevention, and early intervention efforts.
8. Develops and implements public information and educational media campaigns and other special-outreach and knowledge-transfer prevention programs.
9. Maintains a national drug-use prevention database to provide information on substance-abuse prevention programs.
10. Provides technical assistance and materials to small businesses for the development of EMPLOYEE-ASSISTANCE PROGRAMS.
11. Operates the National Volunteer Training Center for Substance Abuse Prevention.

To promote interagency cooperation and facilitate jointly sponsored prevention activities, CSAP's staff meets routinely with various federal organizations, including the departments of defense, justice, education, transportation, labor, housing and urban development, the Bureau of Indian Affairs, and others.

CSAP also develops partnerships with the research community, parent groups, foundations, policymakers, health-care practitioners, state and community leaders, educators, law enforcement officials, and others to enhance opportunities for comprehensive approaches to prevention and early intervention.

(SEE ALSO: *Education and Prevention; Parents Movement; Prevention Movement*)

ELAINE JOHNSON

Center for Substance Abuse Treatment (CSAT) The Center for Substance Abuse Treatment (CSAT) was established in January 1990 as the Office for Treatment Improvement (OTI) of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) in the Department of Health and Human Services (DHHS). Dr. Beny J. Primm, a physician who had spent more than twenty years developing a major treatment program in New York City, was appointed its first director. Following reorganization of ADAMHA in 1992, the agency was renamed and is now part of the Substance Abuse and Mental Health Services Administration (SAMHSA), which replaced ADAMHA.

The congressional mandate of CSAT is to expand the availability of effective treatment and recovery services for people with drug and alcohol problems. One of its goals is to ensure that new treatment technology is absorbed by the addiction-treatment infrastructure—that is, the system of state and local government agencies and public and private treatment programs providing addiction-treatment services. In carrying out this responsibility, CSAT collaborates with states, communities, and treatment providers to upgrade the quality and effectiveness of treatment and enhance coordination among drug-treatment providers, human-services, educational and vocational services, the criminal-justice system, and a variety of related services. CSAT provides financial and technical assistance for this purpose to targeted geographic areas and patient populations, with emphasis on assistance to minority racial and ethnic groups, ADOLESCENTS, HOMELESS people, WOMEN of childbearing age, and people in rural areas.

CSAT also collaborates with other government agencies, such as the National Institute on Drug Abuse (NIDA), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), the National Institute of Mental Health (NIMH), the Center for Substance Abuse Prevention (CSAP), and state and local governments to promote the utilization of effective means of treatment and to develop treatment standards. In addition, CSAT has interagency agreements with the Department of Labor and the Department of Education that are designed to improve the coordination of health and human services, education, and vocational training. CSAT also promotes the mainstreaming of alcohol-, drug-abuse, and mental-health treatment into the primary health care system, and it is responsible for administering the Substance Abuse Prevention and Treatment (SAPT) Block Grant program, which provides federal support to state substance-abuse prevention and treatment programs (funded at $1.13 billion in fiscal year 1993).

Research has generated a vast body of knowledge regarding the nature of chemical dependency and about what works in the treatment of addiction and addiction-related primary health and mental-health disorders. From this research, three key observations formed the basis for CSAT's initial treatment philosophy. First, addiction is a complex phenomenon; people's addiction cannot be treated in isolation from addressing their primary health, mental health, or socioeconomic deficits. Second, addiction is frequently a chronic, relapsing disorder; the gains made during treatment often are lost following a person's return to the community. CSAT therefore tried to foster programs that provided those treated for chemical dependency with a series of interventions along a sustained continuum. These two observations constituted the basis for CSAT's Comprehensive Treatment Model, which was a central principle in all of its demonstration grant programs and technical-assistance initiatives. During its first few years of existence, CSAT targeted resources to the people it perceived as most adversely affected by extreme socioeconomic problems and at highest risk for addiction because of exposure to CRIME, abuse, POVERTY, and HOMELESSNESS, and also because of lack of access to primary health and mental health care, social services, and vocational training and education. For this reason, the early CSAT Comprehensive Treatment Model demonstration grants fostered a wide array of primary interventions geared to addressing each patient's health and human service needs, coupled with a readily accessible, intensive aftercare component.

At the core of CSAT's overall approach is, quite simply, the conviction that treatment works. Treatment has proved effective in reducing the use of illicit drugs and alcohol, improving rates of employment, reducing rates of HUMAN IMMUNODEFICIENCY VIRUS (HIV) seroconversion, reducing criminal activity, and reducing overall patient morbidity.

In addition to the SAPT Block Grant, CSAT awarded grants for a variety of demonstration and service programs: The treatment-capacity expansion program provided resources to the states to

expand capacity in areas of demonstrated shortage; Target Cities assists metropolitan areas with particularly high-risk populations in providing treatment services and in developing systems to coordinate and improve the infrastructure of the programs. Critical Populations is a demonstration project for treatment program enhancement aimed at particularly at-risk groups—ADOLESCENTS; racial and ethnic minorities; residents of public housing; women and their infants and children; rural populations; drug and alcohol abusers who are homeless; patients with HIV or AIDS. Criminal justice-related programs include drug-abuse treatment programs in PRISONS AND JAILS; diversion to treatment; special services for probation or parole clients; screening, testing, referral, and treatment services for HIV/AIDS, TB, and other communicable diseases; literacy, education, job training, and job placement services; and case management and DRUG TESTING. CSAT also supported demonstration treatment campus programs; several programs aimed specifically at WOMEN and their infants and children; AIDS outreach for substance abusers; linkage of primary care and substance abuse model programs; state systems development programs; professional training and education; and collaborative efforts with other federal agencies.

After Dr. Primm's return to New York in 1992 and following Mr. David Mactas's appointment to head the agency in 1994, and as part of the Clinton administration's effort to reinvent government (redefine and refine its functions), CSAT's demonstration grant program emphasis shifted from improvement of services for the populations in greatest need to the development of knowledge about the effectiveness of treatment for different subgroups of the drug-using population.

Information regarding CSAT's current programs and technical initiatives is available from the CSAT Public Affairs Office, Center for Substance Abuse Treatment, Substance Abuse and Mental Health Services Administration, 5600 Fishers Lane, Rockville, MD 20857.

(SEE ALSO: *Ethnic Issues and Cultural Relevance in Treatment; Treatment Types; Vulnerability As Cause of Substance Abuse*)

BENY J. PRIMM

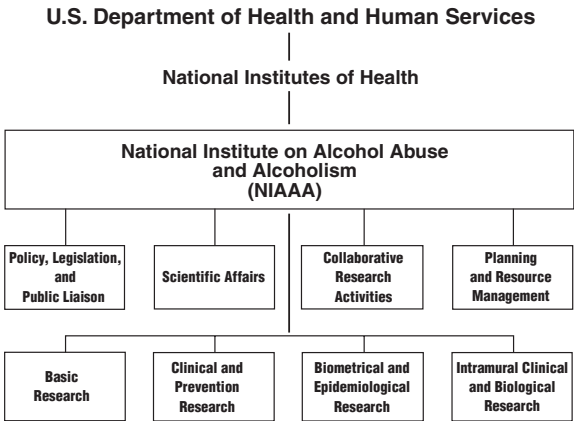
The National Institute on Alcohol Abuse and Alcoholism The National Institute on Alcohol Abuse and Alcoholism (NIAAA) is the principal Federal agency for research on the causes, consequences, treatment, and prevention, of alcohol-related problems. NIAAA supports studies both biological and behavioral research; research training and health professions development programs; and research on alcohol-related public policies. The NIAAA budget for Fiscal Year 2000 is $293 million.

ORGANIZATION

NIAAA is one of 18 research institutes of the prestigious National Institutes of Health (NIH), a component of the U.S. Department of Health and Human Services. Three principal staff offices and four Divisions manage and coordinate NIAAA activities: **Office of Collaborative Research Activities**-manages activities with other NIH Institutes, government agencies, and other organizations interested in alcohol-related problems and the Institute's international activities and science education programs; **Office of Policy, Legislation, and Public Liaison** monitors alcohol-related legislative developments and proposals; provides science-based recommendations for changes in public policies; and supports programs aimed at bridging the gap between research and practice; **Office of Planning and Resource Management** provides financial, grants, contracts, and other administrative support for Institute programs and activities; **Division of Basic Research** manages the Institute's biological research grants portfolio in areas such as neurosciences, genetics, and molecular biology. **Division of Clinical and Prevention Research** supports studies aimed at developing practical and effective ways to prevent and treat alcohol use problems, including new medications development; interventions with high-risk populations s; and behavioral therapies; **Division of Intramural Clinical and Biological Research** manages the NIAAA intramural research program.

MAJOR PROGRAMS AND ACTIVITIES

NIAAA supports research principally through extramural grants awarded to scientists at leading U.S. research institutions and through research conducted by NIAAA's own intramural staff scien-



tists. Findings from these research areas are made available and accessible through a wide variety of research dissemination activities.

Extramural Research. *Genetics*. NIAAA supports research aimed at discovering the genes that predispose individuals to alcoholism and the environmental factors that influence its development. Areas of genetics research include: twin studies to define precisely what is being inherited; genetic linkage and association studies to identify the genes for alcoholism and their precise number, identity, and modes of action; genetic analysis of alcohol-related behavior in animals, the genes that influence these behaviors, and studies to determine the contributions of the environment and genetics to an individual's susceptibility for developing alcohol-related medical disorders such as liver cirrhosis, pancreatitis, and fetal alcohol syndrome.

Alcohol and the Brain. Many of the behaviors associated with alcohol use problems are the result of alcohol's effects in the brain. NIAAA research is designed to learn how these effects influence the development of alcohol abuse and alcoholism. Molecular biology and genetic techniques, including the use of transgenic animals, are becoming an integral part of this research. In addition, noninvasive, functional imaging techniques are used in animal and human studies to identify neural circuits influenced by alcohol.

Medications Development. NIAAA is strongly committed to developing medications to diminish the craving for alcohol, reduce risk of relapse, and safely detoxify dependent individuals undergoing treatment. Naltrexone, an opioid antagonist, the first medication approved as a safe and effective adjunct to psychosocial treatment for alcoholism since 1949 was developed from neuroscience research. NIAAA anticipates that this number will increase over the next several years as findings from neuroscience and from genetics point to promising targets for pharmacological intervention.

Prevention. NIAAA prevention research is aimed at developing effective measures to reduce alcohol-related problems, including studies of alcohol-related intentional and unintentional injury, alcohol-related violence, alcohol in the workplace; drinking and driving deterrence, and the relationship between alcohol availability and alcohol-related problems. New methodologies permit prevention researchers to target high-risk neighborhoods within larger cities.

Treatment. NIAAA continues to emphasize research to improve treatment of alcohol abuse and alcoholism and supports a range of treatment or clinical studies including clinical trials of treatment therapies, patient-treatment matching studies, and behavioral/pharmacological treatment approaches.

Epidemiology. Alcohol epidemiology provides the foundation for monitoring the health of the population, developing and evaluating prevention and treatment services for alcohol problems, and establishing alcohol-related social policies. NIAAA-supported epidemiology research examines the context, volume, and specific drinking patterns that lead to particular alcohol-related problems as well as the impact of age, gender, race/ethnicity, and other sociodemographic factors; genetic, environmental, and other factors which influence injury or disease occurrence.

Intramural Research. Scientists in the NIAAA Intramural Research Program (IRP) focus on research opportunities that allow intensive, long-term commitment as well as the flexibility to adjust research priorities in response to new findings. Because clinical and laboratory studies occur side by side, new findings from basic research may be transferred readily for appropriate testing and application, and clinical hypotheses may, in turn, be posited to lab scientists. Areas of study include identification and assessment of genetic and environmental risk factors for the development of alcoholism; the effects of alcohol on the central nervous system, including how alcohol modifies brain activity and behavior; metabolic and biochemical effects of alcohol on various organs and systems of the

body; noninvasive imaging of the brain structure and activity related to alcohol use development of animal models of alcoholism; and the diagnosis, prevention, and treatment of alcoholism and associated disorders. NIAAA utilizes a combination of clinical and basic research facilities, which enables a coordinated interaction between basic research findings and clinical applications in pursuit of these goals. An 11-bed inpatient ward and a large outpatient program are located in the NIH Clinical Center in Bethesda, Maryland.

RESEARCH DISSEMINATION

NIAAA shares relevant findings from alcohol research with health care practitioners, policy makers and others involved in managing alcohol-related programs, and the general public through publications in scientific and clinical journals, general and specialized brochures, and pamphlets, manuals clinical bulletins. Research findings are also shared with the alcohol and general health care communities through three online database services supported by the institute: Quick Facts, an epidemiological data base; ETOH, an alcohol-related bibliographic reference database; and the NIAAA clinical trials database.

Publications, reports, and database services are accessible online at http://www.niaaa.nih.gov.

ENOCH GORDIS, M.D.

National Institute on Drug Abuse (NIDA)

The National Institute on Drug Abuse is the world's premier research institute supporting research on the health aspects of drug abuse and addiction. NIDA's vast portfolio supports research on all drugs of abuse from opiates and cocaine to new and emerging drugs such as methamphetamine and ecstasy. In addition to research on illegal drugs, NIDA supports an extensive research portfolio to combat what may be the nation's most critical and costly public health problem—tobacco use. NIDA's nicotine research continues to increase our understanding of the social, economic, cultural and biological factors that influence smoking initiation and vulnerability to nicotine addiction, and continues to bring the nation the most effective prevention and treatment approaches available. Additionally, NIDA supports research on the health consequences of nicotine as well as on the medical consequences of all illicit drugs. Given that drug abuse is the greatest vector for the spread of HIV, a significant portion of NIDA's research investment is spent on researching effective prevention and treatment strategies to combat HIV/AIDS and other infectious diseases. NIDA's comprehensive research portfolio includes studies on the causes and consequences, the prevention and treatment, and the biological, social, behavioral, and neuroscientific bases of drug abuse and addiction. NIDA is also charged with the development of medications to treat drug addiction. Additionally, NIDA supports research training and career development, science and public education, and research dissemination.

NIDA is the largest institution devoted to drug-abuse research in the world, supporting almost 85 percent of all drug-abuse research through grants to scientists, primarily at major research facilities in the United States, abroad, and at NIDA's own Intramural Research Program (IRP).

HISTORY

Drug-abuse research and treatment have been a concern of the U.S. Public Health Service since the early 1930s. The Public Health Service Hospitals at Lexington, Kentucky, and at Fort Worth, Texas, were established in 1929—and the research laboratories were established at Lexington in 1935.

NIDA was formally established in 1974 as one of three research institutes within the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA), a Public Health Service agency within the Department of Health and Human Services. NIDA's mandate was to collect information on the incidence, prevalence, and consequences of drug abuse, to improve the understanding of drugs of abuse and their effects on individuals, and to expand the ability to prevent and treat drug abuse. Through scientific research, NIDA has built a base of information on how drugs affect us—what they do to our bodies; to our behavior, thoughts, and emotions; to our relationships; and to our society. This understanding of the biological, social, behavioral and environmental influences that place individuals at risk for drug abuse is of great importance to prevention and treatment practitioners, to educators, and to policymakers.

In October 1992, the drug, alcohol, and mental-health activities within the Department of Health

and Human Services (NIDA, along with the National Institute on Alcohol Abuse and Alcoholism and National Institute on Mental Health) were transferred from ADAMHA to the National Institutes of Health.

FUNCTIONS

To improve the ability to prevent drug abuse, NIDA is concentrating on the variety of biological, behavioral, social, and environmental factors involved in vulnerability to drug abuse. This information enables NIDA to improve both prevention and treatment approaches—which are key to overcoming the demand for drugs—and to inform effective U.S. demand-reduction policies.

Drug addiction is a chronic, relapsing disorder, but research has shown that treatment can be an effective tool in helping some to break the addiction cycle. Successful treatment offers the best means for overcoming a life cycle revolving around drug-seeking behaviors and also reduces the spread of AIDS and other infectious diseases among drug abusers. Accordingly, NIDA is researching ways to improve the effectiveness of treatment and working to increase retention rates and reduce relapse rates. Through an understanding of the effects of drugs on the brain, NIDA is developing more effective treatments-including medications-for specific drugs of abuse, such as COCAINE and HEROIN, and for the toxic effects on the BRAIN and other organs that drugs of abuse produce. NIDA has engaged in a major effort to improve research on, and its application to, services for drug-abusing pregnant and postpartum women. NIDA also seeks to develop strategies to prevent or ameliorate the consequences of drugs of abuse on the children of drug-abusing parents.

To support this array of research programs, the research community needs an adequate supply of scientists with up-to-date skills and knowledge. Accordingly, NIDA sponsors drug-abuse research programs in the biomedical and behavioral sciences. These programs include support of pre- and post-doctoral training in medical schools, universities, and other institutions of higher education in basic, clinical, behavioral, and epidemiological research, to assure the steady supply of trained scientists. A final important function of NIDA is to make research findings available to the widest audience possible. NIDA has an extensive outreach and public education program to rapidly provide research-based information to scientists, practitioners, policy makers, and the general public. NIDA staff works closely with local community-based networks to hold town meetings at various locations across the country, as well as other major conferences to ensure that the latest scientific information is disseminated to those working to prevent and treat drug abuse and addiction. NIDA also develops written and electronic materials for researchers, prevention practitioners, treatment practitioners, young people, parents, policy-makers, and others. Additionally, NIDA has a Science Education Program, which develops materials for K-12 students and teachers, as well as the general public, and funds grants with educators and scientists for the development of programs, materials and museum exhibits. Through NIDA's research dissemination programs, science-based information can then be used to educate, prevent, treat, and rehabilitate.

CONCLUSION

NIDA conducts and supports RESEARCH that has as its underlying principles the goals of eliminating drug abuse, treating those whom prevention fails, increasing retention and decreasing relapse, and improving the health and well-being of all Americans, their families, their communities, and the nation.

NIDA collaborates with other research institutes, and with other agencies and departments of the U.S. government. For more information visit the NIDA website at www.nida.nih.gov.

RICHARD A. MILLSTEIN
REVISED BY ALAN. I. LESHNER

Office of Drug Abuse Law Enforcement (ODALE) Located within the U.S. Department of Justice, the Office of Drug Abuse Law Enforcement (ODALE) was established by President Richard M. Nixon with Executive Order 11641 in January 1972. Myles J. Ambrose was appointed director of ODALE and held two other concurrent titles: special consultant to the president for drug abuse law enforcement and special assistant attorney general.

FEDERAL, STATE, AND LOCAL TEAMWORK

Complementing federal efforts directed at "high-level drug traffickers," ODALE was charged with attacking the heroin-distribution system at the street level to reduce the drug's availability there. Patterned after the justice department's Organized Crime Strike Forces, the ODALE program included task forces of federal, state, and local law-enforcement officers and attorneys. The full use of federal, state, and local narcotics laws, the availability of assigned attorneys, and the use of the investigative grand jury made possible a wide range of approaches in pursuing violators.

ODALE established task forces in thirty-four cities in 1972 and encouraged citizens to "report information regarding alleged narcotics law violators in strict confidence." The federal government paid for task force equipment and operational expenses, including payments for a portion of the salaries and overtime of state and local officers. ODALE was credited with more than 8,000 narcotics arrests with a conviction rate of more than 90 percent during its 17 months of operation. Nevertheless, ODALE agents were widely criticized for conducting several drug raids involving unauthorized forcible entries into private homes and failures in identifying themselves as law officers during drug raids.

REORGANIZATION

ODALE was abolished on July 1, 1973, by Presidential Reorganization Plan No. 2 of 1973 and "those Federal operations designed to attack narcotics traffic at the street level in cooperation with local authorities" were transferred to the newly established Drug Enforcement Administration (DEA). The ODALE program was redesignated as DEA's State and Local Task Force program. ODALE's Deputy Director John R. Bartels, Jr., became the first administrator of the DEA.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy*)

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Office of Drug Abuse Policy In March 1976, Congress authorized the creation of the Office of Drug Abuse Policy (ODAP) in the Executive Office of the President, with an annual budget of $1.2 million. President Jimmy Carter opened the office in March 1977 and appointed Dr. Peter G. Bourne as director.

The director of ODAP was given wide responsibilities in assisting the president with all federal drug-abuse matters, including providing "policy direction and coordination among the law enforcement, international and treatment/prevention programs to assure a cohesive and effective strategy that both responds to immediate issues and provides a framework for longer-term resolution of problems." The statutory authority included setting objectives, establishing priorities, coordinating performance, and recommending changes in organization.

During the first year of operation, ODAP conducted several international missions and worked closely with United Nations narcotics organizations. In coordinating federal drug activities, ODAP relied on biweekly discussion meetings with the heads of the principal drug agencies. Policy determination was executed through cooperative interagency study efforts. ODAP completed six comprehensive interagency policy reviews: border management, drug law enforcement, international narcotics control, narcotics intelligence, demand reduction, and drug abuse in the armed forces.

The ODAP staff coordinated preparation of President Carter's August 1977 Message to the Congress on Drug Abuse and initiated the planning for a comprehensive federal strategy to be published by the revitalized Strategy Council.

REORGANIZATION

After one year of successful operation, ODAP was abolished by Reorganization Plan No. 1 of 1977, effective March 31, 1978. Six ODAP staff members were transferred to a special drug-policy unit (Drug Policy Office) within the White House Domestic Policy Staff. The drug-policy staff continued to report to Dr. Bourne who became special assistant to the president for health issues.

(SEE ALSO: *Anslinger, Harry J. and U.S. Drug Policy*)

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Office of National Drug Control Policy

The Office of National Drug Control Policy (ONDCP) was established on January 29, 1989, by Public Law 100–690 (21 USC 1504) as the drug-coordination agency for the Executive Office of the President (EOP) under President George H. Bush. ONDCP is responsible for coordinating federal efforts to control illegal drug abuse. It is the product of almost two decades of congressional efforts to mandate a so-called drug czar—the law providing for cabinet-level status and congressional involvement in drug-control policy. Its initial five-year authorization, which expired November 17, 1993, was extended.

ONDCP oversees international and domestic antidrug functions of all executive agencies and ensures that such functions sustain and complement the government's overall antidrug efforts.

THE DIRECTOR

ONDCP is led by a director (commonly referred to as the drug czar) with cabinet-level rank (Executive Level 1), two deputies (supply reduction and demand reduction), and one associate director (state and local affairs), all appointed by the president with the advice and consent of the Senate.

The director has a broad mandate for establishing policies, objectives, and priorities for the National Drug Control Program. Serving as the president's drug-control adviser and as a principal adviser to the National Security Council (NSC), the director has extraordinary management tools available to influence the national drug-control efforts.

ONDCP is required to produce an annual National Drug Control Strategy for the president and Congress and is responsible for overseeing its implementation by the federal departments and agencies. Included is an annual consolidated National Drug Control Program budget and the director's certification that the budget is adequate to implement the objectives of the strategy. In addition to the strategy and program oversight, the director has two other legislated management tools—(1) approval of reprogramming of each agency's drug funds and (2) formal notification to the involved agency and the president when a drug-program agency's policy does not comply with the strategy. The director also recommends changes in organization, management, and budgets of departments and agencies engaged in the drug effort, including personnel allocations.

Reflecting congressional desire to participate in drug policy, the director must represent the administration's drug policies and proposals before Congress. Additionally, the authorizing legislation specifically allows Congress access to "information, documents, and studies in the possession of, or conducted by or at the direction of the Director" and to personnel of the office.

The first director of ONDCP was William J. Bennett, 1989–1990, previously the secretary of education during the administration of President Ronald W. Reagan. Director Bennett had the difficult job of starting the new agency from scratch and developing a new national drug-control strategy within the first year of operation. Reagan's successor, President Bush, declined to include the cabinet-level ONDCP director in his immediate cabinet, bringing congressional criticism. Bob Martinez (the former governor of Florida) was the next director, 1991–1992. The third director, Lee P. Brown, a criminologist and a former New York City police commissioner, was appointed by President Bill Clinton in 1993 and was given cabinet status. The fourth director, retired Army General Barry R. Mc-

Caffrey, a decorated combat veteran in Vietnam, was also appointed by President Clinton, in 1996. McCaffrey is expected to be replaced with a change in administrations after the November 2000 Presidential election.

ORGANIZATION AND AUTHORITY

Initially, ONDCP had approximately 127 staff positions and 40 additional members detailed from other federal agencies. ONDCP's Fiscal Year (FY) 1992 appropriation of $105 million included $86 million to be transferred to support the High Intensity Drug Trafficking Areas (HIDTA). The HIDTA funding provides $50 million for federal law-enforcement agencies and $36 million for state and local drug-control activities. President Clinton drastically reduced the size of the ONDCP staff soon after his election, from 146 to 25. With the appointment of General Barry R. McCaffrey President Clinton intended to bring the number of staff back up to its original capacity. Additionally, President Clinton wished to appropriate money from the Department of Defense.

The director is responsible for a Special Forfeiture Fund, funded by the department of Justice Assets Forfeiture Fund, "to supplement program resources used to fight the war on drugs." For FY 1992, this fund included over $50 million for transfer to federal program agencies.

Additionally, ONDCP reviews and recommends funding priorities for the annual budget requests for over fifty federal agencies and accounts involved in the drug program (more than $12 billion in FY 1993).

ONDCP's authority to provide direction to diverse federal departments and agencies is based on a program-management structure known as the National Drug Control Program. The ONDCP program and budget authority coexists with the line authority of the cabinet departments and with the president's annual budget process (directed by the Office of Management and Budget). The structure for the parallel drug-control system is created by designating National Drug Control Program agencies, defined as "any department or agency and all dedicated units thereof, with responsibilities under the National Drug Control Strategy." The designated federal departments and agencies have special program and budget responsibilities to the director of ONDCP.

ONDCP's broad coordination authority over budgets and program activity also presents extraordinary opportunities for conflict with the existing line authority in the departments and agencies. Simultaneously, ONDCP receives congressional and press criticism regarding lack of influence over the operating activities.

POLICY DEVELOPMENT AND COORDINATION

The continued success of the complex drug-policy system depends on a continuing high priority for the drug programs, preventing bureaucratic turf battles, and seeking widespread understanding and endorsement of the goals and objectives of the national program. An essential element in communicating is a public document that explains the strategy, goals, and responsibilities—including a dynamic process of evaluating results and updating the strategy.

The annual National Drug Control Strategy, with accompanying Budget Summary (the February 1999 strategy was the most recent in the series) contains a description of the drug-abuse situation, an assessment of progress, and national priorities—with two-year and ten-year objectives and a federal budget "cross-cut" and analysis. ONDCP has brought together a complex set of drug-control program functions and budgets in an understandable way; by function in the strategy and by agency in the budget summary. Under Lee P. Brown the office produced an interim strategy for 1993 and a fully developed strategy in February 1994. McCaffrey's 1999 strategy, similar to previous years' versions, concentrated on five areas: (1) increasing anti-drug education aimed at children; (2) decreasing the number of addicted people by closing the "treatment gap"; (3) breaking the cycle of drugs and crime; (4) securing the nation's borders from drugs; and (5) reducing the overall drug supply. The goal of this strategy is to shrink the use and availability of illegal drugs by 25 percent by 2002 and by 50 percent by 2007. Additionally, the plan assures a 30 percent reduction in drug-related crimes by 2007, as well as a 25 percent reduction in health- and social-related drugs costs. (Advocates, 1999).

The National Drug Control Strategy acknowledges that no single tactic will solve the drug problem. Therefore, the annual strategies call for im-

proved and expanded treatment, prevention and education; increased international cooperation; aggressive law enforcement and interdiction; expanded use of the military; expanded drug intelligence; and more research.

ORGANIZATION FOR COORDINATION

ONDCP has established a drug-control management agenda, including federal coordinating mechanisms and senior-level management committees and working groups. The organization of ONDCP includes staff for supply reduction, demand reduction, and state and local affairs. ONDCP working groups and committees coordinate the implementation of the policies, objectives, and priorities established in the National Drug Control Strategy.

The federal drug-control agencies and departments are represented on the various working groups and committees, along with ONDCP staff. The organizational structure includes the following coordinating mechanism:

ONDCP Supply Reduction Working Group. Chaired by the ONDCP deputy director for supply reduction, the working group includes three committees:

The Border Interdiction Committee. Coordinates strategies and operations aimed at interdicting drugs between source and transit countries and at U.S. borders. The ONDCP may become more internationally-oriented in the future as the policy of source control continues to dominate US policy. For example, McCaffrey continues to work with the Mexican government to control drug trafficking at the U.S. southern border (Dettmer, 1997). Also, there has been a recent push by McCaffrey, with support from President Clinton, to provide more than a billion dollars in aid to Colombia for drug interdiction endeavors (ONDCP, Statement, 2000). According to a March 29, 2000 press release from the ONDCP that aid package was passed by the House of Representatives (ONDCP, Press Release, 2000).

The Public Land Drug Control Committee. Coordinates federal state, and local drug control programs (primarily marijuana eradication efforts) on federal lands.

Southwest Border and Metropolitan HIDTA Committees. Coordinates drug law enforcement activities in designated areas, including federal, state, and local enforcement task forces and intelligence activities. Four metropolitan HIDTAs have been designated: New York City, Miami, Houston, and Los Angeles.

ONDCP Demand Reduction Working Group. Chaired by the ONDCP deputy director for demand reduction, the working group coordinates policies, objectives, and outreach activities for treatment, education and prevention, workplace, and international demand reduction.

Research and Development Committee. Chaired by the director of ONDCP, the committee provides policy guidance for R&D activities of all federal drug control agencies, including the following R&D working committees—

The Data Committee. Improves the relevance, timeliness, and usefulness of drug-related data collection, research studies, and evaluations of both demand-related and supply-related activities.

The Medical Research Committee. Coordinates policy and general objectives on medical research by federal drug-control agencies and promotes the dissemination of research findings.

The ONDCP Science and Technology· Committee. Chaired by the ONDCP chief scientist, the committee is responsible for oversight of counterdrug research and development throughout the federal government.

RELATED POLICY ACTIVITIES

The Counter-Narcotics Technology Assessment Center, established by Public Law 101–509 in 1991, provides oversight of the federal government's counternarcotics research and development activities. ONDCP's chief scientist is responsible for defining scientific and technological needs for federal, state, and local law-enforcement agencies, and for determining feasibility and priorities. The chief scientist also coordinates the technology initiatives of federal civilian and military departments, including research on substance-abuse addiction and rehabilitation.

ONDCP works with the NSC, chairing the Policy Coordinating Committee for Narcotics to oversee coordination among agencies with law-enforcement and national-security responsibilities. The director also participates in meetings of the Domestic Policy council, which reviews the annual drug control strategy before it goes to the president.

ONDCP's state and local affairs staff sought wide public involvement in developing and imple-

menting drug policy at all levels of government. Several national conferences on state and local drug policy were sponsored by ONDCP during 1990 and 1991 to highlight successful state and local programs, seek input to the national strategy, and inform participants of funding and initiatives available to them. ONDCP staff coordinated with both the White House Office of National Service and the president's Drug Advisory Council in encouraging private-sector and state-and-local initiatives for drug prevention and control.

ONDCP also provides administrative support to the president's Drug Advisory Council. With thirty-two private citizens as members, the Drug Advisory Council focuses on private-sector initiatives to support national drug-control objectives, and it assists the ONDCP. The advisory council is financed by private gifts.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy; Opioids and Opioid Control, History of*)

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Special Action Office for Drug Abuse Prevention (SAODAP) The Special Action Office for Drug Abuse Prevention (SAODAP) was created by Executive Order of President Richard M. Nixon on June 17, 1971, as a response to public concern about drug abuse, particularly heroin addiction. SAODAP was given legislative authority by the Drug Abuse Office and Treatment Act on March 21, 1972. The formation of SAODAP represented the first attempt to establish a stable focus within the federal government for the coordination of the many facets of U.S. drug policy, including law enforcement, border control, control of selected medicines, treatment, prevention, education, and research.

More than twenty agencies, offices, and bureaus within the U.S. government were responsible for activities relating to drug problems. Yet there was no evident central authority other than the president. Congress and the public seemed eager to be able to hold accountable the head of one agency who, unlike the president, could be asked to testify before congress—a "drug czar." Although the term "drug czar" was popularly used, and it was expec-

ted that the person holding the office would exert power over the various agencies dealing with both law enforcement (supply side) and treatment and prevention (demand side) aspects of the problem, neither the president nor the Congress were entirely comfortable with delegating such broad authority to only one individual.

The legislation submitted to Congress by the White House, which finally emerged from debate, gave SAODAP unprecedented authority over demandside activity—treatment, prevention, education, research—wherever these were carried out within the federal government. However, its mandate with respect to drug-control agencies such as the U.S. Customs Bureau, which reported to the secretary of the treasury, and the Bureau of Narcotics and Dangerous Drugs, which reported to the attorney general, was limited to coordination. SAODAP was also charged with developing a formal, written, national strategy for drug-abuse prevention. To head the new office, President Nixon appointed Dr. Jerome H. Jaffe, then a professor of psychiatry at the University of Chicago and director of the Illinois Drug Abuse Programs. Dr. Jaffe, who had helped the White House develop its response to HEROIN use in VIETNAM, was also appointed special consultant to the president on narcotics and dangerous drugs.

A primary goal of SAODAP, stated at the press conference that announced the new office, was to make treatment so available that no addicts could say they committed crimes because they could not get treatment. Although the Bureau of Narcotics and Dangerous Drugs (BNDD) had estimated that there were about a half million heroin users in the United States, in mid-1971 the true extent of the drug-abuse problem was unknown. The estimating techniques that were developed in the 1970s—the NATIONAL HOUSEHOLD SURVEY ON DRUG ABUSE, the DAWN system (or DRUG ABUSE WARNING NETWORK), and the HIGH SCHOOL SENIOR SURVEY—did not yet exist, but the rising rate of heroin-related deaths in several major cities and the thousands of addicts waiting for treatment because there was not enough treatment capacity gave stark evidence for the growing size of the heroin problem. There were drug OVERDOSE (OD) deaths among U.S. troops in Vietnam also. Surveys generally indicated widespread drug use among U.S. servicemen in Vietnam, with the extent of the problem estimated at 15 to 30 percent, but it was not known if these estimates were of drug users or of addicts.

In addition to the mandate to coordinate all the demand side drug-abuse activities of the federal bureaucracy so as to reduce overlap and redundancy and to expand treatment capacity, some of the additional tasks of the office included overseeing and coordinating the Vietnam drug-abuse intervention; creating a new federal agency with competence to develop national policy; creating the data systems by which the effectiveness of national policy could be evaluated; creating a science base so that research might lead to better ways to treat and prevent addiction; and developing a formal, written National Strategy for drug-abuse treatment and prevention.

Four major policy changes helped the agency achieve its objectives. The first was made by the president when the Vietnam testing and treatment program was initiated: Drug use was no longer a court martial offense. The second was having the federal government take responsibility for developing and funding treatment. The third made METHADONE-MAINTENANCE treatment, already being used for 20,000 people, an established and acceptable treatment method rather than an experiment. The fourth had to do with changes that were made in the thinking, language, and means by which treatment was supported.

A central effort for SAODAP was the expansion of treatment capacity, increasing not only the number of programs, but also their actual capacity and geographic distribution. In addition, recipients of funding for treatment programs became accountable for what they provided, such as the number of treatment slots and the type of treatment. While legitimizing methadone-maintenance treatment and developing regulations for its use were highly visible and highly controversial activities, they were only incidental to the overall mission of making effective treatment central to the nation's response to the drug problem. Within the first 18 months of SAODAP's efforts, the number of communities with federally supported drug-treatment programs increased from 54 to 214, and the number of programs grew to almost 400. More federally supported treatment capacity was developed within two years than over the previous fifty years.

Some of the other projects SAODAP initiated, funded, or grappled with were the Vietnam drug

intervention and the Vietnam drug intervention follow-up study; the development of confidentiality regulations to protect the medical records of people seeking treatment; funding clinical research on new pharmacological treatments for drug dependence; initiating with other agencies projects such as TREATMENT ALTERNATIVES TO STREET CRIME (TASC), research centers for clinical and basic research on drug abuse and addiction, the Career Teachers program that incorporated drug abuse into medical school curricula, and a National Training Center. SAODAP introduced formula or block grants that gave money through the NATIONAL INSTITUTES ON MENTAL HEALTH (NIMH) to the states for treatment and prevention programs; it also introduced management concepts and language into treatment systems. SAODAP played a major role in improving drug-abuse treatment in the Veterans Administration; establishing laboratory standards for urine-testing facilities; and initiating several of the epidemiological tools that continue to shape policy, such as the National Household Survey of Drug Abuse and the Drug Abuse Warning Network (DAWN) system. Many of the programs and activities developed with interagency cooperation were implemented by the agencies involved in the collaboration. Many of the activities are ongoing in the mid-1990s. SAODAP also produced the first written national strategy, entitled "Federal Strategy for Drug Abuse and Drug Traffic Prevention."

Since the baseline funding for drug-abuse treatment, prevention, and research was so low in 1971, the new resources given to SAODAP for the task represented a manyfold increase—and in some instances were the very first resources available for the purpose. The same legislation that authorized SAODAP provided for the establishment of the National Institutes on Drug Abuse (NIDA); in addition, the resources and policies for an invigorated research effort were put into place over the three budgetary cycles that preceded NIDA's creation. Dr. Robert Dupont, who succeeded Dr. Jaffe as director of SAODAP, became the first director of NIDA. Dr. Peter Bourne and Mr. Lee Dogoloff, both of whom worked at SAODAP during the first two years, later became key advisors on drug policy to President Jimmy Carter.

A noted researcher, Dr. Solomon Snyder, credits the SAODAP support he received with enabling him to discover the opiate RECEPTOR a year or two later. This discovery forms the basis for much of the neuroscience research into understanding the biology of drug dependence.

SAODAP was able to change the national response to illicit drug use by developing an infrastructure for treatment that is largely still in place, one that recognizes the heterogeneity of the drug-using population, their need for several different types of treatment, and the need for research on the efficacy of treatment. For a brief period after SAODAP's mandate expired in 1975, drug-abuse policy was coordinated by a smaller office within the Office of Management and Budget (OMB) under President Gerald R. Ford, and then by the Drug Abuse Policy Office within the White House under presidents Jimmy Carter and Ronald W. Reagan. However, until President George H. Bush established the Office of National Drug Control Policy (ONDCP), there was no formal agency with substantial authority for coordinating federal drug policy.

(SEE ALSO: *Industry and Workplace, Drug Use in*)

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Substance Abuse and Mental Health Services Administration (SAMHSA) This Agency, established by Congress on October 1, 1992 (Public Law 102-321), works with States, communities and organizations to strengthen the Nation's capacity to provide substance abuse prevention, addiction treatment and mental health services for people experiencing or at risk for mental and substance abuse disorders. The newest agency of the U.S. Department of Health and Human Services, SAMHSA's fiscal year 2000 budget is approximately $2.6 billion; it employs a staff of approximately 550.

The Agency houses three programmatic Centers: the Center for Substance Abuse Prevention (CSAP), the Center for Substance Abuse Treatment (CSAT), and the Center for Mental Health Services (CMHS). SAMHSA also includes an Office of the Administrator, an Office of Applied Studies, and an Office of Program Services.

Grant portfolios include both block and discretionary grants. Block grants enable States to maintain and enhance their substance abuse and mental

health services. Targeted Capacity Expansion grants give communities resources to identify and address emerging substance abuse and mental health service needs at their earliest stages. SAMHSA's Knowledge Development and Application discretionary grants implement and assess new community-based prevention and treatment methods.

The Center for Substance Abuse Prevention (CSAP) is the Nation's focal point for the identification, promotion, and dissemination of effective strategies to prevent drug and alcohol abuse, and the use of tobacco. CSAP programs identify prevention strategies-such as targeted family and community strengthening-that work best for specific populations at risk of substance abuse. Program approaches emphasize both cultural relevance and competence. The Center oversees Federal workplace drug testing programs as well as State implementation of the Synar youth tobacco access reduction law. Finally, CSAP supports the National Clearinghouse for Alcohol and Drug Information (NCADI), the Nation's largest information source on substance abuse research, treatment, and prevention. NCADI's toll-free number is 1-800-729-6686; its Internet address is: www.health.org.

The Center for Substance Abuse Treatment (CSAT) is enhancing the quality of substance abuse treatment services and working to ensure that services are available to everyone who need them. It supports the identification, evaluation and dissemination of science-based, effective treatment services. CSAT administers the State Substance Abuse Prevention and Treatment block grant and undertakes knowledge development, education, and communications initiatives that promote best practices in substance use/abuse treatment and intervention. CSAT's Targeted Capacity Expansion Program–and its specialized program focused on HIV/AIDS services–help communities respond rapidly to emerging local drug use trends.

SAMHSA's Center for Mental Health Services (CMHS) works to improve the availability and accessibility of high-quality care for people with or at-risk for mental illnesses and their families by creating a nationwide community-based mental health service infrastructure. Its education programs are helping to end the stigma associated with these illnesses. While the largest portion of the Center's annual budget supports the Community Mental Health Services Block Grant Program to States, CMHS also supports grant programs to develop and apply knowledge about best community-based practices designed to serve adults with serious mental illnesses and children with serious emotional disturbances. The Center also collects and analyzes national mental health services data to help inform future services decision-making. CMHS's information clearinghouse—the Knowledge Exchange Network (KEN)—can be reached by toll-free telephone (1-800-789-2647) and on the Internet at www.mentalhealth.org.

While SAMHSA's Office of the Administrator and Office of Program Services are primarily administrative in nature, the Office of Applied Studies (OAS) has program authority to gather, analyze, and disseminate data on substance abuse practices in the United States. OAS directs the annual *National Household Survey on Drug Abuse, the Drug Abuse Warning Network, and the Drug and Alcohol Services Information System,* among other studies. Through these studies, SAMHSA is able to identify trends in substance abuse and, soon, also in mental health care. OAS also coordinates evaluation of models developed through SAMHSA's knowledge development and application programs.

New program topics are identified by SAMHSA in varying ways. Some are developed by SAMHSA leadership and staff; others result from Congressional mandate. Still other topics grow from Center-sponsored meetings that highlight empirically validated, intervention models ripe for replication. Some new program directions originate at the State and local levels, some from SAMHSA and Center National Advisory Councils, and some from the research community.

Programs are bringing new science-based knowledge to community-based prevention, identification and treatment of mental and substance abuse disorders. The results are being measured in improved approaches to addiction treatment, substance abuse prevention and mental health services at the federal, state and community levels. Equally important, the results are being measured in the improved quality of people's lives. For further information, write to SAMHSA Office of Communications, Room 13C05, 5600 Fishers Lane, Rockville, MD 20857.

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U.S. Customs Service The U.S. Customs Service (USCS), in the Department of the Treasury, is the principal border-enforcement agency. Customs conducts a wide range of statutory and regulatory activities ranging from interdicting and seizing contraband entering the United States to intercepting illegal export of high-technology items. Customs officers also assist over forty other federal agencies with border-enforcement responsibilities, including public-health threats, terrorists, agricultural pests, and illegal aliens.

With a fiscal year 1993 budget of over $1.6 billion and 18,000 employees, Customs is a major revenue-producing agency; it collected $21.5 billion in duty, taxes, and fees in 1993.

CUSTOMS ROLE IN DRUG ENFORCEMENT

Customs is both a leader and a major player in stopping drug contraband from entering the United States. Approximately $570 million of the 1993 Customs budget was related to antidrug operations. Customs' inspection and control function is directed at stopping illegal entry of drugs and other contraband while accommodating the normal traffic of persons and cargo entering the United States and enforcing export laws.

As the federal lead agency at U.S. ports of entry, Customs inspects individuals, conveyances, mail, and cargo entering the United States at these ports (land, sea, and air). Customs has broad search and seizure authority at the U.S. borders and handles enormous workloads; for example, some 450 million international travelers arrive at U.S. borders each year. Customs operates a comprehensive computerized border information system and uses other domestic and international drug-intelligence networks. Priority efforts are targeted on illegal traffic in precursor chemicals, improving interdiction intelligence, and special high-intensity enforcement operations, particularly along the southwest border.

As a large, multipurpose border-control agency, Customs has considerable flexibility in determining the most effective means to meet its responsibilities. The traditional approach involves the physical presence of uniformed officers at the border to detect and seize violators and contraband. Customs emphasizes development of the best possible detection capabilities and information systems, including drug-sniffing DOGS, electronic chemical detectors, advanced computer systems, and sophisticated surveillance equipment. Reflecting the high priority for drug interdiction, over 650 National Guard personnel in twenty-seven states have been assigned to assist Customs with inspection of containerized cargo, vessels, and aircraft.

Customs has also developed major aviation and marine interdiction programs since the 1970s. Initially dependent on aircraft borrowed from the Department of Defense (DOD) and seized from smugglers, Customs now operates over 130 aircraft and 150 vessels. Customs supports a series of Command, Control, Communications, and Intelligence Centers (known as C3I) to provide coordinated tactical control for air interdiction. Using sophisticated aircraft, helicopters, and vessels, Customs works closely with the U.S. Coast Guard and U.S. military forces in providing surveillance, interception, and deterrence against drug smuggling by air and sea.

In addition to the tactical interdiction program, Customs conducts investigations of financial reporting and smuggling violations, developing both criminal and civil cases. USCS is represented in various interagency enforcement task forces.

Customs is an active participant in developing federal drug policy and has used its high public visibility to contribute to national drug-abuse prevention efforts, emphasizing "user responsibility" and drug education. Historically, Customs has provided staff assistance to executive and congressional drug-policy offices and committees. The Customs commissioner was included in the Executive Office of the President (EOP) drug-policy coordinating activities, including the Principals' Group, the Oversight Working Group, the National Narcotics Border Interdiction System, and others. The commissioner of Customs chairs the Office of National Drug Control Policy's (ONDCP) Border Interdiction Committee, with subcommittees that develop and guide the implementation of strategies for air, land, and sea interdiction. Customs also works with the international Customs Coordinating Council in developing new procedures and techniques.

(SEE ALSO: *Anslinger, Harry J., and U.S. Drug Policy; Drug Interdiction; International Drug Supply Systems; Operation Intercept; Zero Tolerance*)

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U.S. Public Health Service Hospitals In 1929, President Herbert C. Hoover signed a law enacted by the U.S. Congress to establish two federal institutions for treatment of narcotic addiction. The principal purpose of the institutions was to confine and treat persons addicted to narcotic drugs who had been convicted of offenses against the United States. However, the law also provided for voluntary admission and treatment of addicts who were not convicted of any offense. The two institutions were named U.S. public health service hospitals. One was opened in 1935 at Lexington, Kentucky, and the other in 1938 at Fort Worth, Texas. The Lexington hospital had a capacity of 1,200 patients; the Fort Worth hospital could accommodate 1,000 patients. From opening to closure in 1974, the hospitals admitted over 60,000 narcotic addicts; because of readmissions, the total admissions exceeded 100,000. Most of the admissions were voluntary. The term *narcotic addiction* has been replaced in modern diagnostic terminology by the term *opioid dependence*, but in this discussion the older term is retained because it was regularly used during the era reviewed here. The history of the hospitals is divided into three periods.

FIRST PERIOD, 1935–1949

From the start, the hospitals were designed to treat not only the physical dependence but also the mental and emotional problems thought to be related to addiction. This was an advanced conception, for treatment of narcotic addiction until then had been focused almost exclusively on the PHYSICAL DEPENDENCE. The initial treatment programs at both hospitals emphasized residence in a drug-free environment for at least six months, during which time the patient could not only recover from the physical dependence but perhaps also overcome the mental difficulties or learn to adapt to them without using drugs. While all patients received psychological help in the form of encouragement and persuasion, only small numbers received formal psychotherapy. That was because few of the staff were trained in psychotherapy. All patients considered physically able had work assignments, and all had access to educational and vocational services, recreation, and religious activities. Treatment of voluntary patients was hindered because most left during or shortly after WITHDRAWAL treatment (often to return to lower doses of their drug—before readmission). In 1948, the research division of the Lexington hospital reported that a new synthesized narcotic drug called METHADONE was effective in the treatment of opiate withdrawal. Methadone substitution followed by a gradual decrease of its dose subsequently became the standard treatment for morphine and heroin withdrawal in the United States. Also in 1948 the research division of the Lexington hospital was administratively separated from the hospital, renamed the Addiction Research Center (ARC) and made a part of the National Institute of Mental Health (NIMH).

SECOND PERIOD, 1950–1966

After World War II, the prevalence of HEROIN addiction in the United States markedly increased. Heroin replaced morphine as the primary narcotic used. Annual admissions to the two hospitals doubled from the 1940s to the 1950s. The prewar addicts differed from their postwar counterparts. More of the postwar addicts came from large cities, and more came from minority groups (mainly black and Hispanic).

While residence in a drug-free environment continued as a major feature, new psychosocial treatments were made a part of the program. Psychoanalytically oriented PSYCHOTHERAPY was offered, but few patients seemed willing or able to engage in this form of therapy. Group therapy, however,

seemed more acceptable, and most patients participated in it to some extent. Influenced by new concepts of the therapeutic community, staff members tried to improve the quality of the patients' psychosocial experience in the hospital.

THIRD PERIOD, 1967–1974

In 1967, a research mission was assigned to the two hospitals, and each was renamed a National Institute of Mental Health Clinical Research Center. Before the research mission could be developed, however, a new clinical mission was assigned to the two institutions. The NARCOTIC ADDICT REHABILITATION ACT (NARA), enacted in 1966, provided for the CIVIL COMMITMENT of addicts instead of prosecution on a criminal charge, or sentence after conviction, or by petition with no criminal charge. The law authorized the Public Health Service to enter into contracts with any public or private agencies to provide examination or treatment of addicts committed under the NARA, but it was decided to use the two clinical research centers to implement the act quickly. Admission of prisoners and voluntary patients was phased out, and the centers concentrated on service to the NARA patients. From 1967 through 1973, over 10,000 NARA patients were admitted to the two centers. Nearly all were admitted under the provision of the law that permitted commitment with no federal criminal charge.

The NARA civil commitment seemed a promising way to eliminate the problem of voluntary patients who signed out prematurely. In practice, it only reduced the problem. Patients learned that commitment could be avoided or terminated if they refused to participate in treatment activities or engaged in disruptive or antagonistic behavior. Only about one-third of the NARA patients completed a six-month period of institutional treatment.

The NARA program led to the closure of the two centers. As more contracts were made with local facilities for examination and treatment of NARA patients, admissions to the two centers decreased. In addition, a new federal program, started in the late 1960s, of grants to states and communities for drug-abuse treatment programs made the centers less needed. The Fort Worth Center was closed in 1971 and the Lexington Center in 1974. The facilities were transferred to the Federal Bureau of Prisons and were converted into correctional institutions.

HISTORIC ROLES OF THE HOSPITALS

For approximately three decades, from the 1930s into the 1960s, the two Public Health Service hospitals were almost the only institutions in the United States engaged in the study and treatment of narcotic addiction. They became international centers of expertise. Staff members published many reports on the psychosocial characteristics of the addicts, the treatment programs, treatment outcomes, and related topics. Many clinicians and investigators who worked at Lexington and Fort Worth left these institutions to become leaders in treatment of or research on narcotic addiction at other locations. Despite great efforts, however, the hospitals failed to develop an enduring cure for narcotic addiction. Hospital treatment often produced a temporary remission in the addiction, but relapse within a year was the typical outcome.

(SEE ALSO: *Opioid Dependence; Treatment, History of; Wikler's Pharmacologic Theory of Drug Addiction*)

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V

VALIUM *See* Benzodiazepines

VALUES AND BELIEFS: EXISTENTIAL MODELS OF ADDICTION Existential models of addiction focus on beliefs, attitudes, and values of the drug users. For example, psychologists have found that problem drinkers and alcoholics anticipate greater benefits and more powerful effects from drinking than do other drinkers. These beliefs *precede* actual drinking experiences (Miller, Smith, & Goldman, 1990).

Beliefs about oneself and about the role of drugs or alcohol in one's life are sometimes called existential models (Greaves, 1980). Khantzian (1985) has proposed that addicts use drugs to offset or address specific problems they believe they have, such as a lack of confidence in social-sexual dealings, a view sometimes referred to as the adaptive model of addiction. According to Peele (1985), the individual becomes addicted to a substance because it fulfills essential intrapsychic, interpersonal, and environmental needs.

Views about oneself in regard to a substance-abuse problem are crucial for dealing with this problem. If the client and treatment personnel see the problem differently, in viewing it as a disease or not, for example, treatment will generally not succeed.

CULTURAL BELIEFS IN ADDICTION

Cultural differences are among the most powerful determinants of the patterns of substance use and the proclivity to addiction (Heath, 1982). For example, moderate drinking is inculcated as an early and firm cultural style among Mediterranean ethnic groups, the JEWS and the CHINESE. Such cultural socialization incorporates beliefs about the power of ALCOHOL and the nature of those who overindulge or misbehave when drinking. Groups such as the Irish, which invest alcohol with the power to control and corrupt their behavior, have high levels of ALCOHOLISM (Vaillant, 1983). In contrast, Jews, Italians, and Chinese believe that those who overdrink are displaying poor self-control and/or psychological dependence, rather than responding to the power of the alcohol itself (Glassner & Berg, 1984). Similar cultural variations occur in views toward drugs such as MARIJUANA, NARCOTICS, PSYCHEDELICS, and COCAINE.

Cultural recipes for moderate consumption of alcohol and other drugs have been developed, although systematic cross-cultural empirical support for these models is weak. One cross-cultural survey of addictive (loss-of-control) behavior is MacAndrew and Edgerton's (1969) *Drunken Comportment*, which describes cultural beliefs that encourage overconsumption and drunken excesses. Yet cultural attitudes about alcohol and other drugs in relation to their misuse are generally regarded as

cultural oddities, rather than scientifically meaningful factors in models of addiction.

VALUES

If individual and cultural beliefs have been given short shrift in addiction theories, then values have been considered in such models primarily as illustrations of moralistic prejudice.

Whereas a layperson might condemn the values of a mother who uses drugs or drinks excessively during pregnancy or of a person who assaults others when drunk or using drugs, some pharmacologically based theorists instead emphasize the potency of the drug and the irrevocable need of the person to obtain the drug at the cost of any other consideration whatsoever.

Peele (1987) turned this model on its head—claiming that people become addicted due to a failure of other values that maintain ordinary life involvements. In Peele's view, personal values influence whether people use drugs, whether they use them regularly, whether they become addicted, and whether they remain addicted. These values included prosocial behavior (including achievement, concern for others, and community involvement), self-awareness and intellectual activity, moderation and healthfulness, and self-respect. Evidence for the role of values in addiction are the explicit values people cite as reasons for giving up addictions to cocaine, alcohol, and nicotine (Reinarman, Waldorf, & Murphy, 1991).

(SEE ALSO: *Addiction: Concepts and Definitions; Adjunctive Drug Taking; Asia, Drug Use in; Causes of Substance Abuse; Expectancies; Religion and Drug Use*)

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STANTON PEELE

VENTRAL TEGMENTAL AREA The ventral tegmental area, (VTA), is a very important brain area in the field of drug abuse. It is one of only two main areas that contain DOPAMINE cell bodies. The MESOLIMBIC DOPAMINE pathway originates in the VTA. Dopamine neurons in the VTA project to areas of the brain associated with emotion and motivation, the so-called limbic areas. However, the projection to the NUCLEUS ACCUMBENS is the most important in understanding the action of drugs of abuse, especially psychostimulants. In addition, neurons in the nucleus accumbens and other limbic areas project to the VTA, providing the substrate for many neurochemicals to modulate the dopamine cells in the VTA.

There are two main experimental paradigms used in animals to assess the effects of drugs and endogenous neurotransmitters, such as DYNORPHIN, on these dopaminergic cells at the level of the VTA. Chemicals can be injected directly into the VTA in order to study their effects. Conditioned place preference is a method, which allows the animal to be tested for the REINFORCING properties

of a chemical in a drug free state. In addition, increases in locomotor activity can be measured, psychomotor stimulants in addition to being rewarding increase locomotor activity, and one substrate underlying this increase is the VTA.

The most extensively studied drugs of abuse, psychostimulants and opiates, both interact with the mesolimbic dopamine system. Future studies fully elucidating the modulation of VTA dopamine neurons will greatly contribute to the understanding of the mechanism of action of drugs of abuse, and may lead to the development of medications to treat drug abusers.

STEPHANIE DALL VECCHIA-ADAMS

Two American GIs exchange vials of heroin in their living quarters in Quang Tri Province, South Vietnam. (© Bettmann/CORBIS)

VIETNAM: DRUG USE IN In the spring of 1971, two members of Congress (John Murphy and Robert Steele) released an alarming report alleging that 15 percent of U.S. servicemen in Vietnam were addicted to HEROIN. The armed forces were attempting to cope with the drug problem by combining military discipline with "amnesty." Anyone found using or possessing illicit drugs was subject to court martial and dishonorable discharge from the service; but drug users who voluntarily sought help might be offered "amnesty" and brief treatment. This policy apparently was having little impact, since heroin use had increased dramatically over the preceding year and a half.

Because the United States was trying to negotiate settlement of the war, military forces in Vietnam were being rapidly reduced. About 1,000 men were being sent back to the United States each day, many of them to be discharged shortly thereafter to civilian life. If the reported rate of heroin addiction among servicemen were accurate, this rapid reduction in force meant that hundreds of active heroin addicts were being sent home each week. Concerned about the social problems that could ensue from such an influx of addicts, President Richard M. Nixon charged his staff with seeking an effective response. Domestic Council staff members Jeffrey Donfeld and Egil Krogh, Jr., sought advice from Dr. Jerome H. Jaffe, then on the faculty of the University of Chicago, who had previously prepared a report for the president on the development of a national strategy for the treatment of drug dependence. Dr. Jaffe recommended a radical change in the policy for responding to the problem of drug use in the military. The suggested plan included urine testing, to detect heroin use, and treatment rather than court martial when drug use was detected. President Nixon endorsed the plan and the military responded with such remarkable rapidity that, on June 17, 1971, less than six weeks from the time it was proposed, the plan was initiated in Vietnam.

In fact, there was no way to know whether the new approach would be better than the old one, no reliable information on the actual extent of drug use and addiction, and no solid information on which to base estimates of how many servicemen would require additional treatment after discharge. To obtain information on the extent of drug use, the effectiveness of treatment, and the relapse rates it would be necessary to find and interview the servicemen at time of discharge and at various intervals after discharge.

In June 1971, President Nixon also announced the formation of the SPECIAL ACTION OFFICE FOR DRUG ABUSE PREVENTION (SAODAP) charged with coordinating the many facets of the growing drug problem and named Dr. Jaffe as its first director. One of the first tasks of the office was to evaluate the results of the new drug policy for the military, especially as it was implemented in Vietnam. SAODAP arranged for Dr. Lee Robins, of Washington University in St. Louis, to obtain records from the Department of Defense and the Veterans Administration to conduct the study. The findings on drug use prior to and during service are summarized here. The drug-using behaviors of the servicemen after their return to civilian life are de-

scribed in a separate article (see VIETNAM: FOLLOW-UP STUDY).

Around 1970, before going overseas, about half the army's enlisted men had had some experience with illicit drugs. However, only 30 percent had tried any drug other than MARIJUANA. At that time, the most common civilian drugs other than marijuana were BARBITURATES and AMPHETAMINES. Before going to Vietnam, only 11 percent of soldiers had tried an OPIATE, and those who did so generally took cough syrups containing CODEINE, not heroin or OPIUM.

The men sent to Vietnam had either been drafted or had enlisted. Toward the end of the war, when drug use in the United States was highest, draftees were chosen by a lottery designed to make selection less susceptible to social-class biases. This produced draftees who were a reasonably representative sample of young American men. Those who enlisted voluntarily, however, who made up about 40 percent of the armed forces, were disproportionately school dropouts. Many of them enlisted before reaching draftable age because of their limited occupational opportunities. They also arrived in Vietnam with considerably more drug experience than the draftees.

Men who were sent to Vietnam before 1969 found marijuana plentiful but little else in the way of illicit drugs (Stanton, 1976). Some amphetamines were available—in part, because the military issued them to help men stay alert on reconnaissance missions. In 1969, heroin and opium began to arrive on the scene, and by 1970–1971 these opiates were very widely available. Marijuana was still the most commonly used illicit drug, but opiates outstripped amphetamines and barbiturates in availability. Heroin and opium were relatively cheap and very pure, so pure that the soldiers could get ample effect by smoking heroin in combination with TOBACCO or marijuana. This made opiates appealing to men who would have been reluctant to inject them.

At the height of the use of opiates, in 1971, almost half the army's enlisted men had tried them; of those who tried them, about half used enough to develop the hallmarks of addiction—TOLERANCE and WITHDRAWAL symptoms (Robins et al., 1975). Marijuana use was even more common; about two-thirds of these soldiers used it. The estimates come from an independent survey of a random sample of army enlisted men eight to twelve months after their return from Vietnam, after the great majority had been discharged (Robins et al., 1975). Previous studies in Vietnam (Stanton, 1972; Roffman & Sapol, 1970; Char, 1972) or among men still in service after return (Rohrbaugh et al., 1974) were less reliable, because of difficulties in collecting a random sample, use of questionnaires rather than interviews (which can lead to careless responses or failure to answer completely), and because the surveys were being done by the army itself, while the men were still subject to possible disciplinary action.

The standard tour of duty for Vietnam soldiers was twelve months. Drug use typically began soon after arrival in Vietnam, showing that it was not at all difficult to find a supplier. Older men used less than younger soldiers, career soldiers less than those serving their first term. Drug experience before induction was a powerful predictor of use in Vietnam (Robins et al., 1980). Essentially all those with drug experience before enlistment used drugs in Vietnam. Of course, there were also some soldiers who used drugs there for the first time.

One interesting observation was that men who drank ALCOHOL in Vietnam tended not to use opiates, and opiate users tended not to drink (Wish et al., 1979). This is a very different pattern from the one seen in the same men both before and after Vietnam, when drinkers were much more likely to use illicit drugs than abstainers.

Soldiers who used drugs had more disciplinary problems, on average, than those who abstained. However, the great majority of drug users received little or no disciplinary action and were honorably discharged. Although there were instances in which drug use impaired a soldier's combat readiness, evidence is lacking that it had much impact on soldiers' ability to carry out orders or wage war.

(SEE ALSO: *Addiction: Concepts and Definitions; Drug Testing and Analysis; Military, Drug and Alcohol Abuse in the U.S.*)

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VIETNAM: FOLLOW-UP STUDY In the summer of 1971, the U.S. military forces in Vietnam were being rapidly reduced. To deplete the forces there quickly, many men were being sent home before the usual tour of twelve months was complete. A urine-screening program was established in July to detect the recent use of illicit drugs by men scheduled to depart Vietnam for the United States. Those detected as positive were kept for DETOXIFICATION for about seven days, retested, and sent home only if they had a negative test. The urine screening was initiated in response to great concern that many members of the military had become addicted to HEROIN in Vietnam. The fear was that they might continue their addiction in the United States. Because the great majority of those returning were due for discharge on return, the MILITARY would have no further control over them. They might present overwhelming problems to the legal system and to veterans' hospitals.

To learn whether this fear was justified, the SPECIAL ACTION OFFICE FOR DRUG ABUSE PREVENTION (SAODAP) launched a follow-up study with the collaboration of the Department of Defense, the Veterans Administration, the National Institute of Mental Health, and the Department of Labor. The goal was to learn how many men had actually been addicted in Vietnam, whether those addicted would continue to use heroin after return and how many would be readdicted after return. The study was conducted by Washington University in St. Louis, with Lee N. Robins, Ph.D., as principal investigator (Robins, 1973, 1974; Robins et al., 1975).

The group believed to be most at risk of addiction was army enlisted men, who spent their whole tour of duty on Vietnam soil, rather than on ships or in the air like men in the navy or air force. Thus, two groups of 500 army enlisted men were selected for the follow-up, a random sample of men returning in September 1971, and a sample of men whose urines had been positive when tested just prior to departure for the United States that month. The overlap between the two groups selected made it possible to estimate what proportion of all army enlisted men had tested positive. Military records of all those selected were reviewed to verify the date of their departure from Vietnam and to obtain a civilian address and the names of close relatives who would know where to contact them. Records were also used to verify the men's reports of drug problems in the service. To protect from subpoena the confidentiality of the information given by the men, a certificate of confidentiality was obtained. Then each interview was identified only by a randomly selected number placed on its mailing envelope but not on the interview proper. The interview was then mailed to another country, where a second random identification number was selected to replace the original one. A list connecting the first number to identifiers was held in the United States, and a list linking the first number to the second one was kept abroad, so that no one in either country could link names to interviews.

Almost 900 men were personally interviewed eight to twelve months after their return from Vietnam. The response rate was extraordinary: 96 percent of the sample initially selected were personally interviewed. The men were extremely frank—97 percent of men whose military record showed drug use had reported it to the interviewer. Two findings were especially surprising. First, use of narcotics in Vietnam was much more common than the military had estimated. Almost half (43%) of the army enlisted men had used heroin or opium in Vietnam, and 20 percent had been addicted to narcotics

there. Second, only a tiny proportion (12%) of those addicted in Vietnam became readdicted in the year after return (Robins et al., 1974). Follow-up again two years later showed that this low rate of readdiction continued (Robins et al., 1980). During their second and third years home, addiction rates among men drafted were not significantly greater than among men who qualified for the draft but did not serve. This surprisingly low rate of relapse could not be attributed to abstention from narcotics after return; half of those addicted in Vietnam did use again after return. Those who went back to narcotics were predominantly men who had used drugs before they entered the service.

Although the principal finding of this study was that heroin addiction in Vietnam had a much better outcome than expected, there were men whose addiction continued on return home. Treatment for them was no more effective than for men who developed addiction in the United States (Robins, 1975).

(SEE ALSO: *Addiction: Concepts and Definitions; Drug Testing and Analysis; Opioid Dependence; Treatment; Vietnam: Drug Use in*)

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VIOLENCE AND SUBSTANCE ABUSE

See Crime and Drugs; Family Violence and Substance Abuse; Gangs and Drugs; International Drug Supply Systems

VITAMINS Vitamins are organic substances that are required in small amounts for normal functioning of the body. Lack of adequate quantities of vitamins results in well-known deficiency diseases, such as scurvy from Vitamin C deficiency and rickets from Vitamin D deficiency in childhood. For the most part, vitamins are not synthesized by the body but are found in a variety of foods, hence the need for a well-balanced diet or supplementation by taking the vitamins separately.

In the United States, daily minimum requirements for vitamins are recommended, and periodically reassessed, by the Food and Nutrition Board of the National Academy of Science—National Research Council. Some professionals advocate taking larger amounts of certain vitamins is for better health or for disease prevention or therapy. The question of whether vitamins are drugs is, in one sense, a semantic issue. Sometimes, very high doses of a vitamin can actually be used as a medication. For example, in very high doses—twenty or more times higher than needed to prevent the vitamin deficiency disease pellagra—niacin, a member of the B vitamin complex, lowers blood levels of cholesterol and triglycerides and niacin is commonly prescribed for this purpose.

It is possible to OVERDOSE and have serious side effects from large quantities of certain vitamins, such as vitamins A and D. Therefore, taking larger than needed amounts of vitamins should be done only with the advice of a physician. Deficiencies in vitamin intake can occur under a variety of situations including poverty, dieting, or certain disease states where antibiotics or other factors reduce vitamin absorption. Individuals who drink large quantities of ALCOHOL, for example, without adequate attention to diet often become deficient in some vitamins, such as B_1 (thiamine), and may

require their administration to avoid serious and permanent toxicity. Prolonged serious shortages of Vitamin B_1 can cause the death of certain NEURONS in the brain, a situation that leads to confusion and severe impairment of short-term memory (the Wernicke-Korsakoff syndrome).

(SEE ALSO: *Complications*)

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VULNERABILITY AS CAUSE OF SUBSTANCE ABUSE This section contains some articles that discuss one of several *Causes of Substance Abuse*—vulnerability. In addition to an *Overview* article, the following topics are discussed as vulnerability factors: *Gender; Genetics;* the *Psychoanalytic Perspective; Race; Sensation Seeking; Sexual and Physical Abuse;* and *Stress*. For more information, see *Comorbidity and Vulnerability*, *Families and Drug Use*, and *Poverty and Drug Use*.

An Overview There are marked individual differences in drug use and abuse. Some people never use drugs although drugs may be readily available to them. Others use drugs sporadically or regularly for years but never escalate their use to drug DEPENDENCE. Others become chronic, compulsive users and have difficulty functioning without drugs. These individual differences in drug-use patterns are the result of a combination of environmental and genetic factors. Environmental factors include the experiences of an individual, such as family and social conditions, as well as other conditions under which the person lives. Genetic factors refer to the genes that are passed down from parent to child and which are shared in part by other family members.

Environmental and genetic factors combine to produce risk factors, which are influences that increase the likelihood of drug use. They may also combine to produce protective factors, which are influences that decrease the likelihood of drug use. Vulnerability refers to the sum total of an individual's risk and protective factors. It defines the overall likelihood of drug use. Individuals with many risk factors and few protective factors are more likely than individuals with few risk factors and many protective factors to use drugs.

GOALS OF VULNERABILITY RESEARCH

In vulnerability research, attempts are made to identify risk and protective factors for both drug use and drug dependence, refine existing risk and protective factors by enhancing their specificity in predicting drug use, reduce the number of risk and protective factors to their most fundamental number, and understand the environmental and genetic influences (i.e., mechanisms) that underlie risk and protective factors.

Risk-Factor Identification. A large number of risk factors for substance abuse have been reported (Table 1). They include characteristics that fall within the demographic, environmental, sociocultural, family, personality, behavioral, psychiatric, and genetic domains. Among these are POVERTY, unemployment, poor quality of education, racial discrimination, ready availability of drugs, family discord, family alcohol and drug use, sexual abuse, lack of family rituals, neuropsychological deficits, childhood aggressiveness, low self-esteem, teenage pregnancy, rebelliousness, delinquency, drug use by peers, mental health problems, and cultural alienation.

A number of protective factors for substance abuse have also been reported (Table 2); however, these are considerably fewer than the reported number of risk factors, primarily because less attention has been focused on their identification. In general, the protective factors that have been reported are the opposite of known risk factors. As such, they include an adequate income, high-quality schools, positive self-esteem, and the like.

Given the fact that a large number of risk factors are commonly present in modern society, many people possess multiple risk factors for drug use. Becoming a drug user is not an inevitable outcome for these people, however, since many individuals with multiple risk factors do not become drug users. Similarly, some individuals who are drug users or drug dependent have few risk factors.

TABLE 1
Risk Factors in Substance Abuse

Ecological Environment
- Poverty
- Living in economically depressed area with:
  - High unemployment
  - Inadequate housing
  - Poor schools
  - Inadequate health and social services
  - High prevalence of crime
  - High prevalence of illegal drug use
- Minority status involving:
  - Racial discrimination
  - Culture devalued in American society
  - Differing generational levels of assimilation
  - Cultural and language barriers to getting adequate health care and other social services
  - Low educational levels
  - Low achievement expectations from society

Family Environment
- Alcohol and other drug dependency of parent(s)
- Parental abuse and neglect of children
- Antisocial, sexually deviant, or mentally ill parents
- High levels of family stress, including:
  - Financial strain
  - Large, overcrowded family
  - Unemployed or underemployed parents
  - Parents with little education
  - Socially isolated parents
  - Single female parent without family/other support
  - Family instability
  - High level of marital and family conflict and/or family violence
- Parental absenteeism due to separation, divorce, or death
- Lack of family rituals
- Inadequate parenting and little parent/child contact
- Frequent family moves

Constitutional Vulnerability of the Child
- Child of an abuser of alcohol or other drugs
- Less than 2 years between the child and its older/younger siblings
- Birth defects, including possible neurological and neurochemical dysfunctions
- Neuropsychological vulnerabilities
- Physical handicap
- Physical or mental health problems
- Learning disability

Early Behavior Problems
- Aggressiveness combined with shyness
- Aggressiveness
- Decreased social inhibition
- Emotional problems
- Inability to express feelings appropriately
- Hypersensitivity
- Hyperactivity
- Inability to cope with stress
- Problems with relationships
- Cognitive problems
- Low self-esteem
- Difficult temperament
- Personality characteristics of ego undercontrol:
  - Rapid tempo, inability to delay gratification, overreacting, etc.

Adolescent Problems
- School failure and dropping out
- At risk of dropping out
- Delinquency
- Violent acts
- Gateway drug use
- Other drug use and abuse
- Early unprotected sexual activity
- Teenage pregnancy/teen parenthood
- Unemployment or underemployment
- At risk of unemployment
- Mental health problems
- Suicidal

Negative Adolescent Behavior and Experiences
- Lack of bonding to society (family, school, and community)
- Rebelliousness and nonconformity
- Resistance to authority
- Strong need for independence
- Cultural alienation
- Fragile ego
- Feelings of failure
- Present versus future orientation
- Hopelessness
- Lack of self-confidence
- Low self-esteem
- Inability to form positive close relationships
- Vulnerability to negative peer pressure

SOURCE: Adapted from Goplerud, E. N. (Ed.). (1990), *Breaking new ground for youth at risk: Program summaries.* (DHHS Publication No. [ADM] 89-1658). Washington, DC: Office for Substance Abuse Prevention.

TABLE 2
Protective Factors in Substance Abuse

Ecological Environment	Family Environment
Middle or upper class	Little marital conflict
Low unemployment	Family stability and cohesiveness
Adequate housing	Plenty of attention during first year of life
Pleasant neighborhood	Sibling as caretaker/confidant
Low prevalence of neighborhood crime	
Good schools	**Constitutional Strengths**
School climate that promotes learning, participation, and responsibility	Adequate early sensorimotor and language development
High-quality health care	High intelligence
Easy access to adequate social services	Physical robustness
Flexible social service providers who put clients' needs first	No emotional or temperamental impairments
	Traits of the Child
Family Environment	Affectionate/endearing personality
Adequate family income	Easy temperament
Structured and nurturing family	Autonomy
Promotion of learning by parents	Adaptability and flexibility
Fewer than four children in family	Positive outlook
2 or more years between siblings	Healthy expectations
Few chronic stressful life events	Self-esteem
Multigenerational kinship network	Self-discipline
Nonkin support network—e.g., supportive role models, dependable substitute child care	Internal locus of control
	Problem-solving skills
Warm, close personal relationship with parent(s) and/or other adult(s)	Social adeptness
	Tolerance

SOURCE: Adapted from Goplerud, E. N. (Ed.). (1990), *Breaking new ground for youth at risk: Program summaries.* (DHHS Publication No. [ADM] 89-1658). Washington, DC: Office for Substance Abuse Prevention.

Risk-Factor Specificity. Unfortunately, many risk factors are so broadly defined that they are not useful as predictors. For example, we know that males are more likely than females to use illicit drugs and that underemployed people are more likely than employed people to become HEROIN addicts. Being male or being underemployed, however, is not a useful predictor of drug use. Most males do not use illicit drugs and most underemployed people are not heroin addicts. Combining GENDER and employment status into a single risk factor (i.e., the risk factor of being an underemployed male) increases specificity somewhat, and combining these factors with other risk factors (e.g., having an ANTISOCIAL PERSONALITY disorder) increases the predictive value even more.

The problem with lack of specificity is that it leads to overinclusion of people in risk groups. Many people are thus included in a risk group who are not actually at risk of becoming drug users. For example, although being male and being underemployed are factors statistically associated with heroin addiction, it is important to remember that this is only a statistical association. Most individuals with these characteristics never become heroin addicts. Thus, underemployed males represent a category that includes a large number of individuals who are not actually at risk for heroin addiction. Increasing specificity in risk factors is important because it allows the resources for PREVENTION to be directed toward the people in greatest need. Specificity also minimizes the problem of inappropriately stigmatizing people because they have a characteristic that is statistically associated with drug use.

Fundamental Risk Factors. Because of their current lack of etiological specificity, concern has been expressed about the usefulness of the large number of risk factors that have been reported for drug use. Over seventy risk factors for drug use have been reported to date, but it is not clear if they are all independent factors. Some reported risk fac-

tors may be the product of other risk factors. For example, neuropsychological deficits may precipitate learning problems, which in turn may lead to excessive CHILDHOOD aggressiveness. Similarly, family alcohol and drug use may result in family discord, and poor-quality schools may contribute both to underemployment and HOMELESSNESS.

Other risk factors may reflect different manifestations of more basic factors. For example, rebelliousness, DELINQUENCY, and aggressiveness may reflect a more basic personality characteristic or be the result of common genetic influences. Although the actual number of basic risk factors in drug use is not known, they are certain to be fewer than the large number of risk factors reported to date. The large number of reported risk factors probably reflects the highly interrelated nature of the influences involved in drug use.

Underlying-mechanism Identification. A risk factor may itself be a product of the interaction among environmental and genetic influences, or it may only be correlated with those influences. In either case, it is useful for predicting drug use. To most efficiently prevent drug use, however, it is necessary to understand the basic mechanisms that control drug use. As one increases the specificity of risk factors and reduces them to their most fundamental number, one comes ever closer to identifying the specific environmental and genetic mechanisms involved.

At present, most risk factors are hypothetical constructs and only conceptually defined. Consequently, the risk factor does not identify the mechanisms responsible for drug use. To understand how the risk factor increases the likelihood of drug use, one must identify the mechanisms involved. For example, having drug-using peers is recognized as a risk factor for drug use (because drug use by ADOLESCENTS is frequently associated with having drug-using peers). Although the specific mechanisms mediating this influence are not definitely known, it is likely that the influence is mediated in part through drug-using peers increasing drug availability and providing social reinforcement for drug use. Similarly, coming from an impoverished environment is thought to be a risk factor for drug use because it fails to provide reinforcers as an alternative to drug use.

GENETIC influences may also underlie many risk factors for both drug use and dependence. These influences may contribute to drug use through personality characteristics (e.g., SENSATION SEEKING, risk taking) that increases the likelihood of drug use and that may be genetically determined. Genetic influences may also contribute to the development of drug dependence by altering the effects of a drug (e.g., causing greater euphoria in some people than in others). In addition, they may contribute to both drug use and dependence by being responsible for the absence of normal protective factors (e.g., failure to experience a hangover after excessive alcohol use). The specific genetic mechanisms involved will be the genes (as yet unidentified) that contribute to personality development, drug response, and other important components.

The specific mechanisms that control drug use are undoubtedly the same environmental and genetic mechanisms that control human behavior in general. The mechanisms responsible for the initial drug use and for the progression to regular use and possibly drug dependence may not be the same. Once these mechanisms are understood, however, it will be possible to more directly address risk factors for drug use by means of intervention measures. The ultimate goal of those engaged in vulnerability research is to develop efficient, cost-effective prevention programs that specifically target individuals at risk for both drug use and drug dependence.

VULNERABILITY RESEARCH STRATEGIES

A variety of strategies are available for achieving the goals of vulnerability research. They include both epidemiological and experimental studies, genetic studies, and ANIMAL RESEARCH.

Cross-sectional Epidemiological Studies. Risk factors are initially identified through their statistical association with drug use. Most of the risk and protective factors reported to date have been identified by comparing drug abusers and controls on the basis of currently existing characteristics or reports of conditions existing prior to onset of drug use. For example, individuals are divided into drug users and non-drug users on the basis of a survey, and compared as to demographic characteristics and other traits. The factors that distinguish the drug users from the non-drug users are then identified as risk factors for drug use.

This strategy permits the inexpensive identification of a large number of possible risk factors for drug use. The ability of the strategy to detect possi-

ble risk factors is limited only by the selection of characteristics to be compared. With this strategy, however, it is sometimes not clear if a characteristic existed prior to onset of drug use or developed as a consequence of drug use. Since, moreover, the reports of the preexisting conditions are often based on retrospective recall, people's memory problems as well as their attempts to justify their drug use may confound the accuracy of the self-reports. Finally, inappropriate control groups are sometimes employed whose subjects differ from drug users in important aspects (e.g., demographic and clinical features), and this confounds the research design.

Longitudinal Epidemiological Studies. A better research method for identifying risk and protective factors in drug use is the longitudinal study design. With this design, individuals are assessed for various characteristics prior to the age of risk for drug abuse and then followed over time to determine those who do and those who do not become drug users. After drug users have been identified, earlier characteristics that distinguished them from nonusers can be determined.

The advantages of this method are that the drug users and nonusers are drawn from the same population and therefore constitute appropriate comparison groups. Furthermore, because the study design is prospective, it does not rely on the retrospective recall of events or conditions that might have existed prior to the onset of drug use and therefore might be confounded by incorrect memory or other problems. Finally, because this design provides for initial assessment of the subjects prior to the onset of drug use, preexisting conditions can be separated from the consequences of drug use. This design has not been widely employed, however, owing to the expense and time required to conduct the studies. There is also the problem of sample bias that might occur as a result of the attrition of subjects. For example, drug users with severe dependence or psychiatric disorders might be lost in the longitudinal follow-up process, thus leaving only the less severe drug users in the subject sample.

In general, both cross-sectional and longitudinal epidemiological strategies are useful in identifying risk factors for drug use and dependence. They are also both useful in increasing the predictive specificity of risk factors and in allowing fundamental features of various risk factors to be identified by use of sophisticated statistical modeling.

One problem that may affect both types of epidemiological studies is the failure to define risk factors operationally or objectively. This occurs less often when the risk factor involves direct measurement of the individual or use of standardized tests than when individuals are asked about a trait and no definition or operational criteria for the trait is given. For example, if subjects are asked to report on their current level of self-esteem (i.e., whether it is low, medium, or high), failure to define the concept operationally may cause confusion over its presence or absence in a given individual, and this confusion will also increase its variability across individuals.

Experimental Laboratory Studies. This strategy (termed the high-risk design) is aimed at determining the mechanism by which risk factors exert their effects. It compares two groups of individuals who are distinguished by the presence or absence of a particular risk factor. For example, the two groups might consist of children of substance abusers and children of non-substance abusers, or individuals who are depressed and individuals who are not depressed. The two groups are then compared on the basis of various dependent measures, which may include baseline characteristics (e.g., personality) or response to experimental manipulations (e.g., reaction to stress). If the two groups respond differently on a dependent measure, this suggests that the measure is a possible mechanism by which the trait is related to drug use.

This strategy has several advantages. Because it entails selecting subjects on the basis of a specific characteristic, it affords a high degree of control over extraneous factors that might confound the interpretation of epidemiological studies. It also allows researchers to measure subjects' responses directly under standard environmental conditions, rather than relying on self-reports of past events. In addition, it permits the experimental manipulation of test conditions, which in turn allows the generality of an observed effect to be determined. It also enhances the probability that the observed effect is due to the experimental manipulation. Finally, it permits mechanisms underlying the risk factors to be identified and explored, a process that can only be assessed correlationally through statistical modeling in epidemiological studies.

In contrast to epidemiological strategies, however, the high-risk strategy can only address one risk factor per study. It is further restricted by the

appropriateness of criteria used for subject selection and the experimental measures employed. For example, inappropriate subject inclusion criteria may exclude the subjects at risk, or inappropriate response measures may fail to detect group differences that are present. Laboratory studies also typically employ only a relatively small number of subjects. This small number increases the likelihood that a biased sample will result, thus making for reduced generalizability of the findings.

Genetic Studies. A number of strategies are available to determine if genetic influences are involved in drug use and dependence. Family studies determine if drug use or dependence "run in families." If higher rates of drug use are found in the relatives of drug users than in the relatives of nondrug users, then genetic influences may be involved. To separate the effects of genes and environment, however, requires doing adoption or twin studies. In adoption studies, evidence of genetic influences is provided by adoptees having higher rates of drug use if their biological parents were drug users than if their biological parents were not drug users. In twin studies, since identical (monozygotic) twins have more of their genes in common than do fraternal (dizygotic) twins, evidence of genetic influence is suggested by higher concordance rates for drug use or dependence in identical than in fraternal twins.

Other types of genetic strategies are also available. The purpose of linkage and association studies is to identify specific genes involved in drug use and dependence. In linkage studies, different generations of FAMILIES are examined to determine if a genetic marker is inherited along with a disorder (e.g., substance abuse). In association studies, individuals with and without a disorder are compared to determine the association of the disorder with a genetic marker. The previously described high-risk study designs are frequently employed in genetic research. In these studies, subjects who are not yet substance abusers are typically divided into two groups on the basis of their known risk for substance abuse (e.g., having or not having a family history of substance abuse). The two groups are then compared to identify factors that may contribute to their differences in risk for substance abuse.

Most of these genetic strategies have the same strengths and limitations previously described in regard to epidemiological and experimental laboratory studies. In addition, twin and adoption studies are based on certain assumptions about the nature of the genetic influence and parental mating characteristics that may affect interpretation of the results.

Animal Studies. Certain factors contributing to drug use and dependence can be studied experimentally only in animals. For example, it would be unethical to make a human being dependent on drugs in order to study the process of becoming drug dependent. In animals, this process can be brought under experimental control and studied directly. In human beings, drug use or dependence typically becomes evident to researchers only after it has occurred, and then the process can be studied only retrospectively.

A number of strategies are available for studying drug taking by animals. The most common of these are the animal drug self-administration methods. With these methods, animals are equipped with small tubes (catheters) that run directly from the animal's bloodstream to an injection pump located outside the cage. By pressing a lever, the animal automatically activates the injection pump and receives a predetermined amount of drug solution injected directly into the bloodstream. Similar methods are available to study self-administration of drugs by other routes. By means of these methods, it has been found that animals self-administer essentially the same drugs that humans abuse, and this has resulted in the methods being used to predict the abuse potential of new drugs before they are marketed. Keeping drugs with high dependence potential off the market is also an effective strategy for reducing people's vulnerability to drug use and dependence.

Animal drug self-administration methods can also be used to study factors that contribute to a person's acquiring the problem of drug use and dependence. With these methods, factors thought to influence vulnerability can be experimentally manipulated and studied under controlled laboratory conditions. As a result of the research, a large number of factors have been identified with animal drug self-administration methods that are relevant to the development of human drug dependence. Among these are the reinforcing property of the drug itself, the speed with which a drug is injected, the schedule of drug delivery, the availability of other reinforcers, and the aversiveness of the environment. The knowledge gained from the research

can be applied directly to human drug abuse prevention efforts.

Animal methods make possible the experimental study of factors that influence the acquiring of the habit of drug use and dependence, a process that cannot be ethically studied with human beings. Animals, however, differ from human beings in many ways that may be important in the etiology of drug abuse, and therefore care must be taken in generalizing the results of animal studies to human beings. In addition, although animal models provide an excellent way of studying behavioral and environmental factors in drug use, the approach cannot readily be used to study other risk factors (i.e., psychosocial and cultural influences) that are believed to be important in the development of drug abuse by human beings.

(SEE ALSO: *Abuse Liability of Drugs: Testing in Animals; Addiction: Concepts and Definitions; Adjunctive Drug Taking; Complications: Mental Disorders; Conduct Disorder and Drug Use; Disease Concept of Alcoholism and Drug Abuse; Epidemiology of Drug Abuse; Ethnicity and Drugs; Research, Animal Model; Wikler's Pharmacologic Theory of Drug Addiction*)

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Gender Apart from the use of TOBACCO (cigarettes) and PSYCHOACTIVE DRUGS, men show a consistently higher rate of drug use than do WOMEN, especially with reference to ALCOHOL and to MARIJUANA and other illicit drugs (Substance Abuse and Mental Health Services Administration, 1992; Anthony, 1991; Robins et al., 1984; Kandel & Yamaguchi, 1985; Windle, 1990; Robbins, 1989). Women are more likely than men to use the drugs prescribed by a physician, especially psychotrophic drugs (Cafferata et al., 1983), and although men still have a higher rate of CIGARETTE use, this difference is decreasing (Kandel & Yamaguchi, 1985; National Institute on Drug Abuse, 1989 & 1991; SAMSA, 1992).

Gender differentiation in society occurs at many levels and in the major institutions such as government, family, the economy, education, and religion, as well as in face-to-face interpersonal interaction (Giele, 1988). It is therefore not surprising that drug use behavior differs for men and women. Because of the pervasive way in which gender roles affect most aspects of people's lives, it remains a complex task to understand gender differences in patterns of drug use. It is expected that gender will influence patterns of substance use and consequences of substance abuse, in part because men and women are socialized according to different behavior patterns and values. Normative expectations for men include self-reliance and physical effectiveness. By contrast, women are taught to value close relationships and to define themselves in terms of those relationships. With regard to substance use, the literature shows that gender (a) is associated with use of alcohol and drugs; (b) is associated with a variety of psychosocial characteristics that are themselves associated with alcohol and drug use; (c) and may be associated with different etiologies of alcohol and drug use—and with different consequences of substance use and treatment outcomes. The role of gender in drug use has been demonstrated in a number of studies conducted in the United States; several of these have provided comprehensive comparisons of the psychological, social, and biological characteristics of male and female drug users (Kaplan & Johnson, 1992; Lex, 1991; Gomberg, 1986; Ray and Braude, 1986).

According to the convergence hypothesis, the increasing similarity of roles and activities of men and women, as illustrated by the increasing partici-

pation of women in the paid labor force, will result in the drug and alcohol behaviors of women increasingly approximating those of men (see Adler, 1975; Bell, 1980). Although there is some evidence that male and female ADOLESCENTS have similar drug-use behaviors, recent epidemiological data indicate that alcohol and drug problems are still more common among men than among women (Anthony, 1991). Lennon (1987) found no support for the hypothesis that women in "male" jobs resembled men in terms of their levels of drinking. In the case of cigarettes, the increasing similarity of men's and women's behavior has been the result of both women increasing and men decreasing their use of cigarettes. There is little evidence to support the theory of increasing convergence of substance use, although it should be noted that many of the early studies of alcohol or drug use included only men, so that little is known about trends in women's use (Robins & Smith, 1980; see Vannicelli & Nash [1984] for an analysis of sex bias in alcohol studies).

The various perspectives that can be used to explain gender differences in drug and alcohol use include: (1) gender role explanations; (2) the social control theory; and (3) biological explanations. Explanations that draw on gender role theories to explain male-female differences refer to normative expectations and rules regarding the behavior of males and females. According to one hypothesis, there are distinctive gender styles in expressing pathology (Dohrenwend & Dohrenwend, 1976). The male style features acting-out behaviors (including drug and alcohol use), whereas the female style involves the internalization of distress. A finding consistent with this hypothesis was that of several researchers, who observed that for females, conformity to the female identity was related to higher psychological distress and lower substance use than was observed in males (Horowitz & White, 1987; Huselid & Cooper, 1992; Snell, Belk, & Hawkins, 1987; Koch-Hattem & Denman, 1987). The evidence for males has been inconsistent, however. Although there was more alcohol and drug use among males than among females, ascribing to the conventional masculine role did not necessarily lead to more alcohol or drug problems for males.

A second explanation for gender differences in alcohol and drug use is that societal expectations differ for men and women, with the result that using illicit substances for pleasure is more acceptable in men than it is in women (Landrine, Bardwell, & Dean, 1988; Lemle & Mishkind, 1989; Gomberg, 1986). Women are more likely to use substances for therapeutic reasons, specifically for the relief of mental and physical distress, whereas men are more likely to use drugs for recreation. Surveys in which it was found that men use more illicit drugs, primarily for recreation, and women use more psychotherapeutic drugs have borne out this theory.

A closely related hypothesis that is particularly relevant to the higher use of psychotropic drugs by women is that society permits women to perceive more illness (morbidity) and to use more medical care than it does men, who are expected to be stoic in the face of illness. Survey results seem to confirm the behavioral differences suggested by this hypothesis. In a review of morbidity and mortality studies, Verbrugge (1985) found that women consulted physicians more often than men, assumed the patient's role more readily, and appeared to take better care of themselves in general. These behaviors would make women more inclined than men to use prescription drugs and less inclined to use other drugs. The increasing use of cigarettes by younger women, however, is one behavior that runs counter to this hypothesis.

According to the social control theory, those who have strong ties to societal institutions such as family, school, or work are less likely to have a problem with use of substances. This perspective stems from Emile Durkheim's classic study of SUICIDE (1898). Umberson (1987) applied Durkheim's perspective to health behaviors and showed that social ties affect the health behaviors of individuals (e.g., physical activity, alcohol consumption, compliance with doctor's recommendations, etc.) and that consequently they affect health status and mortality rates. Social ties, according to this argument, affect drug use behaviors in two ways. First, there is an increased likelihood that the behavior of those with strong social ties will be monitored by family members and friends, and this would tend to decrease use of illicit or unhealthy substances. Second, the responsibility and obligation entailed in an individual sharing strong ties and frequent activities with family and friends make for more self-regulation of behavior. Marriage and being a parent represent important social ties that may affect people's use of substances, especially in the case of women, be-

cause of their traditional roles in nurturing and maintaining family relationships.

Several studies have shown the increased vulnerability to drug use of women in relation to social ties. Kaplan and Johnson (1992) showed that the attenuation of interpersonal ties resulting from initial drug use caused women, but not men, to increase their drug use. Similarly, Kandel (1984) reported that interpersonal factors were more significant for women than for men in explaining marijuana use. Ensminger, Brown, and Kellam (1982) showed that strong family bonds inhibited drug use in female adolescents but not in male adolescents.

Physiological differences may also be important in accounting for gender differences in patterns of substance use. Mello has (1986) suggested that a woman's use of drugs and alcohol may be influenced by menstrual cycle phases (Mello, 1986), although little evidence exists for this hypothesis. Halbreich et al. (1982) examined the scores on the Premenstrual Assessment Form and found that women who increased their marijuana use at the premenstruum reported significantly greater DEPRESSION, ANXIETY, mood changes, anger, and impaired social functioning than did women whose marijuana use decreased or stayed the same.

The relatively low rate of consumption of drugs by women may be related to biological differences in the ways drugs are cleared from the body in women versus men. The lower ratio of water to total body weight in women causes them to metabolize alcohol and drugs differently (Mello, 1986; Straus, 1984). This and other biological factors may cause women to have higher BLOOD-ALCOHOL CONCENTRATIONS (BACs) than men at equal dosages (Corrigan, 1985; McCrady, 1988). Drugs that are deposited in body fat, such as marijuana, may be slower to clear in women than in men because of the higher ratio of fat in women (Braude & Ludford, 1984).

Gender roles are the major roles in human society, and they influence almost every aspect of an individual's life. Despite the evidence for gender differences in patterns of drug use, little attention has been given either to the potential strategic advantages that this observation presents for furthering our understanding of drug and alcohol use patterns in males and females, or for determining how prevention and treatment programs might be redesigned.

(SEE ALSO: *Comorbidity and Vulnerability; Conduct Disorder and Drug Use; Epidemiology; Gender and Complications of Substance Abuse*)

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Genetics Genes are passed from parent to child in the process of sexual reproduction. These genes determine some of the features of the individual and contribute directly and indirectly to many more. The possibility of genetic influences in sub-

stance abuse has received considerable attention. Evidence that genetic influences may be involved comes from family studies, where substance abuse has been found to run in families. For example, alcoholics have been found to have more relatives who are alcoholic than would be expected from the base rate for ALCOHOLISM in the general population. Similarly, higher rates of HEROIN and COCAINE abuse are also seen in the relatives of heroin and cocaine abusers than occur in the general population.

Both twin and family studies have been conducted to separate genetic from environmental influences in the familial transmission of substance abuse. Most of the research has involved ALCOHOL. There is general agreement that genetic influences are involved in both alcohol use and alcoholism, at least for males. Twin studies of males from the general population have found that if one pair member drinks alcohol, the other pair member is more likely to drink (i.e., they are concordant for this behavior) if the two members shared all the same genes (if they are monozygotic or identical twins) than if they share only about half of their genes (if they are dizygotic or fraternal twins). Similar studies on clinical patients have found higher concordance for alcoholism among men who are monozygotic rather than dizygotic twins. Adoption studies have found that sons of alcoholic biological parents were more likely to be alcoholic as adults than sons of nonalcoholic biological parents, when both groups were adopted out early in life and raised by nonalcoholic adoptive parents. Among men, estimates of the proportion of variance in alcohol-dependence liability due to genetic influences (i.e., heritability) range from 0.50 to 0.60, depending on the subject population and subtype of alcoholism.

For women, the role of genetic factors in alcohol use and alcoholism is less convincing. This is primarily because women have been studied less often than men and in smaller numbers. One reason for this discrepancy is that women are less likely to have alcohol problems, and this fact itself may reflect the greater role of nongenetic influences for women. In twin and adoption studies involving women, evidence of genetic influence has been found less consistently than has been found for men, with heritabilities for women ranging from 0.00 to 0.56. depending on the study. Nevertheless, women have similar percentages of same- and opposite-sex alcoholic relatives as do men, and this suggests that there is no differential heritability related to gender.

Although less frequently studied, genetic influences for other forms of drug use and dependence have also been shown, but only males have typically been studied in this context. Heritabilities reported for tobacco smoking range from 0.28 to 0.84 and are not affected by other factors that may contribute to differences in concordance rates in twins. Heritabilities reported for other types of illicit drug use (but not necessarily drug dependence) range from 0.4 to 0.6. Heritability for any substance abuse or dependence (excluding alcohol and tobacco) in alcoholic probands is 0.31.

Linkage and association studies permit the identification of specific genes involved in substance abuse. In linkage studies, different generations of families are examined to determine if a genetic marker is inherited along with a disorder (e.g., substance abuse). In association studies, individuals with and without a disorder are compared to determine the association of the disorder with a genetic marker. To date, no specific gene for alcoholism or for other types of drug dependence has been identified.

Animal models have also been employed to study genetic influences in substance abuse. Evidence of significant genetic influence has been found in the characteristics of many drug responses relevant to drug abuse (e.g., drug preference), and chromosomal loci have been identified that mediate at least some of these effects. To the extent that the genetic structure of mice is similar to that of human beings, the findings derived from animal models suggest testable hypotheses to be explored in human-association studies. In strains of rats that were bred in laboratories to study their preference for alcohol, the strain that developed a strong preference for alcohol had lower brain levels of the NEUROTRANSMITTER serotonin compared to the strain that did not prefer alcohol. This is of interest because alterations in SEROTONIN neurotransmission have also been noted in studies of impulsive aggressive human males (who have a higher likelihood of developing alcohol or drug problems) compared to human males without those behaviorial traits.

(SEE ALSO: *Attention Deficit Disorder; Causes of Substance Abuse; Conduct Disorder and Drug Use;*

Disease Concept of Alcoholism and Drug Addiction; Epidemiology of Drug Abuse)

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Psychoanalytic Perspective Increased vulnerability to ALCOHOL and drugs is related to the coming together of a number of influences, each of which is itself of varying strength. Our biologies, our individual social and cultural settings and backgrounds, our personal idiosyncratic life experiences, and the persons we become as a result of all these may contribute to the likelihood of our using drugs—and then of our continuing to use them. We are neither vulnerable nor invulnerable to using drugs or alcohol, nor to using them to excess; vulnerability is a continuum, ranging from least to most vulnerable. Under the right, or the wrong, circumstances, many of us will use drugs.

ALCOHOLISM runs in families; if an individual's parent, grandparent, or sibling is alcoholic, that individual's own risk is significantly increased. It seems certain that an important contributor to this in many families is GENETIC. While we find a similar increase in the frequency of substance abuse in the children of parents who use all sorts of drugs, we do not yet have evidence that this too is genetic. Certainly, another contributor to this familial pattern is the exposure that a developing child has to the sight and experience of a parent or other important figure in the environment using alcohol and/or other drugs. It tells the child that this is acceptable behavior, particularly if the surrounding social culture echoes that opinion. Cultures and subcultures that traditionally control drinking generally produce people who drink in a controlled way; cultures and subcultures that condone excess also reproduce themselves.

It is important to remember, however, that even those with a strong genetic loading for alcoholism can only become a "practicing" alcoholic if they have alcohol available. Despite its many problems, Prohibition (1920–1933) reduced the number of alcoholics; successful interdiction of drugs would reduce the number of substance abusers. However, growing up in an area where drugs are freely available increases the likelihood of trying them and—assuming community complacence or peer approval and encouragement—of continuing to take them. For example, during the war in VIETNAM, many U.S. soldiers who had not been OPIATE addicts found themselves in the war zone, exposed to STRESS and personal danger, and surrounded by cheap available HEROIN in a context that condoned its use. Many became addicted. On their return home, however, almost all gave up their drug use with relative ease.

We also know that the person one is—the kind of *personality* one has—also plays a role in one's susceptibility to using and misusing drugs. A number of studies suggest that maladjustment precedes the use of illicit drugs; the closer one is in style to an Eagle Boy Scout, the less likely one is to use drugs. Rebelliousness, stress on independence, apathy, pessimism, DEPRESSION, low self-esteem, and low academic aspirations and motivation make the use of illicit drugs more likely. Delinquent and deviant behavior come before the drug use; they are not the result of it.

(SEE ALSO: *Causes of Substance Abuse: Psychological (Psychoanalytic) Perspective; Conduct Disorder and Drug Use; Families and Drug Use; Religion and Drug Use*)

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Race Despite reservations about the use of race and ethnicity in health research (e.g., Bhopal & Donaldson, 1998; LaVeist, 1994; Williams et al., 1994), this variable remains one of the most often reported socio-demographic characteristics in drug abuse/dependence studies.

Data from the Monitoring the Future Study (Johnston, O'Malley & Bachman, 1996) and the Youth Risk Behavior Survey (Centers for Disease Control, 1995) are consistent in showing that black adolescents are less likely to use most drugs than their white and Hispanic counterparts. The National Household Survey on Drug Abuse, which includes adult participants and adolescents who are not in school, shows that after the age of 25 years, African Americans report more illicit drug use than Whites (SAMHSA, 1999). In 1998, among persons 35 years and older, 4.8 percent of blacks versus 3.2 percent of whites had used an illicit drug in the past month, and 1.3 percent versus 0.3 percent had used cocaine, respectively. Blacks had lower rates of past month alcohol use, "binge" drinking, and heavy alcohol use than whites and Hispanics (SAMHSA, 1998).

Data from other large-scale surveys have been used to estimate drug use and dependence in different groups. The Epidemiologic Catchment Area (ECA) Study, a prospective study of drug dependence in the United States, show that black youth are less likely than white youth to initiate licit and illicit drug use (Helzer, Burnam & McEvoy, 1991). This is reflected in the rate of lifetime alcoholism among black males in the 18 to 29 age group when compared to whites, 12.7 percent versus 28.3 percent. With increase in age, rates for blacks exceed those of whites and Hispanics until at 65 and over, blacks are nearly twice as likely as whites to be alcohol dependent. The ECA data also show that young Hispanic men have about the same level of risk of developing alcoholism as Whites.

In a separate analysis of data from the ECA, Anthony & Helzer (1991) found that the rate of illicit drug use for Hispanic men was much lower that those for blacks and whites, with the lowest rate among Hispanic women. Overall, white men had the highest rate of illicit drug use compared to the other two groups, with the most prominent difference seen in the 18 to 29 age group. The lifetime prevalence of drug dependence followed the pattern of drug use in the three groups, but there were few differences in the rates for active dependence.

Another major source of estimates on racial/ethnic differences in drug use and dependence is the National Comorbidity Survey. Data from the NCS agree with estimates from the other household surveys. Blacks and Hispanic are less likely to use drugs than Whites but Blacks do not differ from Whites in the probability of becoming dependent on drugs. What distinguishes the groups is persistence in drug dependence once the problem has started (Kessler et al., 1995). Blacks are 3 times and Hispanics 2.4 times more likely to report past year dependence on drugs than their white counterparts. In other words, while African Americans are less likely to initiate drug use and equally likely to become dependent, they are more likely than Whites to remain dependent.

There is growing evidence that these racial/ethnic differences in drug use and drug dependence are not due to innate racial differences. For example, Crum and Anthony (2000) have shown that, when socio-economic factors (e.g., poverty and neighborhood characteristics) are taken into consideration, race/ethnicity becomes and insignificant influence. Other factors that may help account for observed racial/ethnic differences in the vulnerability to drug use and dependence are dropping out of school (Obot & Anthony, 2000), opportunity to use illegal drugs (SAMHSA, 1998), and perception of risks associated with drug use (Ma & Shive, 2000).

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Sensation Seeking Sensation seeking is a personality trait most recently defined by its originator, Zuckerman (1994), as "the seeking of varied, novel, complex, and intense sensations and experiences, and the willingness to take physical, social, legal, and financial risks for the sake of such experience." ALCOHOL and DRUG abuse and GAMBLING represent expressions of the needs involved in this trait, and over thirty years of research have shown that this trait is central to the initial attraction to drugs and the tendency to engage in social or abusive use of them. Among drug users, high sensation seekers are likely to use more kinds of drugs than moderate sensation seekers (varied experience), to use psychedelic drugs (novelty), and stimulants (intensity). However, they also use depressants like OPIATE drugs for the sake of the highs of the "rush" and the sensations of the subsequent depressant phase.

Drug users rate higher in sensation seeking than users of alcohol, only showing their willingness to take the extra risks associated with the use of illegal substances. Sensation seeking is involved in many other kinds of interests and activities related to alcohol and drug use including smoking, illicit or unsafe sex, disinhibited partying, reckless driving, and criminal activities.

Sensation seeking has been assessed most often using the Sensation Seeking Scale which contains four subscales: *Thrill and Adventure Seeking*, *Experience Seeking*, *Disinhibition*, and *Boredom Susceptibility*. The last three of these are most related to drug use. A total score is obtained by summing the four subscales. A newer scale is called Impulsive Sensation Seeking because it combines sensation-seeking items with those of a closely related trait, impulsiveness.

Many studies have shown that sensation seeking is related to current heavy alcohol use and illegal drug use among adolescents and young adults, and other studies (Bates et al., Cloninger et al., Teichman et al.) have demonstrated that sensation seeking at pre- or early adolescence predicts later alcohol and drug use during early adulthood. Lewis Donohew and his colleagues have designed communications for antidrug campaigns based on the sensation seeking traits of those at risk for use and abuse of drugs. The general tenor of these advertisements is that there are healthier ways to seek stimulation than through drugs. The style of the presentations as well as the content is aimed at high sensation seekers.

This writer's experience with treatment of drug abusers in a therapeutic community suggested that the trait is an important consideration in predicting outcome in combination with other traits and environmental considerations. Drug abusers who were also high sensation seekers had a special susceptibility to boredom. What can substitute for the kind of exciting lives they led as part of the drug scene? If they cannot obtain an interesting job, providing varied kinds of stimulation, or if they cannot find exciting friends like those still involved with drugs, they soon turn to drugs themselves. Therapists sometime assume that drugs were used to deal with ANXIETY and DEPRESSION, or as "self-medication." This only happens in a minority of cases. Early substance abuse is primarily driven by sensation seeking and impulsivity, not by neurotic needs. Anxiety and depression usually emerge as a reaction to drugs or their WITHDRAWAL and to the stresses of drug-life and quickly subside when the user is in effective treatment setting or abstinent after DETOXIFICATION. When bored and frustrated in attempts to find interesting work, or working at a

monotonous job, the high sensation seeker is most vulnerable to relapse.

(SEE ALSO: *Adolescents and Drug Use; Conduct Disorder and Drug Use; Prevention*)

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Sexual and Physical Abuse An increased recognition of the experience of physical and sexual abuse in the lives of many children and ADOLESCENTS has led to the increased interest in the impact of such abuse on drug use (Cavaiola & Schiff, 1989; Straus & Gelles, 1990; Dembo et al., 1988). In their 1985 survey of over 6,000 families in the United States, Straus and Gelles (1990) report that 23 per 1,000 children (2.3%) are seriously assaulted every year. Data from a 1991 telephone national survey of women indicate that about 20 per 100 (20%) of the sample reported one or more childhood sexual-abuse experiences (Wilsnack et al., 1994). Few research studies have focused specifically on the question of whether children who are physically and sexually abused are at increased risk of substance abuse. Dembo et al. (1988) suggest three reasons why child abuse has not been included in the conceptual schemes examining the process by which youths become involved in drug use. First, CHILD ABUSE has only recently (in the 1980s) surfaced as an issue receiving research and policy attention. Second, both child-abuse experiences and illicit drug use are often hidden phenomena, so that any covariation in their occurrence is difficult to observe. Third, the focus on social-psychological and socio-cultural factors left little opportunity for child-abuse variations to be considered. Throughout the 1980s and into the 1990s, there has been increasing recognition of the potential importance of abuse to the child's and adolescent's emotional development and the potential connection to substance use and other problem behaviors (Widom, 1991; Zingraff et al., 1993). The central hypothesis guiding research is that physically and sexually abused children and adolescents may use illicit drugs to help cope with the emotional difficulties caused by their negative self-perceptions or other internal difficulties that result from the abuse (Cavaiola & Schiff, 1989; Singer, Petchers, & Hussey, 1989; Dembo et al., 1988).

Much existing research has concentrated on cohorts of adolescents. The rationale for the vulnerability of childhood victims of abuse to drug dependence in adolescence includes first, the ramifications of abuse for lowering self-image and self-esteem, while increasing self-hatred. Based on Kaplan, Martin, and Robbins' (1984) proposition that self-derogation leads to drug use, this model suggests that the abuse of children is related to illicit drug use, both directly and as mediated by self-derogation (Dembo et al., 1988). Second, drugs may provide emotional or psychological escape and self-medication for young abuse victims; they may turn to drugs to chemically induce forgetting or to cope with feelings of ANXIETY (Miller, 1990). Third, drug use may provide abused children or adolescents with a peer group, in the form of a drug culture, hence reducing feelings of isola-

tion and loneliness (Singer, Petchers, & Hussey, 1989; Widom, 1991).

Methodological limitations have prevented the existing research from giving a definitive answer. According to Widom (1991), most studies of the association between illicit drug use and childhood victimization have focused on sexually or physically abused children in clinical or institutional settings, making it difficult to generalize to other populations; the studies are often cross-sectional in design, include only retrospective information about childhood-abuse experiences, and do not utilize control groups. Therefore, the validity and reliability of these data have been criticized. Since abuse-related consequences can vary across the life span, cross-sectional studies may miss important ramifications of abuse and it may be impossible to determine the developmental-causal sequence (Briere, 1992; Dembo et al., 1988). Furthermore, most of the studies do not control for other childhood characteristics that may mediate the effects of abuse. Studies focusing on the abuse victims as adults run further methodological risks. When asked about abuse from their childhood, these adults may forget, redefine events in terms of the present, or repress certain thoughts and events.

In one of the earliest reviews of the impact of sexual abuse in childhood, Browne and Finkelhor (1986) reported that adult WOMEN victimized as children were more likely to manifest DEPRESSION, self-destructive behavior, anxiety, feelings of isolation, poor self-esteem, and substance abuse than their nonvictimized counterparts. They distinguished initial effects—identified as the manifestations within two years of termination of abuse—from long-term effects.

In a carefully designed study, Widom (1992) followed two groups in arrest records for fifteen to twenty years. One group of 908 individuals with court-substantiated cases of childhood abuse or neglect was matched according to sex, age, race, and socioeconomic status with a comparison group of 667 children not officially recorded as abused or neglected. As indicated by arrest records, the behavior of those who had been abused or neglected was worse than those with no reported abuse—abused or neglected children were more likely to be arrested as juveniles, as adults, and for a violent CRIME. With regard to drug use, as adults, the abused and neglected females were more likely to be arrested for drug offenses compared to the nonabused females. In a large sample (N = 3018) of Alabama 8th and 10th graders, Nagy et al. (1994) found that about 10 percent (13% of females and 7% of males) of the students reported being sexually abused. Sexual abuse was defined to include one or more episodes of forced intercourse. Both sexually abused males and sexually abused females reported a higher use of illegal drugs in the past month than those students who did not report sexual abuse. While the associations were strong, the analyses did not attempt to control for confounding variables and were cross-sectional rather than longitudinal, so that causality cannot be inferred.

Wilsnack et al. (1994), using a national sample of adult women, examined the abuse of alcohol and drugs by women who reported retrospectively on whether they had been sexually abused as children. They found strong positive associations between being abused sexually as a child and six different measures of drinking behaviors and two summary drug-use measures. While these analyses are considered preliminary by the authors, because they do not attempt to control for confounding variables, the findings do suggest that early sexual trauma may be an important risk factor for substance abuse later in life.

In a retrospective study, Miller (1990) compared forty-five alcoholic women with forty women chosen randomly from the same community. The relationships between child abuse by the father and the development of alcoholism was examined by controlling on the parents' alcohol problems, family structure during childhood, income source, and age. Higher levels of negative verbal interaction and higher levels of moderate and serious violence were both predictive of those who were found in the alcoholic group.

In their review and synthesis of empirical studies regarding the impact of sexual abuse on children, Kendall-Tackett, Williams, and Finkelhor (1993) found that poor self-esteem was a frequently occurring consequence of sexual abuse. They also conclude that substance abuse, while being a common behavior for sexually abused adolescents, is not an inevitable outcome. In a residential treatment center, Cavaiola and Schiff compared with two control groups the self-esteem of 150 physically or sexually abused, chemically dependent adolescents. The results showed that abused chemically dependent adolescents had lower self-esteem than the two com-

parison groups; they found negligible difference between those who had been sexually abused and those who had been physically abused.

In two populations of youths studied in a juvenile detention center, Dembo et al. (1988, 1989) compared the lifetime drug use between detainees and a comparable age group in an adjacent county. The studies showed that the detainees' sexual victimization and their physical-abuse experiences related significantly to their lifetime use of illicit drugs. Sexual victimization had a direct effect on the frequency of lifetime drug use, whereas physical abuse had both a direct and an indirect effect on drug use, mediated by the adolescents' feelings of self-derogation. These findings were based on multiple-regression analyses that included family background, other risks for drug use, race, and sex.

CONCLUSION

Despite methodological issues, the body of available evidence suggests that involvement in substance use as an adolescent or adult is linked to an increased likelihood of having experienced physical or sexual abuse as a child. Owing to limitations in the retrospective, cross-sectional, and correlational designs of the research, causal linkages cannot be definitively attributed, and as Briere (1992) notes, while much of the existing research is flawed in its design, it has set the stage for the development of more tightly controlled and methodologically sophisticated studies that will be able to better disentangle the antecedents, correlates, and impacts of sexual and physical abuse.

Further research is needed to examine questions in which our knowledge is meager. First, are there different effects from physical abuse, sexual abuse, or neglect on substance use or dependence? Do other psychosocial factors lead to substance abuse? Second, does the perpetrator of the abuse matter for the impact? Third, does continuity or duration of the abuse matter? Fourth, and perhaps most important, what are the links between suffering maltreatment as a child and later alcohol or drug problems?

(SEE ALSO: *Families and Drug Use; Family Violence and Substance Abuse*)

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Stress The term "stress" is frequently defined as a process involving perception, interpretation, response and adaptation to harmful, threatening, or challenging events (Lazarus & Folkman, 1984). This kind of conceptualization allows the separate consideration of (1) the events that cause stress (stressors or stressful life events), (2) the cognitive processes that evaluate stress and the availability of resources to cope with the stressor (appraisal), (3) the biological arousal and adaptation associated with the stressor, and (4) behavioral and cognitive response to the stressful event (actual coping). While different models of stress put more or less emphasis on appraisal mechanisms or biological adaptation mechanisms, the concept of an organism responding to substantial threat or danger is basic to most theories of stress (e.g., Cohen et al., 1986; Mason, 1975; Selye, 1976; Hennessy & Levine, 1979).

Stress produces a negative emotional state associated with perception and appraisal of the stressor, its situational and psychological characteristics, and the assessment of resources available for coping. Stress also activates a biological response with sympathetic arousal, activation of the pituitary-adrenocortical axis, and endogenous opioid-peptide release to alert the body to the stressed state and to support adaptation to the situation. Researchers have found two aspects of stressful events that appear to mediate cognitive appraisal and the biological stress response. These are the controllability and predictability aspects of the event. The extent to which an event is predictable (i.e., the individual is aware of an upcoming stressful event and can prepare for it) and controllable (i.e., the individual perceives the situation as one that he/she can control and adapt to) is significantly associated with the magnitude of the biological stress response and the negative emotional state associated with the event (Frankenhauser, 1980; Hennessey & Levine, 1979). Thus, greater the unpredictability and uncontrollability, greater the emotional distress and the biological response associated with the event.

The aversive quality of stressful situations motivate individuals to reduce the stress by using a variety of coping strategies. Lazarus (1966) identified two primary classes of coping: (1) direct action, which is usually behavioral and involves activity aimed at altering the source of stress or one's relationship to it, and (2) palliation, focused on managing one's emotional responses rather than causes of stress. Palliative coping may be behavioral or cognitive; it may include denial, withdrawal, taking drugs, and/or other forms of making oneself feel better (or less bad). Direct action is a manipulative response aimed at changing a stressor, while palliation is generally accommodative. Similar to the above categories are the two types of coping identified by Lazarus & Folkman (1984). These are 'problem-focussed' coping aimed at doing something to alter the source of the stress, and 'emotion-focussed' coping aimed at managing the emotional distress associated with the stressful event. How people cope with stressful events is key to their success in reducing the associated distress and producing an effective adaptive response to similar stressful situations in the future.

STRESS AND INCREASED VULNERABILITY TO DRUG USE

Most major theoretical models of addiction conceptualize stress as an important factor in the motivation to use addictive substances. For example, the Stress-Coping model of addiction proposes that use of addictive substances serve to both reduce negative affect and increase positive affect, thereby reinforcing drug taking as an effective, albeit maladaptive, coping strategy (Wills & Shiffman, 1985). Marlatt's Relapse Prevention model (Marlatt & Gordon, 1985) has proposed that in

addition to other bio-psychosocial risk factors such as parental substance use, peer pressure, and positive expectancies regarding the potential benefits of using substances, individuals who have poor ways of coping with stressful events are at increased risk for problematic use of addictive substances. Finally, the Tension Reduction Hypothesis (Conger, 1956; Sher & Levenson 1982) and the Self-Medication Hypothesis (Khantzian, 1985) have been proposed stating that people use drugs to enhance mood and alleviate emotional distress. The latter hypotheses propose that the motivation to enhance mood may be high in the face of both acute and chronic distress states. A drug may be used initially to modulate tension or distress; then with repeated success in doing so, it may become a more ubiquitous response to stress or because of the positive expectancies from drug effects, people may come to use drugs in anticipation of both the relief and mood enhancement.

Prospective studies, which measure stressful events and subjective perception of stress as they occur and use them to predict future drug use, have been conducted to examine whether stress increases the vulnerability to drug use. Higher levels of stress and maladaptive coping along with low parental support predict escalation of drug use in adolescents (Wills et al., 1996). Evidence from animal studies further suggest that stressful experiences in early childhood may increase the vulnerability to drug use. Higley and colleagues (1991) studied rhesus monkeys who were reared by mothers (normal condition) or by peers (stressed condition) for the first six months of their life. Peer-reared monkeys consumed significantly more amounts of alcohol than mother-reared adult monkeys. Furthermore, when stress was increased in the adult monkeys via social separation, mother-reared monkeys increased their levels of alcohol consumption to that of peer reared monkeys. Others have found that rats who show greater reactivity to stress and novelty show an increased vulnerability to self-administration of psycho-stimulants such as amphetamines (Piazza et al., 1989; Piazza & LeMoal, 1996). These findings suggest that individual responses to stressful events and previous experience of stressful events may increase the vulnerability to use addictive substances.

Several studies have shown that acute stress increases self-administration of drugs. Acute behavioral stress in laboratory animals leads to increased drinking and drug use in the post-stress period (Nash & Maickel, 1988; Piazza & LeMoal, 1996; Shaham & Stewart, 1994; Goeders & Guerin, 1994; Miczek & Mutschler, 1996). Human laboratory studies demonstrated increased use of addictive substances after stress as opposed to non-stress situations (see Marlatt & Gordon, 1985 for review). Laboratory induction of stress has also been shown to increase craving for addictive substances in addicts (Sinha et al., 1999a; 1999b). In support of the tension reduction hypothesis, some evidence has accumulated to suggest that alcohol dampens the biological stress response in social drinkers (Sher & Levenson, 1982; Finn & Pihl, 1991; Levenson et al., 1987; Sinha et al., 1998), but this effect appears mediated by a family history of alcoholism and other individual difference variables.

Converging lines of evidence cited above support the key role of stress in mediating problem use of addictive substances. Findings suggest that stressful experiences significantly impact the vulnerability to increase substance use. In addition, in individuals using substances regularly, stressful experiences may lead to an escalation of drug use to the point that such use can lead to drug-related problems for the individual. Despite the above evidence, the specific ways in which stress increases drug intake are not well understood. Animal studies suggest that stress alters brain reward pathways such that drugs are likely to feel more reinforcing than in non-stress conditions (Koob & LeMoal, 1997). Whether these alterations can be detected in humans and modified to reduce the negative impact of stress on drug use remains to be established in future research.

CHRONIC DRUG USE AND VULNERABILITY TO STRESS

The question of whether addicts are more sensitive to the effects of stress on drug intake has received recent attention. It is now well known that the most commonly used addictive substances such as alcohol, nicotine, psychostimulants such as amphetamines and cocaine, opiates and marijuana which stimulate the brain reward pathways, also activate brain stress systems by stimulating release of corticotrophin-releasing factor (CRF) which in turn activates the hypothalamic pituitary adrenal (HPA) axis and release of catecholamines (Robinson & Berridge, 1993). With the chronic use of

addictive substances, hallmark symptoms of dependence emerge, namely, tolerance and withdrawal, that are associated with changes in the CRF-HPA, dopaminergic and catecholaminergic systems (Robinson & Berridge, 1993; Koob & LeMoal, 1997). Whether this excessive substance use leads to significant 'wear' and 'tear' on the brain systems that it activates, such that these systems may be unable to function normally in addicts is being examined. Stewart and colleagues have shown that in laboratory animals with a history of drug taking, stress results in reinstatement of drug use when the animals are drug free. However, animals experienced in self-administering food, sucrose pellets or sucrose solution, do not show a stress-related increase in these behaviors. Such data has led to the suggestion that it is a history of drug taking that appears to increase vulnerability to stressful events (Stewart, 2000).

Finally, some human studies support the hypothesis that chronic drug use may alter stress and coping. Evidence suggests that baseline responsivity of the CRF-HPA system is altered during acute and protracted withdrawal in alcoholics and cocaine and opiate addicts (Kreek & Koob, 1998). This co-occurs with behavioral symptoms such as increases in irritability, anxiety, emotional distress, sleep problems, dysphoria and restlessness that are common during acute and protracted phases of withdrawal from alcohol, cocaine, opiates, nicotine and marijuana (Diagnostic and Statistical Manual-IV, 1994; Hughes, 1992). Furthermore, high levels of stress are reported in smokers who are unable to quit, while those who abstain show lower levels of stress (Cohen & Lichtenstein, 1990). However, there is also evidence that stressful life events are not associated with subsequent drug use and relapse in addicts after treatment (Hall et al., 1990; 1991). Future research on the psychobiological effects of chronic drug use as they pertain to the addicts' ability to respond to stress and cope with abstaining from drug use, would be relevant in understanding the nature of this association.

SUMMARY

This section outlines the key aspects of stress and coping and how they relate to addictive behavior. Facing stress is basic to all organisms, but how we cope with stress can differ significantly across individuals. The above section outlines two possible ways in which stress has been associated with addictive behavior. The first aspect targets vulnerability to stress and use of addictive substances as a way of coping with stress. The second aspect of the association has only recently received attention, namely, the effect of chronic drug use on stress and coping. Although the above outline presents key evidence to support the important association between stress and addictive processes, the field continues to develop in order to further our understanding on the psychobiological mechanisms that link stress and coping to addictive behaviors.

(SEE ALSO: *Addiction: Concepts and Definitions; Co-morbidity and Vulnerability; Complications; Endorphins; Epidemiology of Drug Abuse; Families and Drug Use; Family Violence and Substance Abuse; Poverty and Drug Use*)

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W-X

WALDEN HOUSE *See* Treatment Programs/Centers/Organizations: An Historical Perspective

WAR ON DRUGS *See* Epidemics of Drug Abuse; Treatment, History of; U.S. Government; Zero Tolerance

WASHINGTONIAN TEMPERANCE SOCIETY/WASHINGTONIANS *See* Temperance Movement; Treatment, History of; Women's Christian Temperance Movement

WELFARE POLICY AND SUBSTANCE ABUSE IN THE UNITED STATES Generally speaking, the American income maintenance system is divided into two "tracks" based on the relationship of beneficiaries to the labor force. For the so-called "insurance-like" programs, notably Old Age and Survivors Insurance (what Americans refer to colloquially as "Social Security"), Social Security Disability Insurance, and Unemployment Compensation, eligibility is linked to an applicant's history of payroll deductions—contributions from wages to the public fund that supports the program. The so-called "welfare" programs, on the other hand, are "means-tested." That is, eligibility hinges on meeting strict limits on current earnings and accumulated wealth. Welfare programs are for very poor people and their benefits are substantially inferior to those paid by the insurance-like programs.

As well, the American income maintenance system is "categorical." For the most part, eligibility is based on membership in a particular category defined by administrative rules: Old age benefits are for those who meet the administrative definition of aged status; disability benefits are for those who meet the medical and vocational standards defining that category, and so forth. Except as discussed below in connection with General Assistance, there are no welfare programs for hale, nonelderly adults without children.

Finally, the income maintenance system in the United States is funded and administered by federal, state, and local (primarily county) governments. Insurance-like programs are usually funded and administered by the federal government, thus creating a significant degree of uniformity in benefits and eligibility rules. Welfare programs, however, usually are funded and administered by two or more levels of government, and benefit levels and eligibility rules vary considerably among political jurisdictions.

This article concerns the intersection of substance abuse and initial and continuing eligibility for welfare programs in the context of policy changes made during the 1990s. It focuses mainly on Temporary Assistance for Needy Families (TANF) and, to a lesser extent, General Assistance

(GA). Supplemental Security Income (SSI), a federally funded and administered welfare program for the elderly, blind, and disabled, is the subject of a serparate entry concerned with addiction as a disabling impairment in the disability programs administered by the Social Security Administration (see ELIMINATION OF DRUG ADDICTION AND ALCOHOLISM AS QUALIFYING IMPAIRMENTS IN SOCIAL SECURITY DISABILITY PROGRAMS).

Temporary Assistance for Needy Families. For 60 years after the enactment of the Social Security Act of 1935, America's cash assistance program for impoverished families was Aid to Families with Dependent Children (AFDC; Aid to Dependent Children until 1961, when a parental or caretaker grant was added). As the result of liberal court rulings in the 1960s and the separation of casework from the financial administration of recipients' grants in 1972, AFDC became substantially free of the punishing moralism that characterized an earlier era when social workers raided the houses of welfare mothers to search closets for evidence of a "man in the house" who might be made to support the women and their children. Although various work incentives were tried over the years, particularly during the 1980s, they had indifferent results and affected relatively few recipients. Even so, only a small percentage of AFDC families remained on the rolls for years at a time, and most AFDC heads of household, the great majority of them women between 18 and 35 years old, worked part-time or intermittently while raising their children.

However, the ascendancy of the Republican Party following the November 1994 elections yielded the Personal Responsibility and Work Opportunity Reconciliation Act (PRWORA) of 1996 (P.L. 104–193). The PRWORA was based on premises laid out succinctly in *Contract with America*, the 1994 campaign manifesto drafted by Republican leaders in the House of Representatives. *Contract* opined that the liberal welfare regime dating from the 1960s "had the unintended consequence of making welfare more attractive than work" (p. 67). Moreover: "Government programs designed to give a helping hand to the neediest of Americans have instead bred illegitimacy, crime, illiteracy, and more poverty." Welfare reform should thus "change this destructive social behavior by requiring welfare recipients to take personal responsibility for decisions they make" (p. 65).

The PRWORA's countermeasures are a complicated combination of incentives and punishments directed at both welfare recipients and the states. The act creates a lifetime limit of 5 years's welfare receipt for TANF families. Further, its funding mechanism requires that each year the states move progressively greater numbers of TANF parents into jobs or face cuts in the overall federal grant to the state (known as a "block grant"). Each state may exempt 20 percent of its caseload from job placement, but in the long run the states are faced with the formidable task of making work-ready and placing in employment thousands of mothers with little work experience and few marketable skills. At the same time, the PRWORA permits the states a great deal of flexibility in using various funds to create training programs, support childcare, and even fund alcohol and drug treatment.

The PRWORA also requires or permits the states to enforce a variety of "behavioral requirements" for continuing eligibility for full TANF benefits. Among these is the PRWORA's permitting states to mandate treatment for alcohol and drug abusers as well as to require random drug testing under the threat of forfeited benefits. (A failed provision of the original legislation would have forced the states to implement these provisions.) However, recent research on TANF parents in some states has produced the startling (to some) finding that the prevalence of substance-abuse disorders in the adult TANF population, *as measured by a rigorous standard*, is very similar to that in the population at large: about 8 to 10 percent. To date, only Louisiana, Michigan, Nevada, and New York have expressed serious interest in drug testing and any implementation plan will face a court test. However, a number of states, including California, are exploring mechanisms for mandatory treatment and the use of "representative payees," a third party who receives and manages a recipient's benefits.

A further drug-related provision of the PRWORA is both more stringent and more common. The act provides that unless a state passes contrary legislation, any person with a felony drug conviction for conduct after August 22, 1996 (the date PRWORA was signed into law), will be banned for life from TANF benefits. This provision, it should be noted, reflects a negotiated compromise on the House of Representatives version of the act

that would have extended the ban to those convicted of misdemeanors. At this writing, nine states (Connecticut, Kentucky, Michigan, New Hampshire, New York, Ohio, Oklahoma, Oregon, and Vermont) have passed the legislation required to opt out of the ban. Eighteen other states have passed legislation to soften it.

In 1996, about sixty-one thousand women were convicted of drug felonies in the United States. A 1997 Legal Action Center survey of seventeen drug and alcohol treatment programs for women with children located in different parts of the country found that 21 percent of the welfare mothers in those programs had felony drug convictions. In the only study to date relevant to the TANF ban's likely effect on single mothers, attorney Amy E. Hirsch interviewed twenty-six affected women in Pennsylvania, a state that has not modified the ban. Most were convicted of possessing small amounts of drugs valued from four to one hundred dollars. Before entering treatment (where Hirsch found them), all had been heavy users, typically of crack cocaine, and most had been charged with possession with intent to deliver. In fact, they were for the most part intermediaries and small-time corner girls bagging and transporting crack and engaging in sex work to subsidized their habits. Often, they were allowed (if not encouraged) to plead guilty to a felony because only by court stipulation could they receive a residential treatment bed.

Two-parent families may also qualify for TANF and the drug-felony ban may have an important and negative cumulative impact on them, perhaps by discouraging drug-felon fathers from living with their families so as not to jeopardize the TANF benefits of the mother and children, At this writing, there are no data on this subject.

General Assistance. General Assistance (known in some places as General Relief) is a form of welfare financed and operated entirely by state, county, or municipal governments. Many states do not have GA programs, or GA exists only in some local jurisdictions. GA benefit levels and eligibility rules also vary from state to state, and in some states, notably California and Wisconsin, from county to county. Some states (or smaller jurisdictions) provide GA benefits merely on the basis of need, but most GA programs are categorical (e.g., Oregon and Washington), restricting eligibility to older people not yet eligible for Social Security or Supplemental Security Income (SSI); to parents waiting for TANF benefits or temporarily suspended from that program; to those with an SSI application pending; or to those who are realistically unemployable by some criteria of age and infirmity but who do not meet the stringent disability criteria of SSI. GA programs also vary in the way that benefits are paid: by cash, by rent and food vouchers, or some combination. Some GA programs are time-limited (in Pennsylvania, e.g.). All GA programs have extremely low benefits, however. In California, the most generous GA allowance is in the City and County of San Francisco, where it is about $330 per month—this in a city where the monthly fair market rent for a studio apartment now exceeds $800.

Probably because of the over representation of single men among GA beneficiaries, many jurisdictions estimate that the prevalence rate of alcohol and drug problems among GA recipients is several times that of the general population. Historically, GA has been the welfare program most accessible to people with alcohol and drug problems. During the heyday of the post-war skid row (see HOMELESSNESS, ALCOHOL, AND OTHER DRUGS), many large cities used some combination of cash, hotel vouchers, and restaurant chits to keep single addicted men (mainly) roughly housed and fed without giving them much money to handle. This system was largely abandoned as the cost of its administration rose. However, with the elimination of addiction as a qualifying impairment in the SSI program, some cities and counties are considering the revival of such arrangements, perhaps to be administered by community-based nonprofits and combined with mandatory treatment and representative payee provisions. Other may adopt Pennsylvania's approach. There, since 1981, diagnosed abusers of alcohol and/or other drugs may receive GA for 9 continuous months on this basis once in a lifetime so long as they are in treatment.

CONCLUSION

The thrust of recent federal welfare reform has been to rely on fiscal incentives and penalties to encourage welfare recipients to work and state governments to see that they do. As a corollary, welfare eligibility is once again being used as leverage on the behavior of poor people and drinking and drug use have been salient targets of this effort—whose complete effects remain to be seen. Given the re-

sources (no small caveat), many state and local General Assistance programs seem inclined to follow suit.

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WERNICKE'S SYNDROME *See* Alcoholism; Complications: Neurological

WIKLER'S PHARMACOLOGIC THEORY OF DRUG ADDICTION Abraham Wikler (died 1981) was one of the first researchers who, in the late 1940s, strongly advocated the idea that drug abuse and relapse following treatment are influenced by basic learning processes. Early in his career, Wikler became interested in reports from relapsed heroin addicts that despite being free of withdrawal symptoms during treatment and upon discharge, they experienced withdrawal symptoms and craving when they returned to their drug-use environments—and that these feelings were responsible for their return to drug use.

Based on these and other anecdotes, Wikler—who was familiar with the recent work of Russian physiologist Ivan Petrovich Parlor (1849–1936) on conditioning—proposed that events which reliably signal drug self-administration or drug withdrawal elicit conditioned responses (CRs) that take the form of withdrawal and drug craving. According to Wikler, these CRs motivate further drug use, which, by terminating negative withdrawal feelings, perpetuates the cycle of drug dependency.

At the heart of Wikler's model lies the notion that classical conditioning mechanisms are activated when events surrounding drug use *reliably* begin to signal upcoming drug administration. These events may be external cues (e.g., the sight of a syringe) or internal states (e.g., depression) that *consistently* precede drug use. In nondependent users (who take drugs infrequently), Wikler proposed that the unconditioned response (UR) elicited by the drug consists of direct effects of that drug on the nervous sytem. In such individuals, stimuli that signal drug use would then come to evoke druglike responses; however, a different set of CRs are thought to occur in long-term drug users who have become physically dependent on the drug. These individuals experience withdrawal symptoms as the drug effect wanes and consequently, stimuli associated with drug withdrawal in these individuals evoke withdrawal reactions.

The aversive symptoms produced by withdrawal in dependent users provide motivation to self-administer the drug. Through a process of operant conditioning, drug taking is rewarded by the termination of the negative withdrawal symptoms. These reward experiences further strengthen the tendency of the drug user to turn to drug use when experiencing withdrawal symptoms. Likewise, stimuli paired temporally with withdrawal may also acquire the ability to elicit drug taking. Because Wikler invoked both classical and operant conditioning mechanisms as contributors to drug use, his model has often been characterized as a two-process model of drug use.

Wikler's model also provides for a powerful account of relapse following treatment for drug use. Because some treatment programs separate the abuser from the drug-use environment, the patient never learns to deal with drug-related events. Upon returning home following treatment, even though no longer physically dependent, the patient encounters drug signals, experiences conditioned withdrawal reactions, and eventually turns to drug use to reduce the negative feelings. Since conditioned responses show little spontaneous decay over time, the drug-use patient is at risk even following an extended treatment program. According to Wikler, treatment programs need to address condi-

tioned responses directly. One suggested approach involves having subjects go through their usual drug-preparation ritual in a protected setting, where drugs are not available. Such exposures should serve to extinguish drug-use responses by failing to reinforce them with relief from withdrawal. Extinction training as well as other techniques for reducing the role of conditioned responses in relapse are currently being explored.

(SEE ALSO: *Behavioral Tolerance; Causes of Substance Absue: Learning; Naltrexone; Research, Animal Model: Learning, Conditioning and Drug Effects*)

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WINE *See* Alcohol; Fermentation

WITHDRAWAL This section contains the articles on withdrawal syndromes, each of which describes and discusses withdrawal signs, symptoms, and treatment. The following substances are covered: *Alcohol; Benzodiazepines; Cocaine; Nicotine (Tobacco)*; and *Nonabused Drugs*. For descriptions and discussions of withdrawal from Amphetamines, see *Amphetamine*; Anabolic Steroids, see *Anabolic Steroids*; Barbiturates, see *Barbiturates*; Caffeine, see *Caffeine*; Cannabis, see *Cannabis*, see also *Marijuana*; for Heroin, Opiates/Opioids, see *Opioid Complications and Withdrawal*. For additional information, see also *Treatment*.

Alcohol The nervous system undergoes adaptation in response to the chronic consumption of alcohol (ethanol). If consumption is heavy enough (adequate dose) and occurs for a long enough time period (duration), a withdrawal syndrome will ensue following a rapid decrease or sudden cessation of drinking. This occurs in association with readaptation of the nervous system to a drug-free state. The dose and duration of alcohol consumption required to produce a withdrawal syndrome in a given population or even a given individual are difficult to predict, since no well-controlled studies have been conducted (or are likely to be, for ethical reasons). Such studies have been done in animals. The goals of treatment are to relieve discomfort and to prevent complications.

In the nondrinker or social drinker who consumes alcohol to the point of legal intoxication, an acute withdrawal syndrome may ensue ("hangover"). Symptoms occur in inverse relation to the fall in BLOOD ALCOHOL CONCENTRATION (BAC). These consist of insomnia, headache, and nausea. Usually no treatment is required and there are no serious consequences of this acute withdrawal. The withdrawal syndrome following chronic long-term alcohol consumption (usually months to years), however, is a more serious disorder.

The natural history of alcohol dependence to the point of requesting or clearly requiring detoxification services is usually fifteen to twenty years. The average age of persons admitted to detoxification units is around 42 years. (That is not to say that persons as young as 20 or as old as 80 do not require detoxification services.) The withdrawal syndrome seen in persons requiring detoxification ranges from a mild degree of discomfort to a potentially life-threatening disorder.

The severity of the withdrawal syndrome is dependent on both the dose and duration of alcohol exposure. This is clearly demonstrated in animal studies (rats) where a severe withdrawal syndrome can be demonstrated following high-level exposure to alcohol in a vapor chamber in as short a time period as a week. Administration of alcohol into the stomach is associated with a longer time period for acquisition of physical dependence. In humans also, the severity of withdrawal depends on the amount of alcohol consumed and the time period during which it has been consumed. For practical purposes this means the amount taken on a daily basis for the weeks and months preceding detoxifi-

Addiction Research Foundation Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar)

Patient _____ **Date** | ___ | ___ | ___ | (y m d) **Time** ___ : ___ **(24-hour clock, midnight = 00:00)**

Pulse or heart rate, taken for one minute: _____ **Blood pressure:** _____/_____

NAUSEA AND VOMITING—Ask "Do you feel sick to your stomach? Have you vomited?" Observation.
0 no nausea and no vomiting
1 mild nausea with no vomiting
2
3
4 intermittent nausea with dry heaves
5
6
7 constant nausea, frequent dry heaves and vomiting

TREMOR—Arms extended and fingers spread apart. Observation.
0 no tremor
1 not visible, but can be felt fingertip to fingertip
2
3
4 moderate, with patient's arms extended
5
6
7 severe, even with arms not extended

PAROXYSMAL SWEATS—Observation.
0 no sweat visible
1 barely perceptible sweating, palms moist
2
3
4 beads of sweat obvious on forehead
5
6
7 drenching sweats

TACTILE DISTURBANCES—Ask "Have you any itching, pins and needles sensations, any burning, any numbness or do you feel bugs crawling on or under your skin?" Observation.
0 none
1 very mild itching, pins and needles, burning or numbness
2 mild itching, pins and needles, burning or numbness
3 moderate itching, pins and needles, burning or numbness
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

AUDITORY DISTURBANCES—Ask "Are you more aware of sounds around you? Are they harsh? Do they frighten you? Are you hearing anything that is disturbing to you? Are you hearing things you know are not there?" Observation.
0 not present
1 very mild harshness or ability to frighten
2 mild harshness or ability to frighten
3 moderate harshness or ability to frighten
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

cation. One study of inpatients (who were federal prisoners and narcotic users) demonstrated that the consumption of 442 grams of alcohol or 32 standard drinks (a standard drink being 13.6 gm of alcohol—12 oz. of beer, 5 oz. of wine, or 1.5 oz. of liquor) per day for about two months results in a major withdrawal syndrome in all subjects, whereas the consumption of 280 to 377 grams (21 to 28 standard drinks) per day results in a mild syndrome of anxiety and tremor (Isbell et al., 1955). Other studies that involve patients (as opposed to research subjects) have not been able to demonstrate a consistent relationship between recent alcohol consumption and severity of the withdrawal syndrome (Shaw et al., 1981). This in part relates to the lack of accurate recall of exact quantities consumed within a given time period. Furthermore, in the real world there are different patterns of consumption (e.g., some drinkers consume alcohol in a binge pattern, whereas others drink in a more regular pattern), and different drinkers have varying durations of lifetime exposure to alcohol. One drinker may take two or three years to become dependent, another fifteen years, and yet another forty years. In addition, a person who has previously experienced significant alcohol withdrawal may be at higher risk for developing repeat withdrawal, both in terms of the severity of the

ANXIETY—Ask "Do you feel nervous?" Observation.
0 no anxiety, at ease
1 mildly anxious
2
3
4 moderately anxious, or guarded, so anxiety is inferred
5
6
7 equivalent to acute panic states as seen in severe delirium or acute schizophrenic reactions

AGITATION—Observation.
0 normal activity
1 sommewhat more than normal activity
3
4 moderately fidgety and restless
5
6
7 paces back and forth during most of the interview, or constantly thrashes about

VISUAL DISTURBANCES—Ask "Does the light appear to be too bright? Is its color different? Does it hurt your eyes? Are you seeing anything that is disturbing to you? Are you seeing things you know are not there? Observation.
0 not present
1 very mild sensitivity
2 mild sensitivity
3 moderate sensitivity
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

HEADACHE, FULLNESS IN HEAD—Ask "Does your head feel different? Does it feel like there is a band around your head?" Do not rate for dizziness or lightheadedness. Otherwise, rate severity.
0 not present
1 very mild
2 mild
3 moderate
4 moderately severe
5 severe
6 very severe
7 extremely severe

ORIENTATION AND CLOUDING OF SENSORIUM—Ask "What day is this? Where are you? Who am I?"
0 oriented and can do serial additions
1 cannot do serial additions or is uncertain about date
2 disoriented for date by no more than 2 calendar days
3 disoriented for date by more than 2 calendar days
4 disoriented for place and/or person

Total CIWA-A Score _____
Rater's Initials _____
Maximum Possible Score 67
This scale is not copyrighted and may be used freely.

syndrome and the rate of reacquisition of physical dependence (since it takes a shorter time to become re-addicted). This more rapid reacquisition has been attributed to sensitization (or "kindling") of the central nervous system (Linnoila et al., 1987). Other factors that may be implicated in the severity of the withdrawal syndrome include age, nutritional status, and presence of concurrent physical disorders or illness (e.g., pancreatitis or pneumonia) (Sullivan & Sellers, 1986). Alcoholics are at increased risk for these and other medical disorders.

The symptoms and signs of alcohol withdrawal appear in inverse relation to the elimination of alcohol from the body. Many alcoholics note this phenomenon on a daily basis—they require a drink in the morning to "steady the nerves," to suppress tremor and anxiety. The following are some of the more common symptoms of alcohol withdrawal: anxiety, agitation, restlessness, insomnia, feeling shaky inside, anorexia (loss of appetite), nausea, changes in sensory perception (tactile: skin itchy; auditory: sounds louder; visual: light brighter), headache, and palpitations. Common signs include vomiting, sweating, increase in heart rate, increase in blood pressure, tremor (shakiness of hands and sometimes face, eyelids, and tongue), and seizures. More severe withdrawal is associated with intensification of the above symptoms and signs together with progression to hallucinations (tactile: feeling things that are not there; auditory: hearing things that are not there; visual: seeing things that are not

there), disorientation, and confusion (DELIRIUM TREMENS, DTs). After stopping alcohol, the more common and milder symptoms usually peak at 12 to 24 hours and have mostly subsided by 48 hours (Sellers & Kalant, 1976). More severe or late withdrawal usually peaks later, 72 to 96 hours, and is potentially life threatening. Less than 5 percent of persons withdrawing from alcohol (depending on how they are selected) are estimated to develop a severe reaction. With appropriate drug treatment, an even lower percentage are estimated to develop a major withdrawal reaction. Under ideal circumstances there should be almost no mortality from this disorder on its own, so overall mortality ought to be similar to that of any concurrent medical disorder.

Assessment of the severity of withdrawal can be accomplished on the basis of clinical experience or with the assistance of various rating instruments. One of the simplest and easiest to administer is the Clinical Institute Withdrawal Assessment for Alcohol-revised (CIWA-Ar). This consists of ten items that can be scored at frequent intervals (Sullivan et al., 1989). The health-care provider can administer this instrument in less than a minute (see Figure 1).

TREATMENT

Treatment for the alcohol withdrawal syndrome consists of supportive care, general drug treatment, and specific drug treatments. *Supportive care* consists of reassurance, reality orientation, reduced sensory stimuli (dark, quiet room), attention to fluids, nutrition, physical comforts, body temperature, sleep, rest and positive encouragement toward long-term rehabilitation. The majority of patients can be treated with supportive care alone; however, it is impossible to be able to predict which patients will or will not require more intensive care. *General drug treatment* includes the B vitamin thiamine, which should be given to all patients. This is given to prevent the brain damage that occurs commonly in alcoholics who are thiamine deficient. Occasionally magnesium may be given if there is a severe deficiency and there are potential cardiac problems. Intravenous fluids may be required in uncommon circumstances.

Specific drug treatments may also be given to suppress the signs and symptoms of withdrawal. While over a hundred drug treatments have been suggested as useful in the treatment of alcohol withdrawal, very few adequate scientific studies have been conducted—the main reasons being that appropriate studies are difficult to conduct and that many patients do very well with placebo and/or supportive care alone. Nevertheless, appropriate and effective specific treatments are available and consist of drugs belonging to the same general class as alcohol (central nervous system depressants). The drugs of choice are the longer-acting benzodiazepines (usually diazepam [Valium], but others include chlordiazepoxide [Librium], lorazepam [Ativan], and oxazepam [Serax]), or occasionally a long-acting barbiturate like phenobarbital. The specific drug treatment is usually given either before most withdrawal has occurred (substitution or prophylactic treatment) or after significant symptoms and signs manifest themselves (suppressive treatment). The advantages of substitution treatment include the prevention of potential discomfort and the possible prevention of more severe withdrawal. The disadvantages include an unnecessary treatment for some patients. The advantages of suppression treatment include more appropriate titration of dose of medication, according to a given patient's needs. The disadvantages include unnecessary patient discomfort, at least initially, possibly the development of more severe withdrawal, and sometimes drug-seeking behavior from patients and unnecessary drug withholding from staff.

BENZODIAZEPINES have been well demonstrated to prevent complications (Sellers et al., 1983) of serious withdrawal, such as seizures, HALLUCINATIONS, and cardiac arrhythmias. In general, high doses of these benzodiazepines (with medium to long half-lives) are provided early in treatment, to cover the patient for the time period of acute withdrawal (usually 24 to 48 hours). Some patients require very large doses of drug (e.g., several hundred milligrams of diazepam) to suppress symptoms and signs. Patients with histories of withdrawal seizures (convulsions) or those that have epilepsy are always treated prophylactically, usually with benzodiazepines and any other anticonvulsant drug (medication) that they are prescribed on a regular basis. Patients who develop hallucinations are given (in addition to benzodiazepines) a phenothiazine (neuroleptic or antipsychotic drug). Typical drugs from this class include haloperidol (Haldol), and chlorpromazine (Thorazine). These drugs are effective in the treatment of hallucinations.

SUMMARY

In summary, alcohol withdrawal syndrome is a constellation of symptoms and signs that accompany the detoxification and readaptation of the nervous system to a drug-free state in chronic users. In most cases, these signs and symptoms are a source of mild discomfort and run a self-limited course. Occasionally, more severe withdrawal occurs or patients have concurrent complications (e.g., seizures). Under these circumstances appropriate drug treatment is mandatory to relieve symptoms and prevent complications.

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Benzodiazepines Like many other drugs that alter central nervous system (CNS) NEUROTRANSMISSION, benzodiazepines may produce a withdrawal syndrome when the drugs are abruptly discontinued. These withdrawal symptoms, including increased ANXIETY and insomnia, are often the mirror image of the therapeutic effects of the drug. Since the term *withdrawal* is usually applied to drugs of abuse, these symptoms are sometimes called abstinence syndrome or discontinuance syndrome when associated with benzodiazepines, thereby distinguishing these substances from drugs such as ALCOHOL, OPIOIDS, COCAINE, and BARBITURATES.

ETIOLOGY

Not all patients who take benzodiazepines will experience a discontinuance syndrome when the drug is stopped. Several conditions must be present before the discontinuance syndrome is likely:

1. *Duration of treatment*. The benzodiazepine must be taken long enough to produce alterations in the CNS that will predispose to a discontinuance syndrome. When benzodiazepines are taken at therapeutic doses, the range of time that usually produces a discontinuance syndrome is from several weeks to several months. Taking benzodiazepines once or twice during a crisis, or even for several weeks during a prolonged period of stress, ordinarily does not set the stage for discontinuance symptoms.
2. *Dose*. The amount of drug taken on a daily or nightly basis is also a critical factor. When higher-than-therapeutic doses are taken—for example, for treatment of panic disorder—then the period required before a discontinuance syndrome may develop is shortened.
3. *Abrupt discontinuance of the benzodiazepine*. Discontinuance symptoms arise because the level of drug at the CNS receptor sites is suddenly diminished. Since drug level in the CNS is proportional to the amount circulating throughout the body, an abrupt decline in CNS drug levels occurs when the blood level abruptly drops. Gradual tapering of benzodiazepines usually prevents the appearance or reduces the intensity of discontinuance symptoms.
4. *Type of benzodiazepine*. Benzodiazepines are classified into short and long half-life compounds. These terms refer to the time it takes for liver metabolism to remove (clear) benzodiazepines from the body. Short half-life benzodiazepines are cleared very rapidly, usually from 4 to about 16 hours, depending on the drug. In contrast, long half-life benzodiazepines may take anywhere from 24 to 100 or more hours to be cleared. Since the appearance of discontinuance

symptoms depends, in part, on the rapidly diminishing blood level of the drug, abrupt cessation of the short half-life benzodiazepines is more likely to produce discontinuance symptoms. Controversy exists about whether other factors that distinguish one benzodiazepine from another are associated with the appearance of a discontinuance syndrome.

MANIFESTATIONS

Virtually all who experience discontinuance symptoms from benzodiazepines describe increased anxiety, restlessness, and difficulty falling asleep. These symptoms may be mild, little more than an annoyance for a few days, or they may be quite severe and even more intense than the symptoms of anxiety or insomnia for which the drugs were initially prescribed. The reappearance of the initial symptom, such as anxiety or insomnia, only in greater severity, is known as the *rebound symptom*. Rebound symptoms usually occur within hours to days of benzodiazepine discontinuance and then gradually fade. In some cases, however, they may be so intense that the patient resumes taking the benzodiazepine to avoid the discontinuance symptoms themselves. Thus a cycle of benzodiazepine dependence may begin—the patient is taking the drug primarily to treat or prevent rebound discontinuance symptoms from appearing, rather than treating an underlying anxiety or sleep disorder.

Benzodiazepines that are given to induce sleep may also be associated with the development of discontinuance symptoms. Rebound insomnia, the most common discontinuance symptom, typically occurs on the first night and sometimes the second night after discontinuance of short half-life benzodiazepines. Rebound insomnia may be so intense during these nights that the patient may be unwilling to risk another sleepless night and so returns to taking the benzodiazepine hypnotic. Rebound insomnia is less common with long half-life benzodiazepines.

If untreated, rebound symptoms may sometimes persist for many months. When this occurs it is difficult to determine whether the symptoms are still manifestations of discontinuance or are the result of the return of the problems (anxiety, insomnia) for which the drug was originally prescribed. Sometimes new symptoms that did not exist before the patient took benzodiazepine appear after discontinuance; these are termed true withdrawal symptoms, indicating a change in CNS functioning. Usual withdrawal symptoms include headache, anxiety, insomnia, restlessness, depression, irritability, nausea, loss of appetite, gastrointestinal upset, and unsteadiness. Patients may also experience increased sensitivity for sound and smell, difficulty concentrating, and a sense that events are unreal (depersonalization). Unusual withdrawal symptoms include psychosis and seizures.

OCCURRENCE OF SEIZURES

From a medical perspective, the most serious of all discontinuation symptoms is the development of withdrawal seizures. Seizures are generally grand mal in type (tonic-clonic; epileptic) and may threaten the life of the patient. They tend to occur only when higher-than-therapeutic doses are abruptly discontinued.

Withdrawal seizures almost always occur when the patient has been taking other drugs, such as ANTIDEPRESSANTS or ANTIPSYCHOTIC agents, together with a benzodiazepine.

COEXISTING PSYCHOPATHOLOGY

Apparently some people are more predisposed to develop the discontinuation syndrome than others. Those who have been previously dependent on benzodiazepines, alcohol, or other SEDATIVE-HYPNOTIC drugs, such as barbiturates, are more likely to experience discontinuance symptoms after the termination of benzodiazepine therapy. It is especially important, therefore, that such patients never stop taking their benzodiazepines abruptly.

TREATMENT

Although a variety of treatments have been proposed for the discontinuance syndrome, the best approach is to prevent its occurrence. Logically, prevention consists of a very gradual tapering of the benzodiazepine dose, with a firm rule never to discontinue these medications abruptly if they have been taken for more than a few weeks on a regular basis.

Even with gradual tapering, however, some patients may continue to experience rebound or withdrawal symptoms that are sufficiently disturbing to

require treatment. Drugs that tend to reduce CNS hyperarousal states, such as anticonvulsants, have sometimes been employed to treat benzodiazepine discontinuance. Alternatively, benzodiazepine treatment is restarted using a long half-life compound that is then very gradually tapered.

CONCLUSION

For the great majority of patients, benzodiazepine discontinuance is a relatively benign and short-lived syndrome; many, if not most, patients have no difficulty. It is generally agreed that the therapeutic benefits of taking benzodiazepines far outweigh any problems with discontinuance when drug treatment is no longer necessary.

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Cocaine Withdrawal from cocaine was mentioned by H. W. Maier in his 1928 classic *Der Kokainismus* (Cocaine Addiction), but systematic efforts to describe and understand cocaine withdrawal did not begin until the 1980s, during the most recent epidemic.

The features of withdrawal from depressant drugs such as ALCOHOL and OPIOIDS are more robust and recognizable than from a stimulant drug such as cocaine—since the grossly observable pattern of physiologic disturbances seen in depressant withdrawal syndromes are not observed when a person stops using cocaine. This difference highlights and contrasts depressant withdrawal and stimulant withdrawal, such as is seen with cocaine.

In alcohol withdrawal, for example, the drinker may manifest all or several of the following set of symptoms and signs: tremulousness, elevated pulse and blood pressure, sweatiness, nervousness, and (rarely) seizure. Craving, or desire, for alcohol is typically high during this period, since the drinker knows it will quickly relive the withdrawal symptoms. These symptoms and signs will generally resolve within three to ten days of ceasing the intake of alcohol. Finally, the withdrawal syndrome is reproduceable—individuals tend to experience the same symptoms every time they withdraw from alcohol. Withdrawal from OPIATES such as HEROIN and MORPHINE similarly involves physiologic symptoms and signs—diarrhea, gooseflesh, changes in pulse and blood pressure, muscle cramps, stomach cramps, and anxiety.

In the cocaine abuser, the absence of early apparent physiologic symptoms and signs of cocaine withdrawal led to a widely held misperception (among the public and medical professions alike)—that cocaine was not an addicting drug. This misperception was based in part on cocaine's lack of a withdrawal syndrome that was as easy to characterize as those associated with alcohol or opioids.

If cocaine withdrawal does not evidence physiologic symptoms and signs, then how can it be recognized? The concept has been advanced that cocaine withdrawal is mediated through the central nervous system, that observable symptoms are limited to subjective states such as depression, lack of energy, agitation, and craving for cocaine. Evidence that neurophysiologic dysfunction may underlie reported symptoms consists of electroencephalogram (EEG) changes, neurohormonal dysregulation, and dopamine-receptor alteration (Satel et al., 1993).

In 1986, Gawin and Kleber were among the first to describe the clinical course of the symptoms following cocaine cessation, and they proposed a three-phase model of cocaine abstinence. Although this triphasic model has gained wide acceptance, other recent data suggest the model may not be applicable in all clinical situations, as will be discussed below.

The triphasic model postulated by F. Gawin and H. Kleber on the basis of interviews with outpatients comprises three phases that occur after cocaine cessation: (1) crash, (2) withdrawal, and (3) extinction. The *crash* is described as an extreme state of exhaustion that follows a sustained period of cocaine use (binge); it can last between nine hours and four days. The beginning of the crash is marked by craving, irritability, dysphoria, and agitation; the middle is characterized by yearning for

sleep; and the late crash by hypersommnolence (excessive sleep). Certain individuals may experience especially severe depressed mood in the early stages of cocaine abstinence and are at risk for suicidal ideation and action at this time. This may be particularly true for those who are struggling with ongoing problems with depression. When alcohol is used with cocaine, depressed mood can intensify. Also alcohol-induced reduction of impulse control, combined with cocaine crash-related despair, creates a high-risk situation for suicide.

As depression and desire for sleep increase, craving subsides. Upon awakening from a lengthy sleep, the individual enters a brief euthymic (normal) period with mild craving. This is followed by a protracted period of milder *withdrawal*, lasting 1 to 10 weeks, during which time craving reemerges and anhedonia (loss of pleasure) prevails. This is succeeded by an indefinite period of *extinction*, marked by euthymic mood and episodic craving.

According to the triphasic model, protracted withdrawal is represented by phase 3, thus beginning after two weeks or more. These clinical phenomena are believed to reflect disturbances in central catecholamine (neurotransmitter) function produced by long-term cocaine use. The crash phase, however, can occur even in first-time stimulant users—if their initial episode is of sufficient duration and dose.

Recently, two groups of investigators have observed a mild constellation of subjective features of the post-crash cocaine abstinence syndrome as described by Gawin and Kleber, but without the phases those investigators described. Weddington et al. (1990) documented the absence of cyclic or phasic changes in mood states, cocaine craving, or interrupted sleep in twelve cocaine-dependent inpatients examined during a four-week period. All had abstained from continuous cocaine use within the preceding forty-eight hours. No euthymic window was evident, although subjects reported significantly greater depressed mood than nondrug-using controls at admission. Subjective symptoms of mood, craving, and anxiety displayed a steady and gradual improvement during the course of the study. By the end of week 4, the cocaine users and the nondrug-using controls had comparable scores. Thus, withdrawal had been completed over the course of one month.

Similar subjective findings emerged from a study by Satel and coworkers (1991), in which 22 newly abstinent COCAINE-dependent males were observed during a 21-day hospitalization. Over the 21 days, both subjective and objective ratings of mood and arousal showed gradual improvement. Although all subjects had consumed cocaine within twenty-four hours of admission, some claimed that they had slept prior to admission and thus the crash phase may have been missed in both studies.

The major differences between the triphasic model and the reports made by the two groups of investigators who actually observed cocaine users during withdrawal reside in the *euthymic interval*, the severity of symptoms, and the time-to-recovery of mood and craving. Nevertheless, all three studies are consistent with at least a mild postcessation syndrome. It may be important that the original conceptualization of the triphasic cocaine withdrawal was derived from observations of outpatients. The subsequent studies involved inpatients, who were largely protected from environmental cues.

Divergent findings with respect to a delineation between acute and protracted withdrawal is related to the difficulty in distinguishing acute cocaine withdrawal symptoms from those that characterize protracted withdrawal. (This distinction is less blurred in alcohol and opiate withdrawal, where the intense physiologic symptoms take place within the first week of ceasing usage—and the protracted syndromes, though uncomfortable, are considerably milder.) Conditioned withdrawal symptoms have been documented in opiate users and in alcoholics. These represent actual physiologic correlates of pharmacologic withdrawal (e.g., changes in skin temperature, gooseflesh, diarrhea, and cramps, accompanied by intense craving for the drug) elicited in *drug-free* individuals after they complete acute withdrawal and are exposed to reminders of drug use (e.g., visual or olfactory cues).

Conceivably, Gawin and Kleber's subjects may have experienced a delineated withdrawal, with a clear transition to a protracted state—because as outpatients they were constantly exposed to environmental cues and reminders of drug use. In inpatients, symptoms of acute cocaine withdrawal may be less clearly delineated. Constant exposure to cues may intensify a clinically observable acute syndrome, making the acute-protracted distinction easier to recognize. Environmental influences on clinical withdrawal may determine, in part, the severity of the observable manifestations of

changes in neuroreceptors and neurotransmitters that accompany chronic cocaine use. Clearly, the behavioral and subjective manifestations are variable.

In addition, it is possible that nonorganic factors play a role in the prolonged psychic distress following termination of the chronic use of cocaine. Indeed, the period of abstinence following heavy drug use is a time when addicts must squarely face the shambles of their lives—the destruction of their families, loss of jobs, financial ruin, insults to health and self-esteem. Cocaine craving during this period is likely triggered by negative mood states as well as a conscious desire to obliterate the psychological pain with more drug—a return to drug use.

Pharmacologic treatment for the crash phase of withdrawal has received attention, although most treatment centers do not use medicines to help detoxify crashing cocaine addicts. The two major drugs that have been reported useful during the crash phase are bromocriptine and AMANTADINE. The action of these two drugs is to enhance transmission of the NEUROTRANSMITTER dopamine. Indeed, drugs that have this action were specifically chosen by investigators for use in treatment trials, because they assumed such drugs would reverse the reduction in dopamine levels in the brain that normally follows cocaine binging. This reduction is presumed to account for the depression, irritability, agitation, and drug craving during the crash phase.

Pharmacotherapy for detoxifying cocaine addicts becomes especially important when a person is also dependent on alcohol or opioids. Such codependent states are very common. The usual choice for alcohol detoxification is a BENZODIAZEPINE drug (e.g., Librium); for opiate withdrawal, a choice exists for METHADONE, CLONIDINE, NALTREXONE, or combinations of these. Important interactions occur between cocaine and other drugs of abuse. For example, cocaine plus alcohol in the body produces a compound called COCAETHYLENE. This compound produces more intense and longer euphoria—but it also heightens the risk of death, due to cardiac arrythmia. Also, in methadone clinics, cocaine use has been noted to be of epidemic proportion; the opiate methadone mediates the jitteriness and paranoia that often accompanies cocaine use. Some evidence shows that cocaine addicts, who are also dependent on opiates, may have less severe opiate withdrawal than those who do not use cocaine.

Cocaine CRAVING is the major cause of relapse in individuals trying to attain and sustain abstinence. Such craving is typically most severe in the early stages of withdrawal from cocaine, although, as Gawin and Kleber noted in their model, cocaine addicts are extremely cue-responsive; reminders of drug use in the community (old copping areas, people with whom they used to get high, etc.) can stimulate craving at any stage of abstinence. Thus, people with severe addiction trying to relinquish cocaine must often enter a rehabilitation program with an outpatient phase that lasts from one to two years, at minimum.

Ideally, a heavy cocaine user with good social support and resources could enter an inpatient program to undergo detoxification (when sustained craving is usually at its peak) for a minimum of one week, before beginning outpatient work. Individuals without social support or a stable living situation can often benefit from weeks to months in a residential-treatment setting. Since it appears that the immediate postcessation phase may be milder for inpatients, this might be a way for addicts to experience less distress and to better concentrate on therapy and education. It might also be a period of time when they feel a somewhat greater sense of control over themselves—control being especially difficult to achieve when craving for cocaine is high. It is critical to realize, however, that many patients can develop a false sense of control over the addiction because as inpatients they are protected from environmental cues that trigger craving. Thus gradual reintroduction to the ambulatory environment, psychological preparation of the patient for the likely return of craving, and therapy using relapse-prevention techniques (a form of cognitive therapy) are all necessary.

(SEE ALSO: *Amphetamine; Cocaine*)

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Nicotine (Tobacco) Nicotine is one of the most addicting substances known; indeed, the risk of becoming dependent on nicotine following any tobacco use is higher than the risk of becoming dependent on alcohol, cocaine, or marijuana following any use of those substances. Among multiple drug users, quitting tobacco use is often cited as more difficult than giving up alcohol or cocaine. Most current views of tobacco use include physiological addiction as a factor in the difficult course of achieving smoking cessation.

As with other drugs that result in dependency, nicotine, the active ingredient in tobacco, shares characteristics with other drugs that result in addiction. First, the administration of such drugs alters central nervous system function at specific receptors and often changes structure; in addition increases (up regulation) or decreases (down regulation) in receptor numbers occur. Second, repeated exposure to the drug results in tolerance, and the individual must progressively self-administer higher doses of the drug to obtain the same effects that initially occurred at lower doses. Third, as cellular and neurological functioning adapt to the continuous presence of the drug during tolerance development, a state of physical or physiological dependence is produced so that removal of the drug is accompanied by feelings of dysphoria and an inability to function normally. The individual then needs continued drug intake to function normally. Finally, a hallmark of dependence-producing drugs is that they serve as biological reinforcers for animals, including humans.

NICOTINE TOLERANCE AND DEPENDENCE

Nicotine is the pharmacologic agent that acts on the central nervous system (CNS). Its actions are seen in the brain where it operates on cholinergic receptors. The cigarette is a very fast and effective delivery system and effects occur rapidly after a single inhalation of tobacco smoke. Nicotine quickly crosses the blood–brain barrier and, once in the brain, interacts with brain receptors. Nicotine alters moods and acts on pleasure-seeking receptors in the brain, including dopamine and serotonin. The nicotine alkaloid affects numerous body systems: It raises blood pressure and the heart rate. It also affects the peripheral nervous system (PNS) and both stimulant and depressive effects are observed in cardiovascular, endocrine, gastrointestinal, and skeletal systems.

Initial exposure to nicotine is not a pleasant experience, often causing sickness, intoxication, and disruptions in physiologic functioning. After a period of daily smoking (assumed to be at least a few weeks), the body adapts to nicotine and the unpleasant effects are less pronounced. Tolerance develops and physical dependence occurs. Smokers are free to self-administer the dose of nicotine they desire, and tolerance increases so that the amount of nicotine used per day continues to increase. The level of dependence is strongly related to the dose of nicotine.

As a smoker becomes physically dependent on, that is, addicted to, smoking, the smoker feels normal, comfortable, and effective when taking nicotine, and dysphoric, uncomfortable, and ineffective when deprived of nicotine. The process of dependence development weakens the ability of the person to achieve and sustain even short-term abstinence. Thus, in the nicotine-dependent person, "normal" function depends on nicotine, and the removal of nicotine results in impairment.

NICOTINE WITHDRAWAL SYMPTOMS

The DSM-IV recognizes nicotine dependence as a substance-related disorder, with a well-defined withdrawal syndrome. The potential withdrawal symptoms include dysphoric or depressed mood; insomnia; irritability, frustration, or anger; anxiety; difficulty concentrating; restlessness; decreased heart rate; and increased appetite or

weight gain. The severity of the symptoms will depend on the severity of nicotine dependence. Withdrawal symptoms are strongest in the first few days after smoking cessation, and usually diminish within a month, although some smokers may continue to have withdrawal symptoms for many months.

A number of other sequelae accompany smoking cessation. There is evidence that cognitive ability is impaired when smoking cessation is attempted. The cognitive deficits are correlated with disruptions in brain electrophysiologic function. Figure 1 shows that deficits in an arithmetic task follow a similar time course as changes in the brain's electrical activity. These effects begin a few hours after the last cigarette (dose of nicotine), peak during the first few days of abstinence (when smokers trying to quit are most likely to relapse), and mostly subside within a few weeks. Another study of cognitive impairment, using four complex cognitive tasks during withdrawal from smoking in heavy smokers, ex-smokers, and those who had never smoked, assessed ability to perform those tasks; smokers with 12 hours of abstinence had the worst scores on the tasks.

Another symptom associated with withdrawal is craving for cigarettes. Craving is strongly related to the degree of nicotine dependence. Craving may last 6 months which is longer than some of the other symptoms associated with tobacco withdrawal. Craving is a major obstacle to cessation and together with other indicators of nicotine dependence is strongly related to relapse, with the majority of smokers who attempt to quit relapsing within the first week of cessation.

Although the foregoing are universal, albeit with some variation among individuals, some withdrawal symptoms are unique to individuals with specific characteristics. Smokers with a history of major depression, for example, are at some risk of having another depressive episode during the cessation process. Smokers with comorbid disorders such as alcoholism or illicit substance abuse are likely to have more severe withdrawal symptoms as they attempt to address more than one dependency.

The withdrawal syndrome is undoubtedly biologically based; however, behavioral factors have a strong influence on smoking cessation. Cigarette smoking involves a number of rituals that become ingrained into the smoker's daily life, resulting in numerous individual, social, and environmental

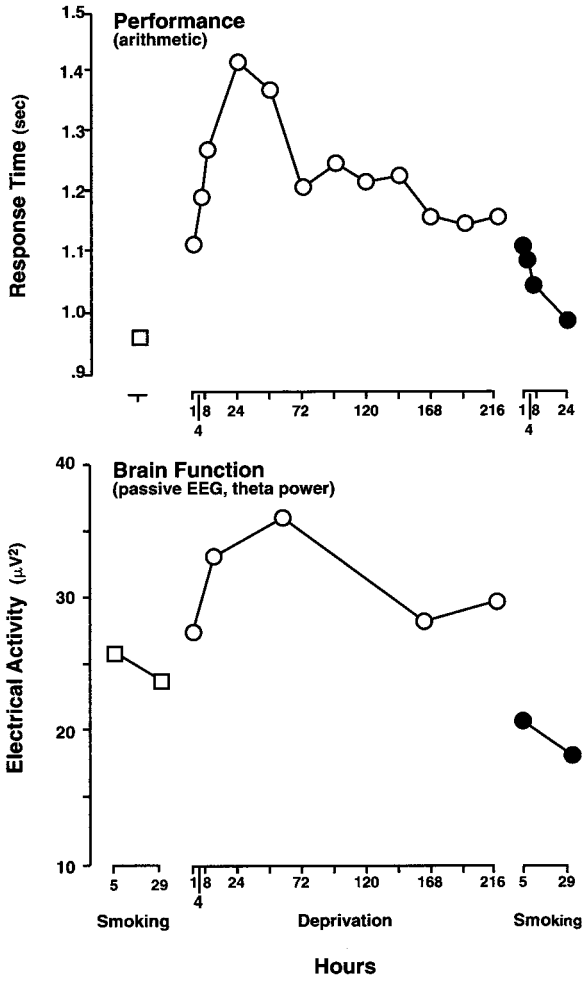

Figure 1
Cognitive Performance and an Electrophysiological Measure of Brain Function during Smoking and Abstinence.

prompts to smoke. At the individual level, the smoker may associate a cup of coffee, the end of a meal, or watching television as a prompt to light a cigarette. Socially, being with friends or family members who smoke represents other cues to smoke, while presence in a situation where smoking is not allowed may result in powerful negative feelings about smoking cessation. Environmental stimuli—being in bars or other places where the prevalence of smoking is high—are likely to reinforce the smoker's desire to smoke. Exposure to any of the cues to smoke may result in relapse.

TREATMENT OF NICOTINE WITHDRAWAL SYMPTOMS

Two pharmacologic approaches, nicotine replacement therapy and drugs to manage symptoms associated with withdrawal, have been taken to reduce nicotine withdrawal symptoms. In addition, behavioral approaches for withdrawal have been tested.

Nicotine replacement therapy. The purpose of nicotine replacement is to substitute a safer and controllable form of nicotine to the smoker to aid in cessation. Although nicotine replacement delivery systems vary, all attempt to reduce the amount of nicotine available during cessation so that an individual is weaned from nicotine addiction. Two nicotine replacement therapies are available over-the-counter: nicotine polacrilex gum and the transdermal nicotine patch. Two other delivery systems are available through prescriptions: an oral nicotine inhalation system and a nasal nicotine spray. The effectiveness of each of the systems has been well-established in randomized, controlled trials.

Symptom treatment. A number of drug therapies have been approved to alleviate or reduce some of the discomfort that accompanies smoking cessation. The best known is bupropion (Zyban), which is effective as an antidepressant. Bupriopion, however, is also effective in smokers who have no history of depression; thus, other factors may be involved in the success of this drug in smoking cessation. Another antidepressant, nortriptyline, has also been shown to be useful for smoking cessation. Clonidine, originally used to treat hypertension, appears to be modestly effective in blocking the cravings for nicotine, especially in women. Other pharmacologic therapies are being tested for their value in ameliorating the withdrawal symptoms of cessation. These include mecamylamine, which is thought to block the reinforcing action of nicotine, and anxiolytics and benzodiazepines, which generally lower stress and decrease anxiety.

Behavioral approaches. Behavioral approaches for preventing relapse have a long history of use in smoking cessation. Behavioral strategies generally focus on the social reinforcers of smoking. The most effective behavioral programs are those that have multiple components. Various behavioral strategies include contracting to quit, with the smoker making a monetary donation if success is not attained; group support, where individuals support each other in their quit attempts; and cognitive restructuring, where smokers are taught to think differently about smoking and cigarettes. Other components include relaxation exercises, coping tactics, visualization and addressing of tempting situations, simple messages to deal with withdrawal symptoms (e.g., deep breathing, delay so the urge will pass, drink water, do something else), and stimulus control (e.g., getting rid of ashtrays, having a smoke-free home). Multicomponent behavioral programs have had much success in helping smokers achieve cessation. Much research suggests that nicotine replacement or pharmacologic approaches without a behavioral component have significantly lower success rates than those with a behavioral component.

SUMMARY

Nicotine is a very addictive drug that affects the central nervous system. Its use results in tolerance and dependence, so that the user feels most normal when using tobacco. A clear nicotine withdrawal syndrome is known; smokers attempting cessation may have dysphoria, insomnia, irritability, anxiety, difficulty concentrating, restlessness, decreased heart rate, and increased appetite. Further, cognitive ability is somewhat impaired during cessation, strong craving for the drug is present, and powerful behavioral cues make cessation difficult. New approaches to the withdrawal syndrome include the administration of nicotine in a safer delivery system that can be tapered over time, and drugs to counter the unpleasant symptoms of withdrawal. Along with behavioral treatment, such pharmacologic tools may assist the smoker in achieving cessation.

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Nonabused Drugs Although drug withdrawal is often considered synonymous with matters relating to drug abuse, a number of drugs which have no abuse potential and are prescribed for medical illness are associated with clear symptoms of withdrawal when their use is abruptly discontinued. The symptoms do not necessarily indicate drug dependence, a syndrome that has several features, including tolerance, inability to control drug use, and continued drug use despite deleterious effects.

CARDIOVASCULAR DRUGS

Beta-Adrenergic Blockers. These drugs are taken by many people to treat hypertension (high blood pressure), angina pectoris (chest pain from heart muscle deprived of oxygen), heart arrhythmias following heart attack, and for migraine headache. The mechanism for each of these effects is related to the drug occupying the beta-adrenergic receptors in the blood vessels and the heart. When a patient abruptly stops taking a beta blocker, particularly when angina pectoris is the symptom being treated, a marked increase in the frequency and/or severity of angina pectoris may occur. This occurs within the first few days of discontinuing the beta blocker; it may be prevented by slowly decreasing the drug dose over several days before completely stopping the drug. The discontinuation symptom is probably related to an increased sensitivity of the beta receptor for the body's own hormones NOREPINEPHRINE and epinephrine, when its antagonist, the beta blocker, is suddenly removed. The withdrawal syndrome disappears in a few days, consistent with the time required for beta-adrenegic receptor reregulation.

Clonidine. This drug is used for hypertension and to treat withdrawal from opiate narcotics. Its mechanism of effect is stimulation of alpha(type 2)-adrenergic receptors in the central nervous system, which results in decreased stimulation of nerves that release norepinephrine and epinephrine in blood vessels. When CLONIDINE is abruptly stopped, blood pressure increases to well above baseline levels and may become dangerously high. This occurs within one to two days after stopping the drug and is prevented by slowly (over several days) decreasing the drug dose before stopping it completely. This may be due to a "rebound" overstimulation of norepinephrine and epinephrine re-

leasing nerves in blood vessels. This rebound hypertension disappears within a few days, again consistent with the time required for alpha-adrenergic receptor reregulation.

Nitroglycerin and Other Nitrates. These drugs are taken to treat angina pectoris. They cause the relaxation of blood vessels by the activation of an intracellular enzyme, guanylyl cyclase, which catalyzes formation of cyclic GMP (guanosine monophosphate). The coronary arteries (blood vessels which supply heart muscles) relax when exposed to nitrates. If the coronary arteries are blocked by atherosclerosis, causing insufficient blood supply to the heart, angina pectoris can occur. Relaxation of these arteries improves blood supply to the heart and the chest pain rapidly disappears. When nitrates are taken continuously for relief of chest pain, then abruptly discontinued, rebound angina pectoris which is more frequent or more severe than the angina experienced pretreatment may occur. This begins within a few hours of the last nitrate dose and in a time course consistent with the metabolism and removal of the nitrate drug from the body. If the nitrate dose is slowly decreased before discontinuation, the rebound angina may be prevented. The mechanism for this withdrawal syndrome is not certain, however, it is probably related to loss of the chronic activation of guanyl cyclase during nitrate therapy and abnormal regulation of the contractile apparatus in the blood vessel muscle, leading it to have rebound contraction.

NEUROPSYCHOPHARMACOLOGICAL DRUGS

Antidepressants. These drugs are used to treat major depressive illnesses; therefore they are frequently administered daily for periods of weeks or months. Abrupt discontinuation of any of the major classes of ANTIDEPRESSANTS may result in discontinuation reactions. Antidepressants vary in their ability to cause reactions, and reactions are more common after abrupt discontinuation and longer courses of treatment. Common symptoms include gastrointestinal problems like nausea, abdominal pain, and diarrhea. In addition, some patients complain of a flulike illness consisting of weakness, chills, fatigue, headaches, and muscle aches. Central nervous system dysfunction characterized by difficulty falling asleep, anxiety, vivid dreams or nightmares, or jitteriness can also occur, as can such affective symptoms as irritability and low mood. Symptoms usually start a few days after termination of the antidepressant and continue anywhere between one day and three weeks. The mechanism of withdrawal may result from up-regulation and increased sensitivity of the muscarinic receptor, which is blocked by these drugs. During chronic heterocyclic-antidepressant treatment, muscarinic-receptor sensitivity increases. When receptor blockade is suddenly stopped, overactivity of these receptors in the digestive tract and brain causes the withdrawal symptoms.

Withdrawal symptoms of a class of antidepressants known as selective serotonin reuptake inhibitors (SSRIs) can be particularly deceptive and therefore problematic because some of the symptoms are like those an individual experiences with a relapse of depression. In such instances, individuals may be at risk of being prescribed even more antidepressants. This cycle of drug treatment is a significant problem, especially since many government agencies have stepped up efforts to treat depression and managed care plans are increasingly turning to antidepressants as a treatment for depression. However, SSRIs have several distinct discontinuation symptoms, including dizziness and such sensory abnormalities as electric shocklike sensations, numbness, and paraesthesia. The symptoms typically go away the day after antidepressant treatment has resumed, unlike a true depressive relapse, which takes longer. Therefore, with care, a misdiagnosis of a relapse of a psychiatric illness can often be avoided. In addition, to reduce the risk of withdrawal symptoms, some physicians have recommended that antidepressants be gradually reduced over a four week period rather than abruptly discontinued.

Monoamine Oxidase Inhibitor (MAOI) antidepressants drugs interfere with the enzymatic breakdown of NEUROTRANSMITTERS (such as norepinephrine) in the brain. Sudden discontinuation after high chronic dosing has been associated with psychosis and delirium—consisting of visual hallucinations as well as mental confusion. Milder symptoms consisting of anxiety, vivid dreaming, or nightmares may also occur. The exact mechanism of withdrawal has not been well studied, but it may relate to the way nerve cells regulate the release of neurotransmitters in the brain. Presynaptic receptors serve to provide a message to nerve cells about how much neurotransmitter is present in the

synapse—the space between two nerve cells where messages, in the form of neurotransmitters, flow between cells. When activated, these types of receptors (present on the surface of the nerve cell releasing the message) inhibit any further release of neurotransmitters. As a result of treatment with MAOI, decreases in the number of presynaptic receptors occur, resulting in larger amounts of neurotransmitter being released before the cell shuts down release. The increase in the amount of neurotransmitter may result in withdrawal symptoms that abate over a period of days after discontinuation.

Major Tranquilizers. NEUROLEPTIC agents are commonly used in psychiatric practice for the treatment of psychotic disorders such as schizophrenia. These agents all block brain dopaminergic receptors—the basis for their effectiveness in treating psychotic illness. These agents also inhibit emesis (vomiting), which is caused by dopaminergic blockade in the brain as it affects the perception and initiation of vomiting. Chronic blockade results in increased numbers of these receptors. The abrupt discontinuation of this class of drugs results in nausea, vomiting, and headaches. The antipsychotic and antiparkinsonian effects of neuroleptics are also still present for a prolonged period. According to some research, it is not known whether the prolonged effects of neuroleptic drugs in humans are due to the continued presence of drug in brain tissue or to long-lasting, drug-induced physiologic changes.

Clozapine is in a class of atypical antipsychotic drugs associated with discontinuation symptoms. Although atypical antipsychotics may be different from other neuroleptic drugs, there are also significant differences among these drugs in their effects on the receptors of the central nervous system. Clozapine interacts with a wide range of neurotransmitter receptors, especially serotonin receptors. Common discontinuation symptoms of clozapine include delusions, hallucinations, hostility, and paranoia. The underlying mechanism of these symptoms is thought to be cholinergic supersensitivity.

OTHER DRUGS

Baclofen. As a muscle relaxant, this drug is used to treat muscle spasticity associated with certain paralytic states. It acts as an agonist (mimic) of the inhibitory neurotransmitter in the spinal cord, GAMMA-AMINOBUTYRIC ACID (GABA). Therefore baclofen inhibits excitatory neural pathways, which are modulated by GABA and which ultimately stimulate skeletal muscles to contract. This is a rather selective effect as there are two types of GABA receptors and pathways, GABA-A and GABA-B, of which baclofen only acts on GABA-B receptors. When baclofen is used to treat muscle spasm, the excitatory pathways of the spine are chronically modulated and inhibited. When baclofen is abruptly discontinued, this inhibition is released and, within a few hours as is consistent with the rate of disappearance of baclofen, the excitatory pathways rebound—probably due to a transient unregulated state. The symptoms experienced by a person suddenly discontinuing baclofen may include auditory and visual hallucinations, severe anxiety, increased heart rate and blood pressure, and generalized seizures. Such clinical symptoms are consistent with the impaired modulation of neural-excitatory pathways. When baclofen dosage is gradually reduced before discontinuation, these symptoms either do not occur or are attenuated, indicating that the inhibitory/excitatory-neural-pathway balance, which has been disturbed by the excessive inhibitory modulus of baclofen, has the capacity to reregulate over a few days.

Corticosteroids. The drug prednisone will be discussed specifically; however, the biological changes that result in withdrawal phenomena after discontinuation of long-term prednisone treatment hold for all members of the glucocorticoid group. When, for example, a significant dose (5–10 mg daily) of prednisone is taken for a period of several weeks, a series of feedback regulatory events occurs resulting in the patient becoming functionally adrenally insufficient. Specifically, in mimicking the endogenous corticosterone cortisol, prednisone signals the pituitary gland to stop the synthesis and release of the adrenocorticotrophic hormone (ACTH) and, perhaps, the hypothalamus to stop the release of the corticotropin-releasing hormone (CRH). ACTH release from the pituitary, which normally stimulates the adrenal glands to produce corticosterones and which is modulated by the hypothalamic CRH, is blocked by the drug prednisone when ingested in the above dose or greater. Not only does adrenal production of cortisol decrease but also the adrenal glands atrophy.

When prednisone therapy is abruptly discontinued, the atrophic adrenal glands no longer respond to ACTH stimulation, so the patient has symptoms

of adrenal insufficiency. Clinically, this is manifested by fatigue, weakness, electrolyte imbalance, and the lack of many bodily responses to stress. If an individual remains in this state for more than a few hours, severe illness and death can be expected. When the adrenal glands become atrophic during long-term prednisone treatment, if the prednisone is to be discontinued, it must be done with slowly decreasing doses over many weeks to permit the adrenal glands sufficient time to regrow to their normal size under the influence of ACTH stimulation and to have sufficient stores of the body's own cortisol to respond to stress in a physiologically appropriate manner.

COMPARISONS WITH DRUGS OF ABUSE

ALCOHOL is one of the most common drugs of abuse. If alcohol withdrawal is used as a basis for comparison, marked similarity in effect is noted when considering the cardiovascular drugs (beta-blockers, clonidine, nitrates) and baclofen. Alcohol, a nonspecific central nervous system depressant, leads to an ill-defined reregulated state, allowing habituated individuals some level of function during their chronic alcohol-induced depressive state. Abrupt cessation of alcohol consumption results in loss of the depressive state, with a rebound state of psychic and physical excitation. This is not unlike the cardiovascular drugs and baclofen; there, the withdrawal syndrome is the clinical manifestation of a neural- or cellular-regulatory system that has reached a new homeostatic state under the influence of the drug and the sudden drug removal leaves insufficient time for physiological reregulation. In the case of corticosteroids, the reverse of this mechanism occurs. Here, the physiological regulation which has occurred during prednisone therapy leads to loss of the capacity to have a physiological response, instead of an over-response.

Human physiology is characterized by the coordinated and finely tuned operation of multiple messaging systems, exhibiting both positive and negative feedback regulation, with multiple levels of control. All the drugs mentioned exert both their desired and undesired effects by interfering with these systems. In the drug-treated individual, homeostasis is maintained by counteracting some of the drug effects at the cellular level. Such adaptation is not without cost. The sudden discontinuation of a drug to which the system has adapted results in a period of disequilibrium between the affected messaging systems. The disturbed physiology is expressed by specific withdrawal symptoms.

(SEE ALSO: *Anabolic Steroids; Withdrawal: Alcohol*)

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WOMEN AND SUBSTANCE ABUSE

There are gender differences in the prevalence of substance abuse.

ALCOHOL AND TOBACCO USE

General population studies indicate that fewer women drink than men, and women who do drink consume less alcohol than men. Of the estimated 15 million alcohol-abusing or alcohol-dependent individuals in the United States, fewer than one-third are women. In the 1993 NATIONAL HOUSEHOLD SURVEY on Drug Abuse (NHSDA), 57 percent of men reported they drank alcoholic beverages in the previous month, compared with 43 percent of women. The NHSDA defines heavy alcohol use as 5 or more drinks per day on each of 5 or more days in the past 30 days. By this definition, in 1993 men were much more likely than women to be heavy drinkers (10 and 2 percent, respectively).

It has been suggested that male and female sex roles, and therefore drinking norms, have become more similar in recent years. Some sex-role changes that could increase opportunities for, and acceptability of, female drinking include greater female labor force participation, delayed marriage and childbearing, and more equitable sex-role attitudes. According to this convergence thesis, greater sex-role equality may cause PROBLEM DRINKING and ALCOHOLISM to increase among women. However, recent epidemiological data reveal little evidence of increased female alcoholism or problem drinking. Changing female drinking patterns have resulted more in a reduction in female abstainers than an increase in problem drinkers. Nevertheless, there is some evidence for convergence in the youngest cohorts, with the smallest sex differences in heavy drinking being for youths aged twelve to seventeen (2 percent of boys and 1 percent of girls in 1993). Among adults aged thirty-five and older, men are eight times as likely as women to be heavy drinkers (8 percent compared with 1 percent).

There is greater evidence of sex-role convergence in TOBACCO use. In 1955, 52 percent of adult men smoked, compared with 25 percent of adult women. Since then, the proportion of men who smoke has decreased markedly while rates among women have held fairly steady. Among adults aged 35 and older in 1993, 27 percent of men and 21 percent of women were current smokers. Among youths aged twelve to seventeen, girls have surpassed boys in their rates of current cigarette use (10 percent of girls compared with 9 percent of boys in 1993). Because boys are more likely than girls to use smokeless tobacco products, however, their overall rates of nicotine addiction still exceed girls' rates.

Biener (1987) reviews factors that have contributed to the convergence in male and female smoking. Product developments such as filtered and low-tar cigarettes have made smoking easier for women to tolerate physically. Tobacco companies have targeted ADVERTISING to make smoking attractive to young women. Once tobacco use is initiated, women are less likely than men to quit smoking and, compared with men who have quit smoking, women quitters are more likely to relapse.

The convergence in male and female smoking rates has been accompanied by a convergence in smoking-related health problems. For example, lung cancer deaths among women have increased markedly since the 1970s, and lung cancer now surpasses breast cancer as the leading cause of CANCER deaths among women.

ILLICIT DRUG USE

Males are far more likely than females to be arrested for possessing or selling illicit drugs. In 1992, for example, the Federal Bureau of Investigation reported that only 16 percent of those arrested for drug-abuse violations were female. At all

ages, males are more likely than females to use illicit drugs. Gender differences are smallest among adolescents aged twelve to seventeen and among adults aged thirty-five and older, and largest among young adults aged eighteen to thirty-four, the age range in which illicit-drug use is most prevalent. In the 1993 NHSDA, 11 percent of men, compared with 6 percent of women, aged twenty-six to thirty-four reported they had used some illicit drug in the previous month. Nineteen percent of men and 8 percent of women reported current (i.e., past month) illicit-drug use in 1993. Among both men and women, marijuana is the most frequently used illicit substance, with 16 percent of men and 6 percent of women aged eighteen to twenty-five reporting current use.

COCAINE use has decreased since the mid-1980s, and is rare compared with marijuana use. Sex differences in regular cocaine use are small. In the young adult age group, where use is most common, 1.7 percent of men and 1.4 percent of women reported cocaine use in the past month. In 1993, among youths aged twelve to seventeen, boys and girls were equally liken to report cocaine use in the past month (0.4 percent).

Prior to the HARRISON NARCOTICS ACT of 1914, the typical OPIATE addict in the United States was a white, middle-aged, middle-class housewife who had become addicted to medically prescribed drugs or nonprescription PATENT MEDICINES. Following criminalization of most opiate use through the Harrison Act and subsequent legislation and court interpretations, overall levels of opiate use declined dramatically. When HEROIN addiction reemerged as a social problem in the 1950s and 1960s, the typical opiate addict was a nonwhite urban male from a lower socioeconomic class. Although the VIETNAM war exposed a broader spectrum of young American men to heroin use, and although many servicemen tried opiates and even became addicted in Vietnam, most were able to discontinue use when they returned to the United States.

In the 1970s and 1980s, heroin use decreased and became quite rare in the United States. In 1993, only about one in 1,000 Americans aged twelve and older reported use of heroin in the past year, and the majority of users were men. An increase in drug seizures, arrests, and heroin-related emergency room episodes in the early 1990s led to assertions that heroin was making a comeback and that women would be especially vulnerable to addiction. Although these trends merited watching, such speculation was premature, given current evidence.

MEDICAL DRUG USE

In the 1970s feminist scholars drew attention to possible overmedication of women with PSYCHOACTIVE DRUGS. These early critiques derived from content analyses of sex-stereotyped advertisements in medical publications. Most of the ads depicted woman patients, and survey research on representative populations confirmed that women were using more prescription psychoactive drugs than were men.

Critics of these patterns are concerned that drugs are being used beyond traditional medical psychiatric concepts of disease. For example, medical ads suggested prescribing TRANQUILIZERS and ANTIDEPRESSANTS to alleviate normal life transitions, such as menopause, starting college, or a woman's adult children moving out. It has been suggested that prescribing psychoactive drugs is a subtle form of social control that diffuses or channels women's discontent with limiting and inequitable sex roles.

Some of the prescription psychoactives have dangerous side effects and a high potential for producing dependency. Further, since women also use more OVER-THE-COUNTER medications and women's alcohol problems are often undetected by physicians, use of prescription psychoactive drugs may make women especially vulnerable to adverse drug interactions. Alcohol in combination with other substances is the most frequent cause of emergency-room episodes in the DRUG ABUSE WARNING NETWORK (DAWN) system. Although women drink less and are less likely to use illicit drugs, they have equaled or exceeded men in drug-related emergency room episodes since the mid-1980s. This is because more women needed emergency treatment related to tranquilizer, sedative, and nonnarcotic analgesic use.

GENDER DIFFERENCES IN THE ETIOLOGY OF SUBSTANCE ABUSE

Studies of ADOLESCENTS generally find similar correlates of substance abuse among both boys and girls. The strongest predictor of adolescent alcohol, tobacco, and illicit-drug use is having friends who use alcohol, tobacco, and drugs. Other factors that predict substance abuse by boys and girls include

parental substance abuse, poor academic performance, and low commitment to educational pursuits.

Researchers, however, have identified some gender differences in the development of alcohol and drug problems. Relationship issues are particularly salient in the etiology of female substance abuse. For example, alcoholism in women is more strongly correlated with a family history of drinking problems than is alcoholism in men. Girls and women are likely to be introduced to alcohol or illicit drugs by a boyfriend or spouse, and female alcohol or drug dependence frequently develops in a relationship with an alcohol- or drug-dependent male partner.

Alcohol and drug abuse are more often associated with DEPRESSION in girls and women compared with males, but it is not clear whether depression is more likely to cause female substance abuse or is a more typical consequence of substance abuse among girls and women. Women in treatment for substance abuse are more likely than men to say their problem drinking or drug abuse developed after a life crisis or tragedy, such as the death of a family member. Also, a sizable proportion of women in treatment report histories of sexual abuse. Men are more likely to say their problem drinking or drug abuse developed out of social or recreational use.

Some believe these different attributions and recollections reflect genuine sex differences in the etiology of substance abuse. Others caution, however, that the greater stigma attached to female substance abuse may motivate women to develop an explanation for their problem drinking or drug use, and that personal crises and emotional difficulties serve as socially acceptable reasons.

The course of problem drinking and drug addiction varies by gender. Women entering treatment for alcoholism or drug abuse tend to have begun heavy drinking or drug use at a later age, on average, compared with men entering treatment. The term "telescoping" has been used to describe a more rapid progression from controlled alcohol or drug use to alcohol and drug dependency in women, compared with men.

GENDER DIFFERENCES IN THE CONSEQUENCES OF SUBSTANCE ABUSE

It is generally presumed that alcohol and drug abuse will produce more deleterious consequences among women than among men. This expectation is grounded both in biological differences and in social-role expectations.

From a biological standpoint, it is frequently noted that the lower ratio of water to total body weight in women causes them to metabolize alcohol and drugs differently than men. Even when body weight is controlled, given equivalent alcohol consumed, women pass more alcohol into the bloodstream and reach higher peak BLOOD ALCOHOL CONCENTRATIONS than men, in part because of differences in enzyme activity in the intestinal wall. Drugs such as marijuana that are deposited in body fat may be slower to clear in women than in men. Slow clearance rates create a potential for cumulative toxicity and adverse drug and alcohol interactions.

The behavioral telescoping of women's uncontrolled drinking and drug use is paralleled by a telescoping of some physical health consequences of alcohol and drug use. Alcoholic liver disease progresses more rapidly in women compared with men. Women also seem to be more prone to alcohol-related brain damage. They show physical brain abnormalities after a shorter drinking history and at lower peak alcohol consumption. Women also exhibit cognitive deficits on psychological tests of memory, speech, and perceptual accuracy with a shorter drinking history than that of men.

Women diagnosed as alcoholic have very high mortality rates relative to both the general population of women and to alcoholic men. A follow-up study of alcoholic women in St. Louis, found that, 11 years after treatment, they had lost an average of 15 years from their expected life span. Another study of 1,000 female and 4,000 male alcoholics in Sweden found the excess mortality was higher for the women (5.2 times the expected rate) than for the men (3 times the expected rate).

Deaths due to drugs other than alcohol and tobacco are relatively uncommon among women. Men are far more likely than women to die from drug use. The higher male death rates are largely explained by males' greater drug use rather than by sex differences in vulnerability among drug users. In 1990, medical examiners in twenty-seven U.S. metropolitan areas reported 5,830 deaths involving illicit and/ or legally obtained drugs. Of those who died from drug-related causes (e.g., OVERDOSE, accidental injury), 71 percent were male.

The HIV virus that causes AIDS is transmitted primarily via infected blood and semen. Sharing

needles and having sexual relations with intravenous (IV) drug users places both men and women at risk for contracting that incurable disease. Although most AIDS cases have resulted from transmission of HIV during intimate sexual contact between men, about 12,000 of the 43,000 people reported to have AIDS in 1990 were IV drug users. Most of these AIDS cases involving IV drug use were male. When women contract AIDS, the most common route of transmission is through their own IV drug use or sexual contact with a partner who is an IV drug user.

Women's reproductive function increases alcohol- and drug-related health risks to themselves and to their unborn children. Alcohol and drug abuse are associated with numerous disorders of the female reproductive system, including breast cancer, amenorrhea, failure to ovulate, atrophy of the ovaries, miscarriage, and early menopause. Men also experience reproductive and sexual difficulties as a result of alcohol and drug abuse, including impotence, low testosterone levels, testicular atrophy, breast enlargement, and diminished sexual interest.

Infants born to women who used alcohol, tobacco, or other drugs during PREGNANCY can experience numerous health problems, including low birth weight, major congenital malformations, neurological problems, mental retardation, and withdrawal symptoms. Although substance abuse at any time during pregnancy can cause birth defects, the very rapid cell division in the first weeks of embryonic development means the teratogenic effects of alcohol and drugs are generally greatest early in pregnancy, before a woman even realizes she is pregnant.

As the medical and social costs of prenatal alcohol and drug exposure become more apparent, so does public pressure for action. Many advocate termination of parental rights in cases where a newborn tests positive for drug or alcohol exposure. In some jurisdictions, mothers who used alcohol or drugs during pregnancy have been charged with child abuse or delivering a controlled substance to a minor. Critics of these policies charge that alcohol and drug screening will discourage substance-abusing women from obtaining necessary prenatal care. Legally, it may be difficult to establish criminal intent if substance abuse occurred early in an unintended and unrecognized pregnancy. Further, it is often difficult to causally disentangle alcohol or drug effects from other adverse conditions the mother may have experienced, such as poor nutrition, acute or chronic illness, and inadequate prenatal care. As currently practiced, prenatal drug-use detection procedures raise important questions of fairness. Hospitals and clinics serving largely poor and minority patient populations are more likely to detect prenatal substance abuse despite evidence that substance abuse occurs in all socioeconomic categories.

The tendency of female problem drinking and drug abuse to develop in a relationship with a substance-abusing male partner may shield women from some consequences of their substance abuse. For example, women alcoholics and addicts are less vulnerable to arrest if their partner procures drugs for the couple or drives when they are intoxicated. On the other hand, substance-abusing partners increase some other risks for alcohol- and drug-dependent women compared with men. Women with substance-abusing partners are vulnerable to domestic VIOLENCE. Also, a substance-abusing partner can be an impediment to women's seeking or complying with alcohol and drug treatment.

Despite women's biophysical vulnerability and the stigma associated with female alcohol and drug abuse, men are more likely than women to experience some problems related to heavy drinking and illicit drug use. Substance abuse is more strongly related to intrapsychic problems among women, and to problems in social functioning (employment difficulties, financial problems, unsafe driving, arrest) among men.

These gender differences may be related to sex-role differences in drinking and drug use. Male substance use is less socially controlled—occurring more often in recreational contexts, public places, and all-male settings—whereas female substance use is more likely to occur in the home, with a male partner, and under medical auspices. Sex roles may also allow males to exercise less personal control while drinking or using drugs. For example, male episodes of intoxication are more often associated with rapid ingestion, blackouts, and AGGRESSION.

GENDER AND SUBSTANCE ABUSE TREATMENT

Men outnumber women in drug and alcoholism treatment units. The 1991 National Drug and Alcoholism Treatment Unit Survey (NDATUS) found

213,681 women in some type of treatment, compared with 562,388 men (U.S. Department of Health and Human Services, 1992). Self-reports of treatment experience indicate a somewhat smaller sex difference. In the 1991 NHSDA, 1.8 percent of males aged twelve and older reported they were treated for substance abuse in the previous year, compared with 0.9 percent of females. The discrepancy may occur because women are less likely to report informal help, such as pastoral counseling or SELF-HELP groups, as TREATMENT.

Among alcoholics and addicts, a greater percentage of women are parents, and among substance-abusing parents, more women have child custody. Parenting considerations are a major barrier to women seeking substance-abuse treatment. Few residential treatment programs make provisions for pregnant women or mothers. Many women are unable to find caregivers for their children if they enter residential treatment, and fear permanent loss of custody if their children enter the foster care system.

Substance-abuse treatment programs have been geared more to the problems and needs of male clients. Some contend that only sex-segregated treatment can meet the unique needs of female clients. Even those advocating integrated programs acknowledge the need for greater attention to women's issues. In addition to parenting responsibilities, it is urged that treatment programs address women's histories of physical and sexual abuse, domestic violence, and relationships with substance-abusing partners. Burman (1994) also suggests that treatment programs for women should emphasize skills such as problem solving, assertiveness, self-advocacy, and LIFE SKILLS (including parenting and job seeking).

(SEE ALSO: *Addicted Babies; Complications: Endocrine and Reproductive Systems; Family Violence and Substance Abuse; Gender and Complications of Substance Abuse; Injecting Drug Users and HIV; Stress; Treatment; Vulnerability As Cause of Substance Abuse*)

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WOMEN'S CHRISTIAN TEMPERANCE UNION The nineteenth century was a time of drastic changes in the way many Americans viewed ALCOHOL. Early in the century, on average, U.S. citizens each consumed approximately 7 gallons of alcohol annually, the equivalent of about 2.5 ounces of pure alcohol daily. Concern that the United States would turn into a "nation of drunkards" led to the TEMPERANCE MOVEMENT of the early nineteenth century. This movement was loosely organized, consisting of the following diverse factions: (1) the neorepublicans, who were concerned with a host of problems that threatened the nation's security; (2) temperance societies, such as the Washingtonians, which served as the forerunners of modern-day self-help groups; and (3) physicians, who came to view habitual drunkenness as a disease. The goals of these groups varied; they ranged from helping habitual drunk-

ards, to discouraging the use of alcoholic beverages, to advocating the prohibition of alcoholic beverages.

This first wave of temperance activists met with some success—thirteen states passed prohibition laws by 1855, and average alcohol consumption rates dropped to less than 3 gallons per person annually—but this was stopped by the growing national concern surrounding the approaching Civil War. Although the role of women was nearly nonexistent during this first temperance movement, the early movement set the stage for the post-Civil War temperance movement, in which women played a crucial part.

The years following the Civil War were a somewhat chaotic time. With the onset of the urban-industrial revolution and the concomitant changes witnessed in postbellum America, many people sought what Lender and Martin (1982, p. 92) term "a search for order." This search found a home in various social-reform movements. Broad-based reform movements attacked a number of issues thought to threaten American society, including education reform, women's rights, and intemperance.

Aaron and Musto (1981) refer to this period as the second great prohibition wave. Many local temperance societies survived the Civil War, as did the American Temperance Union. In 1869, the National Prohibition party was formed. This group supported the abolition of alcohol and recruited women into the anti-liquor fight. The National Prohibition party advocated complete and unrestricted suffrage for women, and their enlistment of women into the temperance movement marked the first public involvement of women in the temperance effort.

The post-Civil War Progressive movement also influenced the issue of temperance. The Progressives believed that alcohol was "the enemy of industrial efficiency, a threat to the working of democratic government, the abettor of poverty and disease" (Bordin, 1981, p. xvi). To the Progressives, temperance reform was a means for confronting genuine social problems. Business leaders increasingly came to view the use of alcohol as incongruous with the new technological society that America was becoming. Alcohol symbolized wastefulness, rampant pluralism, individualism, and potential social disorder.

Frances Willard, the most influential leader of the temperance movement, served as president of the WCTU from 1879 until her death in 1898. (The Library of Congress)

At the same time, a growing number of physicians and temperance workers were coming to regard habitual drunkenness as a disease. At the core of the conception of this disease was its inherently progressive nature. Moderate drinking inevitably led to addiction, according to temperance workers, who proposed that as long as liquor was available to entice people to drink, and as long as moderate drinkers were around to act as models, then there would be drunkards. Increasingly, the blame for such addiction to alcohol was placed less on the individual and more on the society that permitted the sale of liquor and condoned drinking.

Some of the other factors that contributed to the milieu in which the women's temperance movement developed included better education for women, fewer children to care for, and the growing urbanization of America. As more household appliances became available and fewer women had to work around the clock at home or on the farm, they gained more leisure time. In addition, women came to be viewed as the protectors of the home—while,

increasingly, alcohol was seen as a threat to the security of the home. These factors, in combination with an increased middle class and better communications, set the stage for the first mass movement of women into U.S. politics.

DIO LEWIS AND THE WOMEN'S CRUSADE

Ironically, the direct origins of the movement in which women gained entry into the political arena can be traced back to a man—Dio Lewis. By the 1870s, Lewis, a trained homeopathic physician, had given up his practice of medicine to embark on a career as an educator and lecturer. In December 1873, Lewis's lecture circuit included the cities and small towns of Ohio and New York. In each of them, he agreed to deliver an additional lecture as well as his scheduled talk related to women's issues—the topic of his extra speech was the duty of Christian women in temperance work. As an immediate result of his temperance lectures, women in each of these cities organized and marched on saloons and liquor distributors. Praying and singing hymns, the women were able to convince many proprietors of alcohol establishments to pledge themselves to stop selling liquor.

This grass-roots movement, which came to be known as the Women's Crusade, quickly moved through Ohio and into neighboring states. Typically, the women of a community would call a meeting eliciting support from other women. After praying over their cause, they would organize their efforts, which included asking local ministers to preach on the topic of temperance. They also sought pledges of support from local political leaders. Finally, they would take to the streets, marching on distributors of liquor as they attempted to persuade them to cease their sales of alcohol.

HISTORY

By November 1874, the Women's Crusade had grown to the point where a national convention was called. Sixteen states were represented at this convention, out of which the Woman's Christian Temperance Union (WCTU) emerged. Annie Wittenmeyer was named the first president of the WCTU, and a platform of action was agreed upon including the principle of total abstinence for WCTU members. Other plans involved committing the organization to (1) strongly promote the introduction of temperance education in both Sunday schools and public schools; (2) continue to use the evangelical methods, mass meetings, and prayer services that had been successful during their crusades; (3) urge the newspapers to report on their activities; and (4) distribute literature informing people of their cause. Although these first program commitments were later expanded, the convention's first set of resolutions provided the direction the WCTU would initially follow.

1874–1879. Under the leadership of Annie Wittenmeyer, the primary commitment of the WCTU was to gospel temperance. Wittenmeyer contended that the WCTU program should stress personal reform of the drunkard and of the whole liquor industry by moral suasion. She supported conversion to Christianity, religious commitment, acknowledgment of sin, and willingness to abandon evil ways as methods to reform those who drank. She shied away from seeking out legislative mandates as the solution to intemperance, however, and intentionally distanced herself from the women's suffrage movement; she feared possible repercussions for women *in the home*, should they campaign for the right to vote.

Although Wittenmeyer was instrumental in the early success of the WCTU, Frances Willard is recognized as the most influential leader of the women's temperance movement. Willard was chosen to be secretary at the first convention. Her views were often more radical than those of Wittenmeyer, particularly regarding women's rights. In 1879, she was elected president of the WCTU and served in that role until her death in 1898. Twentieth-century observers of the women's temperance movement may be more familiar with the name of Carrie Nation, who was known for raiding saloons armed with axes and hatchets; however, militant individuals such as she constitute a small fringe element of the WCTU. During the latter part of the nineteenth century, the true spirit of the WCTU was embodied in the person of Frances Willard.

1879–1898. While Wittenmeyer's primary commitment was to moral suasion, from the beginning of Willard's involvement in the WCTU, women's rights commanded her deeper loyalty. This commitment would be seen in the direction the WCTU would take after 1879 (and was even evident while Willard served as secretary, as she subtly pushed for commitment to broader political

programs). In 1876, Willard had introduced the concept of "home protection" to the WCTU. Building on earlier arguments that made use of women's traditional roles within the home and the need to defend and protect those roles, Willard proposed extending women the right to vote on prohibition issues as a means of further protecting women. At the time of this proposal, the idea of granting women the right to vote based on their natural or political right to do so was *not* palatable to many people, women and men alike. By introducing the suffrage issue under the guise of home protection, Willard was able to introduce the right-to-vote issue within the WCTU with less opposition than if she had sought solely to address women's suffrage.

As president, Willard ran the WCTU as a "well-oiled reform machine." Emphasizing organization at the local level, Willard was able to establish the mass base necessary for effective action. By 1880 the WCTU easily outstripped other women's organizations in both size and importance. Bordin (1981) estimates that there were 1,200 local unions with 27,000 WCTU members by the time Willard became president.

Under the leadership of Willard, the WCTU continued many of the programs that were adopted while Wittenmeyer was president. A number of states passed compulsory temperance-education laws, in large part due to the influence of the WCTU. In addition, the omnipresent push for abstinence from alcoholic beverages continued to typify the movement's goals—as is evidenced by the brief alliance forged between the WCTU and the Prohibition party. The WCTU of the 1880s, however, also departed from its roots on a variety of issues. It evolved from a temperance praying society to an activist organization. Whereas Wittenmeyer sought for change through moral suasion, Willard saw the advantages of political solutions to both the problems caused by intemperance as well as the problems facing women. Willard supported federal constitutional prohibition as the most effective way to deal with alcohol abuse, and she endorsed the temperance ballot for women as the surest way to achieve prohibition.

By the mid-1880s, the WCTU had expanded to every U.S. state and territory, and its platform had undergone similar expansion. Willard adopted the slogan "Do Everything" to describe the focus of the WCTU under her guidance; initially, she had coined this phrase to depict the lengths to which she was willing to go to support the prohibition cause. By the late 1880s, however, she was committed to broader societal changes. Willard's strongest commitment remained to women's rights, and she argued as well for equal rights.

The membership of the WCTU in the early 1890s grew to an estimated 150,000 dues-paying members, with an additional 150,000 in affiliated groups. The WCTU had reached out to women of all social classes and minority groups. The growing influence of the WCTU was evident in the passage of several state prohibition laws in the 1880s, as well as in the growing support for a federal constitutional prohibition of liquor.

Although the number of women involved in the WCTU would continue to grow to approximately 1.5 million in the early twentieth century, as the nineteenth century drew to a close, the WCTU began losing its power and importance. Most notably, Willard became less visible in the years preceding her death. In her absence, conflicts arose among other leaders of the movement as to the organization's proper direction. In addition, as older leaders died or withdrew from active participation, fewer young women joined the WCTU to replace them.

1898–Present. As other organizations endorsing women's rights and/or prohibition were developed, membership in the WCTU slowly dwindled. Following Willard's death in 1898, the WCTU returned to a single-issue approach, focusing solely on prohibition. Although the ultimate goal of prohibition would eventually be achieved, it was not until the growth of the Anti-Saloon League (established 1896) that national prohibition would be realized. The Eighteenth Amendment to the U.S. Constitution was proposed and sent to the states December 18, 1917, and was ratified by three quarters of the states by January 16, 1919; it became effective January 16, 1920, establishing that the manufacture, sale, or transportation of intoxicating liquors, for beverage purposes, was prohibited. During the 1920s, it was clear that enforcement of the alcohol-beverage industry was almost impossible and that Americans would not give up drinking easily. The Repeal of Prohibition began as a movement that culminated in the Twenty-first Amendment to the U.S. Constitution; it was proposed and sent to the states February 20, 1933, and was ratified December 5, 1933.

Small groups of WCTU members can still be found in, for the most part, rural areas of the

United States. The organization is based in Evanston, Illinois, and listed about 100,000 members in 1990.

(SEE ALSO: *Alcohol; Disease Concept of Alcoholism and Drug Abuse; Treatment, History of*)

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WOOD ALCOHOL (METHANOL) Methanol (methyl alcohol, CH_3OH) is the simplest of the alcohols. It is the natural by-product of wood distillation—an older method of producing drinking ALCOHOL (ethanol). Chemically synthesized methanol is a common industrial solvent found in paint remover, cleansing agents, and antifreeze. It is used to denature the ethanol found in some of these solutions and thereby render them unfit for drinking.

Methanol ingestion is usually accidental, but some alcoholics resort to the desperate measure of consuming methanol when they cannot obtain the beverage ethanol. Persons working in poorly ventilated areas can suffer ill effects from inhaling methanol-containing products, and ingestion of methanol is considered a medical emergency. Methanol is metabolized to formaldehyde and formic acid by the same liver enzymes that break down ethanol (these are alcohol dehydrogenase and aldehyde dehydrogenase). The formaldehyde and formic acid are toxic metabolites responsible for the symptoms of methanol poisoning; these appear several hours or days after methanol ingestion. Blurred vision, leading to permanent bilateral blindness, is characteristic of methanol poisoning. The accumulation of formic acid results in severe metabolic acidosis, which can rapidly precipitate coma and death. Other symptoms of methanol toxicity include dizziness, headaches, cold clammy extremities, abdominal pain, vomiting, and severe back pain.

The treatment for methanol poisoning is sodium bicarbonate, given to reverse the acidosis. In more serious cases, dialysis may be required; in addition, ethanol is given intravenously because it competitively binds to alcohol dehydrogenase, thereby slowing the production of toxic metabolites and allowing unchanged methanol to be excreted in the urine.

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WORKPLACE, DRUGS IN THE *See* Employee Assistance Programs; Industry and Workplace, Drug Use in

WORLD HEALTH ORGANIZATION EXPERT COMMITTEE ON DRUG DEPENDENCE The World Health Organization (WHO) originated from a proposal at the first United Nations (U.N.) conference held in San Francisco in 1945 that "a specialized agency be created to deal with all matters related to health." This proposal resulted in a draft WHO constitution signed by sixty-one governments at an international health

conference held in New York City in 1946. The constitution was subsequently ratified by the twenty-six member states of the U.N. and came into force on April 7, 1948. The enormous proposed scope of WHO led to the early concept of "Expert Committees," and they have become an essential part of the machinery of the organization. Their function is to give technical advice to WHO. Members of these committees are "appointed by the Director-General, in accordance with regulations established by the Executive Board." The members are chosen for their "abilities and technical experience" with "due regard being paid to adequate geographical distribution." Reports of expert committees can only be published with the authorization of the World Health Assembly or the WHO executive board.

One of the first tasks of the U.N. and WHO was to pick up the regulatory work on addiction-producing drugs that had been initiated and carried out by the League of Nations. Thus, the Expert Committee on Habit-Forming Drugs was established in 1948 to provide expert technical advice to the U.N. Permanent Central Opium Board and Drug Supervisory Body and the Division of Narcotic Drugs. The first meeting of the expert committee was held January 24–29, 1949, at the Palais des Nations in Geneva, Switzerland, where it continued to meet until the WHO building was opened in 1961. The expert committee, in its report on the second session, felt that the expression "habit forming" was no longer appropriate and recommended that the designation of the committee be changed to Expert Committee on Drugs Liable to Produce Addiction. This change was adopted by the WHO executive board at its fifth session and remained until 1964, when it was altered to Expert Committee on Dependence Producing Drugs and finally in 1968 to its present designation, Expert Committee on Drug Dependence.

In its early years, the expert committee reported directly to the director-general of WHO through its own secretary. In 1965, it became part of the Division of Pharmacology and Toxicology. During much of the period from its inception to 1972, the Seretariat was in the hands of Dr. Hans Halbach. In 1977, the expert committee became part of the Division of Mental Health, under the direction of Dr. Inayat Khan, where it remained until 1990 when a new Programme on Substance Abuse was created.

The early meetings of the expert committee were mainly devoted to the opioids—including the natural products, semisynthetics, and synthetics. Notifications on specific compounds by individual nations were responded to and recommendations as to international control were communicated to the secretary-general of the U.N. The beginnings of often recurring discussions were initiated concerning definitions, methods for evaluating dependence liability in animals and humans, the need for accurate epidemiological data concerning the extent of abuse and public health problems associated with drugs in general and of specific compounds in particular. During this period, the expert committee had an important consultative role in the development of a new international drug-control treaty, which resulted in an international conference held in New York City in January 1961. From this Conference emerged the SINGLE CONVENTION ON NARCOTIC DRUGS, 1961. This convention was amended in 1972, again with strong input from the expert committee, and remains the current instrument for the international control of the opioids, cocaine, and cannabis (marijuana).

The committee's concern for the potential abuse of the newly emerging ataractics (tranquilizing drugs) began in the mid-1950s and was soon joined in the 1960s by discussions of the problems created by amphetamines, amphetamine-like drugs, and hallucinogens. The difficulties associated with controlling these new heterogeneous groups of drugs under the Single Convention of 1961 became apparent and, at its seventeenth meeting in 1969, the committee began discussions of a draft Protocol on Psychotropic Substances, developed by the U.N. Commission on Narcotic Drugs, which formalized a classification of psychotropic drugs developed by the expert committee at its sixteenth meeting in 1968. The increasingly serious international public-health problems created by these drugs led the United Nations to hold a conference for the Adoption of a Protocol on Psychotropic Substances held in Vienna in February 1971; this resulted in the Convention on Psychotropic Substances, 1971, which the United Nations finally ratified in 1976. One important feature of this convention is that it mandates a WHO assessment of a substance prior to control and states that WHO's "assessments shall be determinative as to medical and scientific matters." This mandate added great responsibility to the functional role of the expert committee.

Only two meetings of the expert committee were held between the adoption of the Convention on Psychotropic Substances in 1971 and its ratification in 1976. The nineteenth meeting in 1972 was mainly devoted to a review of the current status of the epidemiological study of drug dependence. This meeting was also the last attended by Dr. Nathan B. Eddy, before his death in 1973. Dr. Eddy, a giant in the study of drug abuse and dependency, was at all the first nineteen meetings and served as chairman or rapporteur for most of them. The twentieth meeting of the committee was essentially devoted to the topic of prevention and resulted in a thorough review of the literature and a series of conclusions and recommendations, which were of considerable influence in the future development of the field.

The twenty-first meeting of the committee was held in 1977. It was entirely concerned with consideration of the Convention on Psychotropic Substances, and how WHO would handle its obligations under the treaty. This included consideration of appropriate pharmacological studies in animals and humans, assessment of public-health and social problems, assessment of therapeutic usefulness, the problem of chemically generic extensions to the list of scheduled substances, and the decision-making process. The meeting resulted in a number of recommendations that were mainly concerned with international cooperation in the development and collection of the relevant data needed to make rational decisions on controlling substances under the convention.

The expert committee did not meet formally again until 1985. In the interim, however, a number of WHO ad-hoc committees met to consider various aspects of the implementation of the treaty. In 1980, an extensive review of the Assessment of Public Health and Social Problems Associated with the Use of Psychotropic Drugs was carried out. To assist WHO, the U.S. National Institute on Drug Abuse, in collaboration with the Committee on Problems of Drug Dependence, published a monograph on "Testing Drugs for Physical Dependence Potential and Abuse Liability," which updated a similar WHO report published a decade earlier. A particularly difficult section of the psychotropic convention concerns exempt preparations. This involves thousands of pharmaceutical products and how to handle them, and it has still not been completely resolved despite three meetings of WHO advisory groups in 1977, 1982, and 1984.

Initially, to handle WHO's necessary functions under the conventions, it was decided to use ad-hoc advisory groups rather than to call formal meetings of the expert committee. The first of these was held in 1978. In 1979, specific compounds were considered under both conventions and the recommendation was made that, in the future, compounds proposed for control under the psychotropic convention be considered by class. In 1980, nine anorectic substances (things that cause loss of appetite) were reviewed and recommendations as to control were forwarded. Discussions concerning KHAT and its active principals, cathine and cathinone, were begun and research was initiated by a widespread group of laboratories. In 1981, the mixed opioid AGONIST-ANTAGONIST drugs were reviewed, and in 1981 and 1982 the BENZODIAZEPINES as a class were reviewed and recommendations for control were sent to the U.N. Also during this period a more formal method for review emerged from discussions with the U.N. Commission on Narcotic Drugs and the WHO Executive Board. Detailed critical reviews of substances to be considered for control were developed and the Programme Planning Working Group was formed to review these and suggest future classes of compounds for review by the expert committee. Two additional ad hoc advisory committee meetings were held in 1983 and 1984 to consider a variety of individual compounds and exempt preparations.

The twenty-second meeting of the expert committee was held in Geneva in April 1985. The committee adopted the new procedures for review of substances recently approved by the WHO Executive Board. These guidelines mandated a procedural sequence and schedule for the review. WHO was to obtain detailed information on each substance from a wide variety of sources including individual experts, research groups (e.g., WHO Collaborating Centers), the pharmaceutical industry, and relevant publications. It should be noted that this was the first time that the pharmaceutical industry was included in deliberations concerning regulatory control of their products. The twenty-second meeting was held, primarily, to consider twenty-eight phenethylamines for control under the Psychotropic Convention. A large number of groups and individuals was involved in preparing the critical review of these substances. Many of the substances considered were recommended for control under various schedules of the Psychotropic

Convention. Some were not considered to need control, and no recommendation was made on these. Among the recommendations emerging from this meeting were requests for more and better data, particularly epidemiological, and more consideration of structure-activity relationships, isomeric state, and drug metabolism.

The twenty-third meeting in 1986 was nearly entirely devoted to the review of thirty-one BARBITURATES. A number of new factors were considered in the deliberations on this group of drugs. These included therapeutic indication (e.g., ultrashort-acting intravenous anesthetics, intermediate-acting sedative-hypnotics, and anticonvulsants), therapeutic usefulness, and demonstrable international public-health and social problems. Particular concern was expressed concerning PHENOBARBITAL, an inexpensive, effective antiepileptic widely used in developing countries, since it was felt by some that international control might lead to the use of more expensive and less safe medications. The committee also noted a lack of data on many compounds concerning dependence potential from either animals or controlled clinical studies and recommended that this be systematically collected by WHO prior to consideration for control.

The twenty-fourth meeting in 1987 discussed the control of seven nonbarbiturate sedative hypnotics. None of these were recommended for control. The committee also considered the marked increase in the illicit traffic in SECOBARBITAL and recommended that it be moved from Schedule III to Schedule II of the Psychotropic Convention. Finally, the committee recommended control of a number of fentanyl and MEPERIDINE analogs under the Single Convention.

The twenty-fifth meeting in 1988 considered the control of an additional four nonbarbiturate sedative-hypnotics including METHAQUALONE, which had been suggested for control in Schedule I of the Psychotropic Convention at the twenty-fourth expert committee meeting. Of these compounds, only methaqualone was recommended for control. The committee did not recommend rescheduling to Schecule I but urged the secretary-general of WHO that "every effort should be made to urge all countries whether or not they are signatories to the Convention on Psychotropic Substances, 1971, to stop producing methaqualone and to ban its import or export." The expert committee also revisited the opioid agonist-antagonist analgesics and recommended that BUPRENORPHINE and pentazocine be controlled under Schedule III of the Psychotropic Convention. This was a significant departure and was the first time that compounds with some opioid-like properties were considered for control under this convention rather than the Single Convention, 1961. A number of other compounds were considered for control, the most interesting being propylhexadrene. This substance was the first to be considered for decontrol under the Psychotropic Convention. The committee recommended that additional epidemiological data be collected and the substance reviewed again in two years. This was done in 1990, and a recommendation to remove propylhexadrene from control was forwarded to the U.N. secretary-general.

The twenty-sixth meeting of the committee in 1989 considered four additional uncontrolled benzodiazepines and recommended control for only one. The remainder were held over for the twenty-seventh meeting, in which the 33 benzodiazepines already under control were to be reviewed. This meeting also recommended the control of a number of "DESIGNER DRUGS," including analogs of fentanyl, tenamfetamine (MDA), and aminorex. Also considered was the notification from the government of the United States to transfer *delta*-9-tetrahydrocannabinol, the active principle of MARIJUANA, from Schedule I to Schedule II of the Convention on Psychotropic Substances. The committee so recommended, with the exception of two members who felt the decision should be deferred for additional data concerning therapeutic usefulness.

The twenty-seventh and last meeting to date of the expert committee was held in 1990 and was essentially devoted to the scheduling of the benzodiazepines as a class. Of particular interest was the conclusion that differential scheduling of the benzodiazepines was possible. Thus, the committee recommended that of the thirty-three substances currently under control, nineteen were appropriately controlled under Schedule IV. Thirteen of the substances had moderate to high therapeutic usefulness and few or no reports of abuse or illicit activity, and the committee declared that WHO should "monitor these compounds to amass enough data to determine whether or not they should be placed under critical review to consider descheduling." Two compounds, diazepam and

flunitrazepam, "showed a continuing higher incidence of abuse and association with illicit activity." It was recommended that WHO keep these compounds under surveillance "to determine whether or not they merit being placed under critical review to consider appropriate scheduling."

As a result of structural changes within WHO and the creation of the new Programme on Substance Abuse, it is clear that in the future the expert committee will change its focus from reviewing substances for control under the international conventions to a broader consideration of the issues of prevention and reduction of demand.

(SEE ALSO: *Abuse Liability of Drugs: Testing in Humans*)

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LOUIS HARRIS

XTC *See* Slang and Jargon

Y

YIPPIES When large numbers of individuals with shared values engage in certain patterns of drug use, the political consequences can be serious. The Yippies of the late 1960s and early 1970s provide such an example.

Rather than quietly retreating from society as part of the baby-boom's countercultural (hippie) revolution, the Yippies shocked those with conventional values in the United States through spectacular media events. Thousands of young Americans shared the antimaterialistic values of Yippie leaders Abbie Hoffman and Jerry Rubin. In 1967, Hoffman dumped dollar bills from the visitors' gallery onto the floor of the New York Stock Exchange. In 1968, another protest event was staged—the Chicago Yippie Convention—timed to coincide with the Chicago Democratic Presidential Convention and considered an opportunity to protest the VIETNAM War.

Yippies challenged the establishment with a Festival of Life and invited drug-using hippies to attend; it included LSD seminars, rock shows, light shows, films, marches, love-ins, put-ons, guerrilla theater, and bizarre stunts—such as nominating a pig named Pigasus for president. The protest escalated into a confrontation with Chicago authorities; the mayor called out the police; and, in a rioting atmosphere, Yippies were beaten and imprisoned; the presidential convention was disrupted; Yippie leaders were tried in a case that became known as the Chicago Seven; and the Democrats lost the 1968 election.

During that time, a team of scientists surveyed the drug-use activity of 432 Yippies (Hughes et al. 1969). These showed a strong preference for hallucinogenic substances. Weekly MARIJUANA use was reported by 79 percent, HASHISH by 40 percent, LYSERGIC ACID DIETHYLAMIDE (LSD) by 29 percent, MESCALINE by 10 percent, PSILOCYBIN by 5 percent, and PEYOTE by 3 percent. Weekly use of nonhallucinogens was low—ALCOHOL 34 percent, COCAINE 4 percent, and HEROIN 3 percent.

It may be too simplistic to attribute the 1968 political events to marijuana and LSD. Yet we do know that certain chemicals help free users from conventional values and ways of perceiving reality. Researchers need to further examine this issue in future outbreaks of antiestablishment protest.

(SEE ALSO: *Epidemics of Drug Abuse; Hallucinogens*)

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PATRICK H. HUGHES

YOUTH AND SUBSTANCE ABUSE *See* Adolescents and Drugs; Gangs and Drugs; Prevention Programs; Treatment

YUPPIES *See* Slang and Jargon

Z

ZERO TOLERANCE The phrase has come to be associated with government and private employer policies that mandate predetermined consequences or punishments for specific offenses. However, the phrase first became associated with U.S. drug interdiction during the 1980s and 1990s. Most public schools now have zero tolerance policies for firearms, weapons other than firearms, alcohol, drugs, and tobacco. Zero tolerance policies generally are rigid and can produce results that appear out of proportion to the improper behavior. Nevertheless, the courts have endorsed drug-testing programs that allow employers to enforce zero tolerance policies.

ZERO TOLERANCE AND U.S. DRUG CONTROL POLICY

Zero tolerance was a federal drug policy initiated during the War on Drugs campaign of the Reagan and Bush administrations (1981–1993). Under this policy, which was designed to prohibit the transfer of illicit drugs across U.S. borders, no possession, import, or exportation of illicit drugs was tolerable, and possession of any measurable amount of illicit drugs was subject to all available civil and criminal sanctions. Zero tolerance was an example of a criminal justice approach to drug control. Under such an approach, the control of drugs rests within the domain of the criminal justice system, and the use of drugs is regarded as a criminal act, with legal sanction as the consequence.

Zero tolerance is a "user-focused" strategy of drug control, according to which law-enforcement agents target users of illicit drugs as opposed to dealers or transporters. The rationale for this approach is that the users of illicit substances create the demand for drugs and constitute the root cause of the drug problem. If, therefore, demand for drugs can be curbed by exacting harsh penalties on users, the supply of drugs into the country will slow.

The zero-tolerance policy was initiated by the U.S. CUSTOMS SERVICE, in conjunction with the U.S. Attorney's office in San Diego, California, as part of an effort to stop drug trafficking across the U.S.-Mexican border. Individuals in possession of illicit drugs were arrested and charged with both a misdemeanor and a felony offense. Customs Service officials believed the policy to be successful at reducing the flow of drugs across the border and recommended that it be implemented nationwide. Subsequently, the National Drug Policy Board, in conjunction with the White House Conference on a Drug-Free America had all federal drug-enforcement agencies implement zero tolerance in 1988, at all U.S. points of entry (United States Congress, 1988).

The policy did not involve enacting new laws or regulations; it only entailed instituting strict interpretation and enforcement of existing laws. In practice, it meant that any type of vehicle—

including bicycles, transfer trucks, and yachts—would be confiscated and the passengers arrested upon the discovery of any measurable amount of illicit drugs. The U.S. Coast Guard and the U.S. Customs Service began to crack down on all cases of drug possession on the water and at all borders. If, during the course of their regular patrols and inspections, Coast Guard personnel boarded a vessel and found one marijuana cigarette, or even the remnants of a marijuana cigarette, they arrested the individual and seized the boat. Before this policy was instituted, the Coast Guard had either looked the other way or issued fines when "personal-use" quantities of illicit substances were discovered (United States Congress, 1988).

Zero tolerance was criticized because federal agencies expended substantial resources to identify individual drug users instead of concentrating their resources on halting the influx of major quantities of drugs into the country for street sale. The policy of seizing boats upon the discovery of trace amounts of drugs was also controversial. Some believed the policy to be an unfair and unusually harsh punishment; seizing a commercial boat that was the sole source of income for an individual or family was denounced as being too severe a penalty for possession of "one marijuana cigarette." There were some highly publicized cases of commercial fishing boats being seized on scant evidence that the boat owner was responsible for the illicit drugs found.

ZERO TOLERANCE AS A GENERAL POLICY

The term *zero tolerance* has a broader application than the Reagan-Bush drug interdiction approach. Zero tolerance describes a perspective on drug use according to which it is maintained that the use of any amount of illicit drugs is harmful to the individual and society and that the goal of drug policy should be to prohibit any and all illicit drug use. According to the contrasting viewpoint, the simple use of drugs is distinguishable from problem drug use and although absence of all drug use is desirable, the resources of government would be used more efficiently if they targeted individuals who demonstrated problem use or if they addressed problems related to or caused by illicit drug use.

Drug testing in the workplace typically uses a zero tolerance approach. In the late 1970s, employees challenged these policies in the courts. However, the U.S. Supreme Court, in *New York City Transit Authority v. Beazer*, 440 U.S. 568, 99 S.Ct. 1355, 59 L.Ed.2d 587 (1979), ruled that a city agency's blanket exclusion of persons who regularly use narcotic drugs did not violate the Equal Protection Clause of the Fourteenth Amendment. This zero tolerance decision subsequently has been extended to various employment situations. By 2000, many employers routinely required a drug test as part of the employee hiring process. Applicants who failed the test usually are not hired because employers use a zero tolerance drug policy.

Zero tolerance policies have become a standard part of U.S. public schools. With the rash of school shootings in the 1990s, zero tolerance weapons policies have dominated the news, yet zero tolerance drug polices are also part of school rules. Zero tolerance has widespread public support, as it mandates high standards and signifies a "get tough" attitude toward drugs and school violence. Nevertheless, there are many critics of zero tolerance polices. Critics analogize zero tolerance to mandatory minimum sentencing in the criminal justice system. Under both schemes there are no exceptions made for individual circumstances; this results in punishments that appear excessive, such as a student suspension for bringing aspirin to school without permission.

(SEE ALSO: *Drug Interdiction; Operation Intercept; U.S. Government: The Organization of U.S. Drug Policy*)

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Appendix
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APPENDIX I

Poison Control Centers for Drug Overdoses (ODs) and Emergencies

INTRODUCTION

The list of Poison Control Centers has been compiled from information furnished by the American Association of Poison Control Centers (AAPCC), 3201 New Mexico Avenue NW, Suite 310, Washington, DC, 20016.

The list includes facilities that provide information on the treatment and prevention of accidents involving ingestion of poisonous (toxic) and potentially poisonous substances, including alcohol and drugs. Alcohol and drug overdoses (ODs) often cause "blackouts," coma, and death. A call to poison control in your area, with symptoms described, can mean immediate help, first-aid suggestions, and the swift response of emergency medical services (EMS). If in doubt, call Poison Control before 911.

Household products, garden supplies, and hobby materials may be inhaled or swallowed, accidentally or on purpose. Prescription drugs and over-the-counter (OTC) medications are sometimes taken in larger doses than may be safe. "Kiddie dope"— drugs sold legally without prescription, by mail and in shops, which mimic the effects of amphetamines (speed) may be taken in great quantities; these products usually contain a combination of caffeine, phenylpropanolamine, phenylephrine, ephedrine, or pseudoephedrine. Kiddie dope is taken by youngsters who expect increased energy, weight loss, and a pleasant high— but handfuls of such pills often lead to seizures, heart failure, and cerebral bleeding (stroke). Poison control units are available to answer your questions and help out in any suspected poisoning emergency— and any chemical substance can be toxic if inhaled or taken in inappropriate quantities.

The list below is the most current provided by the AAPCC for 2000. Updates are available online at http://www.usmedicine.com/poison.html.

CERTIFIED REGIONAL POISON CENTERS

ALABAMA

Alabama Poison Center
408-A Paul Bryant Drive
Tuscaloosa, AL 35401
(205) 345-0600
(800) 282-0880 (AL only)

Regional Poison Control Center The Children's Hospital of Alabama
1600 7th Ave. South
Birmingham, AL 35233-1711
(205) 939-9201
(205) 933-4060
(800) 292-6678 (AL only)

ARIZONA

Arizona Poison and Drug Information Center
Arizona Health Sciences Center,
Rm. #3204-K
1501 North Campbell Ave.
Tucson, AZ 85724
(602) 626-6016
(800) 362-0101 (AZ only)

Samaritan Regional Poison Center
Good Samaritan Regional Medical Center
Ancillary-1
1111 E. McDowell Road
Phoenix, AZ 85006
(602) 253-3334

CALIFORNIA

Central California Regional Poison Control Center
Valley Children's Hospital
3151 North Millbrook, IN31
Fresno, CA 93703
(209) 445-1222
(800) 346-5922 (Central CA only)

San Diego Regional Poison Center
UCSD Medical Center
200 West Arbor Drive
San Diego, CA 92103-8925
(619) 543-6000
(800) 876-4766 (in 619 area code only)

San Francisco Bay Regional Poison Control Center
San Francisco General Hospital
1001 Potrero Ave., Building 80, Room 230
San Francisco, CA 94110
(800) 523-2222

Santa Clara Valley Regional Poison Center
Valley Health Center, Suite 310
750 South Bascom Ave.
San Jose, CA 95128
(408) 885-6000
(800) 662-9886 (CA only)

University of California, Davis, Medical Center Regional Poison Control Center
2315 Stockton Blvd.
Sacramento, CA 95817
(916) 734-3692
(800) 662-9886 (Northern California only)

COLORADO

Rocky Mountain Poison and Drug Center
645 Bannock St.
Denver, CO 80204
(303) 629-1123

DISTRICT OF COLUMBIA

National Capital Poison Center
3201 New Mexico Avenue, NW, Suite 310
Washington, DC 20016
(202) 625-3333

FLORIDA

Florida Poison Information Center–Jacksonville
University Medical Center
University of Florida Health Science Center–Jacksonville
655 West 8th Street
Jacksonville, FL 32209
(904) 549-4480
(800) 282-3171 (FL only)

The Florida Poison Information Center and Toxicology Resource Center
Tampa General Hospital
Post Office Box 1289
Tampa, FL 33601
(813) 253-4444
(800) 282-3171 (Florida)

GEORGIA

Georgia Poison Center
Hughes Spalding Children's Hospital
Grady Health Systems
80 Butler Street SE
P.O. Box 26066
Atlanta, GA 30335-3801
(800) 282-5846 (GA only)
(404) 616-9000

INDIANA

Indiana Poison Center
Methodist Hospital of Indiana
1701 N. Senate Boulevard
P.O. Box 1367
Indianapolis, IN 46206-1367
(317) 929-2323
(800) 382-9097 (IN only)

KENTUCKY

Kentucky Regional Poison Center of Kosair Children's Hospital
P.O. Box 35070
Louisville, KY 40232-5070
(502) 629-7275
(800) 722-5725 (KY only)

MARYLAND

Maryland Poison Center
20 N. Pine St.
Baltimore, MD 21201
(410) 528-7701
(800) 492-2414 (MD only)

National Capital Poison Center (DC suburbs only)
3201 New Mexico Avenue, NW, Suite 310
Washington, DC 20016
(202) 625-3333

MASSACHUSETTS

Massachusetts Poison Control System
300 Longwood Ave.
Boston, MA 02115
(617) 232-2120
(800) 682-9211

MICHIGAN

Poison Control Center
Children's Hospital of Michigan
3901 Beaubien Blvd.
Detroit, MI 48201
(313) 745-5711

MINNESOTA

Hennepin Regional Poison Center
Hennepin County Medical Center
701 Park Ave.
Minneapolis, MN 55415
(612) 347-3141

Minnesota Regional Poison Center
St. Paul-Ramsey Medical Center
640 Jackson Street
St. Paul, MN 55101
(612) 221-2113

MISSOURI

Cardinal Glennon Children's Hospital Regional Poison Center
1465 S. Grand Blvd.
St. Louis, MO 63104
(314) 772-5200
(800) 366-8888

MONTANA

Rocky Mountain Poison and Drug Center
645 Bannock St.
Denver, CO 80204
(303) 629-1123

NEBRASKA

The Poison Center
8301 Dodge St.
Omaha, NE 68114
(402) 390-5555 (Omaha)
(800) 955-9119 (NE & WY)

NEW JERSEY

New Jersey Poison Information and Education System
201 Lyons Ave.
Newark, NJ 07112
(800) 962-1253

NEW MEXICO

New Mexico Poison and Drug Information Center
University of New Mexico
Albuquerque, NM 87131-2551
(505) 843-2551
(800) 432-6866 (NM only)

NEW YORK

Hudson Valley Regional Poison Center
Phelps Memorial Hospital Center
701 North Broadway
North Tarrytown, NY 10591
(914) 366-3030
(800) 336-6997

Long Island Regional Poison Control Center
Winthrop University Hospital
259 First Street
Mineola, NY 11501
(516) 542-2323
(516) 542-3813

New York City Poison Control Center
N.Y.C. Department of Health
455 First Ave., Room 123
New York, NY 10016
(212) POI-SONS
(212) 340-4494

NORTH CAROLINA

Carolinas Poison Center
1000 Blythe Boulevard
PO Box 32861
Charlotte, NC 28232-2861
(704) 355-4000
(800) 84TOXIN

OHIO

Central Ohio Poison Center
700 Children's Drive
Columbus, OH 43205-2696
(614) 228-1323
(614) 461-2012
(800) 682-7625

Cincinnati Drug & Poison Information Center and Regional Poison Control Center
231 Bethesda Avenue, M.L. 144
Cincinnati, OH 45267-0144
(513) 558-5111
(800) 872-5111 (OH only)

OREGON

Oregon Poison Center
Oregon Health Sciences University
3181 S.W. Sam Jackson Park Road
Portland, OR 97201
(503) 494-8968
(800) 452-7165 (OR only)

PENNSYLVANIA

Central Pennsylvania Poison Center
University Hospital
Milton S. Hershey Medical Center
Hershey, PA 17033
(800) 521-6110

The Poison Control Center serving the greater Philadelphia metropolitan area
One Children's Center
Philadelphia, PA 19104-2100
(215) 386-2100

Pittsburgh Poison Center
3705 Fifth Avenue
Pittsburgh, PA 15213
(412) 681-6669

RHODE ISLAND

Rhode Island Poison Center
593 Eddy St.
Providence, RI 02903
(401) 277-5727

TEXAS

North Texas Poison Center
5201 Harry Hines Blvd.
P.O. Box 35926
Dallas, TX 75235
(214) 590-5000

Southeast Texas Poison Center
University of Texas Medical Branch
Galveston, TX 77550-2780
(409) 765-1420 (Galveston)
(713) 654-1701 (Houston)

UTAH

Utah Poison Control Center
410 Chipeta Way, Suite 230
Salt Lake City, UT 84108
(801) 581-2151
(800) 456-7707 (UT only)

VIRGINIA

Blue Ridge Poison Center
Box 67, Blue Ridge Hospital
Charlottesville, VA 22901
(804) 924-5543
(800) 451-1428

National Capital Poison Center (Northern VA only)
3201 New Mexico Avenue, NW, Suite 310
Washington, DC 20016
(202) 625-3333

WEST VIRGINIA

West Virginia Poison Center
3110 MacCorkle Ave. S.E.
Charleston, WV 25304
(304) 348-4211
(800) 642-3625 (WV only)

WYOMING

The Poison Center
8301 Dodge St.
Omaha, NE 68114
(402) 390-5555 (Omaha)
(800) 955-9119 (NE & WY)

APPENDIX II

U.S. and State Government Drug Resources Directory

INTRODUCTION

This is a guide to state agencies that address substance abuse concerns. It begins with listings of federal agencies and goes on to state and local listings. The listings were originally compiled for a report by the U.S. Department of Justice, Bureau of Justice Statistics. Six groupings are presented here: Federal Information Centers and Clearinghouses; Other Federal Sources; Drug Abuse Resistance Education (DARE) Regional Training Centers; National Prevention Network; Treatment Alternatives to Street Crime (TASC) Programs; and the State Listings.

In Appendix III, which follows, an extensive State-by-State Directory is presented for drug abuse and alcoholism treatment and prevention programs, both public and private.

FEDERAL INFORMATION CENTERS AND CLEARINGHOUSES

CRIMINAL JUSTICE

Drugs & Crime Clearinghouse
1600 Research Boulevard
Rockville, MD 20850
(800) 666-3332
Sponsored by: Office of National Drug Control Policy

National Criminal Justice Reference Service
PO Box 6000
Rockville, MD 20849-6000
(800) 851-3420
http://www.ncjrs.org
Sponsored by: National Institute of Justice

Bureau of Justice Assistance Clearinghouse
PO Box 6000
Rockville, MD 20849-6000
(800) 688-4BJA/4252
http://bjavic.aspensys.com
Sponsored by: Bureau of Justice Assistance

Juvenile Justice Clearinghouse
PO Box 6000
Rockville, MD 20849-6000
(800) 638-8736
http://www.ojjpd.ncrs.org
Sponsored by: Office of Juvenile Justice and Delinquency Prevention

Justice Statistics Clearinghouse
PO Box 179
Annapolis Junction, MD 20701-0179
(800) 732-3277
Sponsored by: Bureau of Justice Statistics

Justice Technology Information Network
PO Box 1160
Rockville, MD 20849-1160
(800) 248-2742
http://www.nlect.org
Sponsored by: National Institute of Justice

National Victims Resource Center
PO Box 6000
Rockville, MD 20849-6000
(800) 627-6872
Sponsored by: Office for Victims of Crime

National Institute of Corrections Information Center
1860 Industrial Circle
Suite A
Longmont, CO 80501
(800) 877-1461
http://www.nicic.org
Sponsored by: National Institute of Corrections

HEALTH

National Clearinghouse for Alcohol and Drug Information
PO Box 2345
Rockville, MD 20847-2345
(800) 729-6686
http://www.health.org
Sponsored by: Office of Substance Abuse Prevention

National Drug Information and Treatment Routing Service
107 Lincoln Street
Worcester, MA 01605
(800) 662-HELP
Sponsored by: National Institute on Drug Abuse

Drug-Free Workplace Helpline
5600 Fishers Lane
Rockville, MD 20857
(800) 843-4971
Sponsored by: National Institute on Drug Abuse

National AIDS Information Clearinghouse
PO Box 6003
Rockville, MD 20850
(800) 458-5231
Sponsored by: Centers for Disease Control

PUBLIC HOUSING

Drug Information and Strategy Clearinghouse
PO Box 6424
Rockville, MD 20850
(800) 578-3472
Sponsored by: Housing and Urban Development

EDUCATION

ACCESS ERIC
2277 Research Boulevard
Rockville, MD 20850
(800) LET-ERIC
http://www.accesseric.org
Sponsored by: United States Department of Education

OTHER FEDERAL SOURCES

Executive Office of the President
Office of National Drug Control Policy
Executive Office of the President
Washington, DC 20503
(202) 395-6700
http://www.whitehousedrugpolicy.gov

U.S. Department of Justice
Office of Justice Programs
810 Seventh Street, NW
Washington, DC 20531
(202) 307-0703
http://www.ojp.usdoj.gov

Bureau of Justice Assistance
810 Seventh Street, NW
Washington, DC 20531
(202) 616-6500
http://www.ojp.usdoj.gov/bja

Bureau of Justice Statistics
810 Seventh Street, NW
Washington, DC 20531
(202) 307-0765
http://www.ojp.usdoj.gov/bjs

National Institute of Justice
810 Seventh Street, NW
Washington, DC 20531
(202) 307-2942
http://www.ojp.usdoj.gov/nij

Office of Juvenile Justice and Delinquency Prevention
810 Seventh Street, NW
Washington, DC 20531
(202) 307-5911

Office for Victims of Crime
810 Seventh Street
Washington, DC 20531
(202) 307-5983
http://www.ojp.usdoj.gov/ovc

Executive Office for United States Attorneys
950 Pennsylvania Avenue, NW
Room 2261
Washington, DC 20530-0001
(2O2) 514-1020
http://www.usdoj.gov/eousa

Drug Enforcement Administration
700 Army Navy Drive
Arlington, VA 22202
(202) 307-7977 Public Affairs
(202) 307-8932 Library
http://www.usdoj.gov/dea

U.S. Courts
Administrative Office of the United States Courts
One Columbus Circle, NE
Washington, DC 20544
(202) 273-0107
http://www.uscourts.gov

Federal Judicial Center
One Columbus Circle, NE
Washington, DC 20002-8003
(202) 633-6011
http://www.fjc.gov

U.S. Department of Health and Human Services
National Institute on Drug Abuse
6001 Executive Boulevard
Bethesda, MD 20892-9561
(301) 443-1124
http://www.nida.nih.gov

Office of Substance Abuse Prevention
Rockwall II Building
5600 Fishers Lane
Rockville, MD 20857
(301) 443-0365
http://www.samhsa.gov/csap

U.S. Department of State
Bureau of International Narcotics and Law Enforcement Affairs
Room 7334
2201 C Street, NW
Washington, DC 20520
(202) 647-0453
http://www.state.gov/www/global/narcotics_law

U.S. Department of the Treasury
Bureau of Alcohol, Tobacco, and Firearms
Office of Liaison and Public Information
Room 8290
650 Massachusetts Avenue, NW
Washington, DC 20226
(202) 927-7777
http://www.atf.treas.gov

United States Customs Service
1300 Pennsylvania Avenue, NW
Washington, DC 20229
(202) 927-1350
http://www.customs.treas.gov

U.S. Department of Transportation
United States Coast Guard
2100 2nd Street, SW
Washington, DC 20593
(202) 267-2229
http://www.uscg.mil

U.S. Department of Education
Safe and Drug-Free Schools
400 Maryland Avenue, SW
Washington, DC 20202-0498
(202) 260-3954
http://www.ed.gov/offices/OESE/SDFS

U.S. Department of Housing and Urban Development
Community Safety and Conservation Division
451 7th Street, SW
Room 4112
Washington, DC 20410
(202) 401-0398
http://www.hud.gov/pih/programs/ph/de/cscd

DRUG ABUSE RESISTANCE EDUCATION (DARE) REGIONAL TRAINING CENTERS

North Carolina State Bureau of Investigation
3320 Old Garner Road
P.O. Box 29500
Raleigh, NC 27626-0500
(919) 662-4500 ext. 277

Missouri State Highway Patrol
P.O. Box 568
Jefferson City, MO 65101-0568
(573) 526-6174

Virginia State Police
7700 Midlothian Turnpike
Box 27472
Richmond, VA 23261-7472
(804) 674-2238

Arizona Department of Public Safety
3110 North 19th Avenue
Suite 290
Phoenix, AZ 85015
(602) 223-2544

Los Angeles Police Department
3353 San Fernando Road
Los Angeles, CA 90065
(213) 485-4856

NATIONAL PREVENTION NETWORK

State Substance Abuse Coordinator
Alabama Department of Substance Abuse Services
PO Box 3710
Montgomery, AL 36193
(334) 242-3961

Executive Director
Alaska Council on Prevention of Alcohol and Drug Abuse
3333 Denali Street, Suite 201
Anchorage, AK 99503
(907) 258-6021

Manager, Office of Prevention
Department of Health Services
Behavioral Health Services
2122 East Highland
Phoenix, AZ 85016
602) 381-8996

Director, Division of Prevention
Arkansas Bureau of Alcohol and Drug Abuse Prevention
Freeway Medical Center, Suite 907
5800 West 10th Street
Little Rock, AR 72204
(501) 280-4511

Deputy Director
Department of Alcohol and Drug Programs
1700 K Street, Second Floor
Sacramento, CA 95814-4037
(916) 323-0633

Prevention Specialist
Program Services
Alcohol and Drug Abuse Division
4300 Cherry Creek Drive South
Denver, CO 80220-1530
(303) 692-2952

Prevention Director
Department of Mental Health and Addiction Services
410 Capitol Avenue, MS 14PIT
Hartford, CT 06134
(860) 418-6827

Director of Training
Delaware Division of Alcoholism, Drug Abuse and Mental Health
1901 North DuPont Highway
New Castle, DE 19720
(302) 577-4980

Administrator
Office of Prevention
Barley Mill Plaza, Building 18
4417 Lancaster Pike
Wilmington, DE 19805
(302) 892-4507

Chief
Office of Prevention and Youth Services
1300 First Street, NE
Washington, DC 20018
(202) 727-0092

State Prevention Coordinator
Alcohol, Drug Abuse and Mental Health Program Office
Office Building 5, Room 304B-1
1317 Winewood Boulevard
Tallahassee, FL 32399-0700
(904) 487-2920 ext. 105

Acting Prevention Unit Director
Substance Abuse Services
2 Peachtree Street NW, Suite 4-320
Atlanta, GA 30303-3171
(404) 657-2136

Prevention Coordinator
Alcohol and Drug Abuse Division
PO Box 3378
Honolulu, HI 96801
(808) 586-4007

Idaho Bureau of Substance Abuse and Social Services
450 West State Street
Boise, ID 83720
(208) 334-5700

Administrator for Prevention
Department of Alcoholism and Substance Abuse
100 West Randolph Street, Suite 5-600
Chicago, IL 60601
(312) 814-6355

Division of Mental Health
402 West Washington Street, Room W353
Indianapolis, IN 46204-2739
(317) 232-7924

Prevention Consultant
Department of Public Health
Lucas State Office Building
321 East 12th Street
Des Moines, IA 50319-0075
(515) 281-4404

Public Service Executive
Alcohol and Drug Abuse Services
Biddle Building
300 Southwest Oakley
Topeka, KS 66606
(913) 296-0511

Manager
Prevention and Training Branch
Division of Substance Abuse
275 East Main Street
1R Health Services Building
Frankfort, KY 40621
(502) 564-2880

Division of Alcohol and Drug Abuse
1201 Capitol Access Road
P.O. Box 3868
Baton Rouge, LA 70821-3868
(504) 342-9352

Office of Substance Abuse
Division of Prevention and Education
State House Station 159
Augusta, ME 04333
(207) 287-8908

Assistant Director
Alcohol and Drug Abuse
Prevention and Treatment Services
201 West Preston Street, Fourth Floor
Baltimore, MD 21201
(410) 225-6543

Director of Prevention Services
Division of Substance Abuse Services
150 Tremont Street
Boston, MA 02111
(617) 727-5141

Chief, Prevention Services
Michigan Department of Public Health
Center for Substance Abuse Services
3423 North Logan
P.O. Box 30295
Lansing, MI 48909
(517) 335-8843

Prevention Coordinator
Chemical Dependency Program Division
444 Lafayette Road
St. Paul, MN 55155-3823
(612) 296-4711

Program Coordinator
Division of Alcohol and Drug Abuse
1101 Robert E. Lee Building
239 North Lamar Street
Jackson, MS 39201
(601) 359-6216

Prevention Director
Division of Alcohol and Drug Abuse
1706 East Elm Street
PO Box 687
Jefferson City, MO 65102
(314) 751-7814

Prevention Coordinator
Division of Addictive and Mental Disorders
Department of Public Health and Human Services
1539 11th Avenue
Helena, MT 59620
(406) 444-1202

Prevention Coordinator
Division of Alcoholism and Drug Abuse
P.O. Box 94728
Lincoln, NE 68509
(402) 471-2851

Prevention Specialist
Bureau of Alcohol and Drug Abuse
1830 East Sahara Avenue, Suite 314
Las Vegas, NV 89104
(702) 486-8250

Director, Prevention, Training, and Education
Division of Alcoholism, Drug Abuse and Addiction Services
129 East Hanover Street, CN362
Trenton, NJ 08625
(609) 292-4414

Prevention Manager
Substance Abuse Bureau
1190 Saint Francis Drive
Santa Fe, New Mexico 87503
(505) 827-2601

Director, Prevention and Intervention
Office of Alcoholism and Substance Abuse Services
1450 Western Avenue
Albany, NY 12203
(518) 485-2123

Chief of Prevention
Alcohol and Drug Abuse Section
325 North Salisbury Street, Suite 531
Raleigh, NC 27611
(919) 733-4555

Acting NPN Designee
Division of Alcoholism and Drug Abuse
600 South Second Street, 1-E
Bismarck, ND 58504-5729
(701) 328-8922

Chief
Prevention Services and Training
Department of Alcohol and Drug Addiction Services
280 North High Street, 12th Floor
Columbus, OH 43215-2357
(614) 466-3445

Director, Prevention Services
Department of Mental Health and Substance Abuse
P.O. Box 53277
Oklahoma City, OK 73152
(405) 522-3866

Prevention Manager
Department of Human Resources
Office of Alcohol and Drug Abuse Programs
500 Summer Street NE
Salem, OR 97310-1016
(503) 945-6189

Director
Bureau of Preventive Health
Pennsylvania Department of Health, Room 929, Health & Welfare Building
Seventh and Foster Streets
Harrisburg, PA 17108
(717) 787-2712

Associate Administrator
Division of Substance Abuse
Department of Health
Cannon Building, 3 Capitol Hill
Providence, RI 02908-5097
(401) 277-4680

Director, Division of Programs and Services
Department of Alcohol and Other Drug Abuse Services
3700 Forest Drive, Suite 300
Columbia, SC 29204
(803) 734-9545

Prevention Coordinator
Division of Alcohol and Drug Abuse
700 Governors Drive
Pierre, SD 57501
(605) 773-3123

Director, Prevention Services
Bureau of Alcohol & Drug Abuse Services
Cordell Hull Building
426 Fifth Avenue North, Third Floor
Nashville, TN 37247-4401
(615) 741-1921

Prevention Council Coordinator
Commission on Alcohol and Drug Abuse
710 Brazos Street
Austin, TX 78701-2576
(512) 867-8847

Prevention Coordinator
Department of Social Services
Division of Substance Abuse
120 North 200 West, Fourth Floor
Salt Lake City, UT 84103
(801) 538-3939

Chief of Prevention
Office of Alcohol and Drug Abuse Programs
103 South Main Street
Waterbury, VT 05676
(802) 241-2170

Program Consultant
Office of Prevention
Department of Mental Health, Mental Retardation, and Substance Abuse Services
P.O. Box 1797
Richmond, VA 23214
(804) 786-1530

Program Manager for Prevention
Division of Alcohol and Substance Abuse
Mail Stop: OB-21W
Olympia, WA 98504
(360) 438-8200

Program Coordinator
Division of Alcohol and Drug Abuse
State Capitol Complex
Building 6, Room 738
Charleston, WV 25305
(304) 558-2276

Prevention Specialist
Bureau of Substance Abuse
1 West Wilson Street, Room 434
P.O. Box 7851
Madison, WI 53707-7851
(608) 266-9485

Substance Abuse Consultant
Alcohol and Drug Abuse Programs
447 Hathaway Building
Cheyenne, WY 82002
(307) 777-6493

Department of Human Resource
Alcohol and Drug Program
Government of American Somoa
Pago Pago, AS 96799
011 (684) 633-4210

Supervisor, Prevention Branch
Guam Department of Mental Health and Substance Abuse
790 Gov. Carlos G. Camacho Road
Tamuning, GU 96911
011 (671) 647-5415

Department of Public Health Services
P.O. Box 409
Saipan, MP 96950
011 (670) 323-6560

Assistant Administrator for Prevention
Services and Mental Health Promotion
P.O. Box 21414
Rio Piedras, PR 00928-1414
(787) 763-3133

Prevention Coordinator
Virgin Islands Division of Mental Health,
Alcohol & Drug Dependency
One Third Street, DeCastro Building
Sugar Estate
St. Thomas, VI 00802
(340) 774-7700

TREATMENT ALTERNATIVES TO STREET CRIME (TASC) PROGRAMS

University of Alabama Substance Abuse Programs
401 Beacon Parkway West
Birmingham, AL 35209
(205) 917-3784

Treatment Assessment Screening Center, Inc.
North Seventh Street
Phoenix, AZ 85006
(602) 254-7328

Treatment Assessment Screening Center, Inc.
5270 North 59th Avenue
Glendale, AZ 85301
(602) 842-4535

Treatment Assessment Screening Center, Inc.
1035 North McQueen, Suite 119
Gilbert, AZ 85234
(602) 497-5602

TASC/Adult Probation
1008 Front Street
Conway, AR 72032
(501) 327-3256

S.T.E.P. Drug Court
TASC Program
715 West Second Street
Little Rock, AR 72201
(501) 374-8613

Sonoma County Drug Abuse Services/TASC
830 Fifth Street, Suite C
Santa Rosa, CA 95404
(707) 527-7200

Southeast TASC
25 North Spruce, Suite 301
Colorado Springs, CO 80905
(719) 444-0882

Mile High TASC
1026 Bannock
Denver, CO 80215
(303) 595-4194

Division of Youth Services
3900 South Carr
Denver, CO 80235
(303) 987-4620

Denver Juvenile Justice Integrated Treatment
Network
303 West Colfax Avenue
Denver, CO 80204
(303) 893-6898

Denver Juvenile Justice Integrated TASC Project
Denver Juvenile Court, Suite 925
303 West Colfax Avenue
Denver, CO 80204
(303) 762-3113

TASC/NCADA
136 North Seventh Street
Grand Junction, CO 81501
(970) 243-3140

Northeast TASC
7255 Irving Street, Suite 106
Westminster, CO 80030
(303) 428-5264

The Connecticut Halfway House, Inc.
Juvenile Drug Treatment Program
310 Collins Street
Hartford, CT 06105
(860) 543-0101

The Connecticut Prison Association
110 Bartholomew Avenue
Hartford, CT 06106
(203) 566-2030

Net Counseling Center
813 West Street
Wilmington, DE 19801
(302) 657-8100

TASC/Delaware Treatment Access Center
1602-B Jessup Street
Wilmington, DE 19802
(302) 577-2711

Department of Health and Rehabilitative Services
Juvenile/Adult TASC
1317 Winewood Boulevard
Tallahassee, FL 32399-0700
(904) 922-4270

Levy County (NM)
Juvenile/Adult TASC
P.O. Box 516
Bronson, FL 32621
(904) 486-5310

Operation PAR
Juvenile TASC
14500 49th Street North
Suite 135
Clearwater, FL 34622
(813) 464-1455

Stewart Marchman Treatment Center
Juvenile TASC
3875 Tiger Beach Road
Daytona Beach, FL 32124
(904) 947-1300

Spectrum Programs, Inc.
Juvenile TASC
2701 West Oakland Park Boulevard, Suite 400
Ft. Lauderdale, FL 33311
(305) 777-2977

Ruth Cooper Center
Juvenile and Adult TASC
2789 Ortiz Avenue SE
Ft. Myers, FL 33905
(941) 275-3222

Gateway Community Services, Inc.
Juvenile TASC
1283 East Eighth Street
Jacksonville, FL 32206
(904) 356-9835

Drug Abuse Treatment Association, Inc.
Juvenile TASC
1016 North Clemons Street, Suite 406
Jupiter, FL 33477
(407) 743-1034

The Village/Virgin Islands TASC
Juvenile TASC
3180 Biscayne Boulevard
Miami, FL 32801
(305) 573-3784

Metro-Dade Office of Rehabilitative Services/TASC
Juvenile/Adult TASC
3300 Northwest 27th Avenue
Miami, FL 33142

David Lawrence Center/Court Related Services
Adult and Juvenile TASC
2806 Horseshoe Drive South
Naples, FL 33942-6125

Human Services Associates
Adult and Juvenile TASC
1703 West Colonial Drive
Orlando, FL 32804
(407) 422-0880

Escambia County
Adult and Juvenile TASC
1800 West Saint Mary Avenue
Box 6
Pensacola, FL 32501
(904) 436-9855 or (904) 436-9856

Coastal Recovery Centers, Inc.
Juvenile TASC
2080 Ringling Boulevard
Sarasota, FL 34237
(813) 365-4469

Regional Professional Center
Juvenile TASC
4000 East Third Street, Suite 2000
Springfield, FL 32404
(904) 872-4825

DISC Village Juvenile and Adult TASC Program
3333 West Pensacola Street
Tallahassee, FL 32304
(904) 575-4388

DACCO Juvenile and Adult TASC
4422 East Columbus Drive
Tampa, FL 33605
(813) 623-3500

ACTS Juvenile TASC
4211 E. Busch Boulevard, Suite H
Tampa, FL 33617
(813) 988-6096

Guidance Clinic of Upper Keys
S.C.A.T. Juvenile TASC
P.O. Box 363
Tavernier, FL 33070
(305) 852-3284

Coastal Recovery Centers, Inc.
Adult TASC
410 Cortez Road West, Suite 410
Bradenton, FL 34207
(813) 727-7719

ACT Corporation
Adult TASC
1220 Willis Avenue
Daytona Beach, FL 32114
(904) 239-6134

Stewart Marchman Treatment Center
Adult TASC
120 Michigan Avenue
Daytona Beach, FL 32114
(904) 947-1357

Central Florida Human Services Center
Program to Aid Drug Abusers (PAD)/Adult TASC
2920 Franklin Street
P.O. Box 1593
Eaton Park, FL 33840
(813) 665-2211

Spectrum
Adult TASC Program
2301 Wilton Drive
Ft. Lauderdale, FL 33305
(305) 563-6413

New Horizons of the Treasure Coast
Adult TASC
415 Avenue A, Suite 304
Ft. Pierce, FL 34950
(407) 468-5656/5663

TASC/Tri-County Services Adult TASC
4300 Southwest 13th Street
Gainesville, FL 32608
(904) 463-3145

Starting Place
Adult TASC
2057 Coolidge Street
Hollywood, FL 33020
(305) 925-1045

River Region Human Services Center
Adult TASC
330 West State Street
Jacksonville, FL 32202
(904) 359-6571

North Florida Mental Health Centers
Adult TASC
P.O. Box 2818
Lake City, FL 32056-2818
(904) 752-1045
Adult: (904) 758-0560

Gateway Community Services Adult TASC
P.O. Box 502
McClenny, FL 32063

The Harbor Behavioral Health Care Institute
Adult TASC
P.O. Box 428
New Port Ritchey, FL 34656
(813) 943-5366

Operation PAR Adult TASC
6655 66th Street North
Pinellas Park, FL 34665
(813) 545-6416

Circles of Care/Brevard County Adult TASC
1770 Cedar Street
Rockledge, FL 32955
(407) 632-9480

TASC/Adult
2080 Ringling Boulevard, Suite 201B
Sarasota, FL 34237
(813) 365-4469

Operation PAR
Adult TASC
10901-C Roosevelt Boulevard, Suite 1000
St. Petersburg, FL 33716-2336
(813) 538-7280

TASC
State Board of Pardons and Paroles Special Services Unit
Two Northside 75, Suite 134
Atlanta, GA 30318
(404) 656-5651

DeKalb County Court Services
Risk Reduction Program
DeKalb Addiction Clinic
1260 Briarcliff Road NE
Atlanta, GA 30306
(404) 894-5806

Hawaii Paroling Authority/TASC
429A Walakamilo Road
Honolulu, HI 96817
(808) 832-3479

AREA I
TASC, Inc.
Adult Programs
2600 South California Avenue, Room 107
Chicago, IL 60608
(312) 376-0950 or 0897

TASC, Inc.
Adult Programs
1500 North Halsted, Second Floor
Chicago, IL 60622
(312) 787-0208

TASC, Inc.
Juvenile Programs
1100 South Hamilton, Room 12
Chicago, IL 66012
(312) 666-7339

AREA II
TASC, Inc.
Adult and Juvenile Programs
119 North Church Street, Suite 202
Rockford, IL 61101
(815) 965-1106

AREA III
TASC, Inc.
Adult Programs
Regency Plaza Office Building
2525 - 24th Street, Suite 101
Rock Island, IL 61201
(309) 788-0816

AREA IV
TASC, Inc.
Adult and Juvenile Programs
Central Building
101 Southwest Adams Street, Suite 420
Peoria, IL 61602
(309) 673-3769 or 3794

AREA V
TASC, Inc.
Adult and Juvenile Programs
Three Old Capitol Plaza West, Suite 8
Springfield, IL 62701
(217) 544-0842

AREA VI
TASC, Inc.
Adult Programs
104 West University
Urbana, IL 61801
(217) 344-4546

AREA VII
TASC, Inc.
Adult and Juvenile Programs
110 West Main Street
Belleville, IL 62220
(618) 277-0410

AREA VIII
TASC, Inc.
Adult and Juvenile Programs
Court Square Offices
400 West Jackson, Suite B
Marion, IL 62959
(618) 997-8181

AREA IX
TASC, Inc.
Adult and Juvenile Programs
103 Plaza Court
Edwardsville, IL 62025
(618) 656-7672

AREA X
Roosevelt Glen Corporate Center
Adult and Juvenile Programs
799 Roosevelt Road
Building 6, Suite 2
Glen Ellyn, IL 60137
(708) 858-7400

ACP (Alcohol Countermeasures/ Probation/TASC)
226 West Wallace Street
Ft. Wayne, IN 46802
(219) 449-7134

St. Joseph's Hospital
1900 Medical Arts Drive
Huntingburg, IN 47542
(812) 683-2121

TASC Component/Municipal Court Probation
Marion County Municipal Courts
Room T641
200 East Washington Street
Indianapolis, IN 46204
(317) 236-3841

TASC
Dubos Superior Court Courthouse
Jasper, IN 47546
(812) 482-1661

St. Joseph County TASC
St. Joseph County Superior Court Courthouse
South Bend, IN 46601
(219) 284-9550

Iowa TASC
Department of Corrections
Capitol Annex
523 East 12th Street
Des Moines, IA 50319
(515) 281-4592

Department of Correctional Services
510 Fifth Street
Ames, IA 50010
(515) 232-1511

Department of Correctional Services
53 Third Avenue Bridge
P.O. Box 74740
Cedar Rapids, IA 52401
(319) 398-3474

Department of Correctional Services
801 South 10th Street
Council Bluffs, IA 51501
(712) 325-0782

Department of Correctional Services
Community Resource Center
605 Main Street, Box 2A
Davenport, IA 52803-5293
(319) 322-7986

Department of Correctional Services
1000 Washington Avenue
Des Moines, IA 50313
(515) 242-6610

Ottumwa Residential Facility
Department of Correctional Services
245 Osage Drive
Ottumwa, IA 52501
(515) 682-3069

Department of Correctional Services
515 Water Street
Sioux City, IA 51101
(712) 252-0590

Department of Correctional Services
527 East Fifth Street
P.O. Box 2596
Waterloo, IA 50704
(319) 291-2091

DCCCA Center
3312 Clinton Parkway
Lawrence, KS 66047
(913) 841-4138

TASC/Early Intervention
Somerset County Jail
Five High Street
Skowhegan, ME 04976
(207) 474-9591

Health Reach Network
Eight Highwood Street
P.O. Box 1568
Waterville, ME 04903-1568
(207) 873-1127

TASC Project
105 Fleet Street
Rockville, MD 20850
(301) 279-1332

Baltimore County Alternative Sentencing/TASC
201 West Chesapeake
Towson, MD 21204
(410) 887-2056

Dimock Justice Resource Services
55 Dimock Street
Roxbury, MA 02119
(617) 442-6769

HSTA-TASC
174 Clark Street
Detroit, MI 48209
(313) 876-4066

Administrative Offices of the Court
Criminal Practice Division
CN982
Trenton, NJ 08625
(609) 984-0114

Warren County TASC
Warren County Court House
Belvidere, NJ 07823
(908) 475-6263

Camden County TASC
Criminal Division
Hall of Justice
Camden, NJ 08103
(609) 225-7186

Newark Target Cities
Probation Department
110 South Grove Street
East Orange, NJ 07018
(201) 677-8042

Union County TASC
Annex Building, Second Floor
Union County Court House
Elizabeth, NJ 07207
(908) 527- 4344

Union County TASC
Criminal Division/PTI
1143-1145 East Jersey Street, Third Floor
Union County Court House
Elizabeth, NJ 07207
(908) 527-4338

Monmouth County TASC
Monmouth County Court House
Court Street
Freehold, NJ 07728
(908) 303-7696

Hudson County TASC
P.O. Box 806
North Arlington, NJ 07037
(908) 322-6721

Atlantic County TASC
Criminal Court House
Main Street, Room 238
Mays Landing, NJ 08830
(609) 625-7000, ext. 5392

Morris County TASC
Morris County Court House
Criminal Division
P.O. Box 900
Morristown, NJ 07960-0900
(201) 829-8052

Burlington County TASC
Burlington County Courts Facility
49 Rancocas Road
Mount Holly, NJ 08060
(609) 265-5335

Middlesex County TASC
Court House
JFK Square
New Brunswick, NJ 08903
(908) 745-3873

Essex County TASC
Criminal Division
New Courts Building, Room 141
Newark, NJ 07102
(201) 621-5086

Newark Municipal Court
Probation Department
31 Green Street
Newark, NJ 07102
(201) 621-5297

Newark Target Cities
New Courts Building, Room 141
Newark, NJ 07102
(201) 621-5086

Newark Target Cities
Family Court Division
Old Court House, Room 312
470 Martin Luther King Boulevard
Newark, NJ 07102
(201) 621-5337

Passaic County TASC
Criminal Division
77 Hamilton Street
Paterson, NJ 07505
(201) 881-7689

Ocean County TASC
Criminal Division
P.O. Box 2191
Toms River, NJ 08754-2191
(908) 929-4780 ext. 2244

Gloucester County TASC
Criminal Justice Complex
Hunter Street
P.O. Box 187
Woodbury, NJ 08096
(609) 853-3588

TASC of the Capital District, Inc.
87 Columbia Street
Albany, NY 12210
(518) 465-1455

Steuben County Probation Department
3 East Pulteney Square
Bath, NY 14810
(607) 776-9631

EAC/Brooklyn Bridge
188 Montague Street, Room 404
Brooklyn, NY 11201
(718) 237-9404

EAC, Inc.
1 Old Country Road, Suite 420
Carle Place, NY 11514
(516) 741-5580

TASC of Orange County
224 Main Street
P.O. Box 583
Goshen, NY 10924
(914) 294-9600

EAC/Suffolk TASC
County Center North
Veterans Memorial Highway
Building 804
Hauppauge, NY 11788
(516) 853-5777

EAC/Nassau TASC.
250 Fulton Avenue
Hempstead, NY 11550
(516) 486-8944

EAC/Queens TASC
124-26 Queens Boulevard
Kew Gardens, NY 11415
(718) 268-5657

ASAC of Ulster County, Inc.
785 Broadway
Kingston, NY 12401
(914) 331-9331

Niagara County Probation Department
Niagara Civic Building
775 Third Street
Niagara Falls, NY 14302
(716) 284-3133

TASC/Monroe County of Probation
80 West Main Street
Rochester, NY 14614
(716) 428-2624

EAC/Staten Island TASC
387 Van Duzer Street
Staten Island, NY 10304
(718) 727-9722

Center for Community Alternatives
351 South Warren Street, Suite 500
Syracuse, NY 13202
(315) 422-5638

Westchester County
Treatment Alternatives To Street Crime
112 East Post Road, Second Floor
White Plains, NY 10601
(914) 285-5265

Crisis Services Section
North Carolina Department of MH/DD/SAS
325 North Salisbury Street, Room 1129
Raleigh, NC 27611
(919) 733-1763

Alcohol and Drug Abuse Services
North Carolina Department of MH/DD/SAS
325 North Salisbury Street
Raleigh, NC 27611
(919) 733-0566

Blue Ridge Area MH/MR and Substance Abuse Services
283 Biltmore Avenue
Asheville, NC 28801
(704) 252-8748

McLeod Center/TASC
145 Remount Road
Charlotte, NC 28203
(704) 332-9001

Durham County Substance Abuse Services
705 South Mangum Street
Durham, NC 27701
(919) 560-7531

Albemarle Mental Health
TASC/Substance Abuse Center
P.O. Box 326
Elizabeth City, NC 27907
(919) 331-7660

Cumberland County TASC
109 Bradford Avenue
P.O. Box 3069
Fayetteville, NC 28301
(910) 433-2712

Gaston-Lincoln Mental Health/ DD/SA/TASC
816 Mauney Avenue
Gastonia, NC 28052
(704) 854-4882

Alamance-Coswell Area MH/DD/ SA Program
114 South Maple Street, Suite D
Graham, NC 27253
(910) 513-4370

Alcohol & Drug Services of Guilford
301 East Washington Street, Suite 101
Greensboro, NC 27401
(910) 333-6860

Pitt County Mental Health TASC Program
301 South Evans Street, Suite 201
Greenville, NC 27834
(919) 758-0034

Skinner House TASC/DWI Program
123 West Third Street
Greenville, NC 27834
(919) 758-0034

VGFW MH/DD/SAS
129 Belle Street
Henderson, NC 27536
(919) 430-3801

Albemarle Mental Health Center TASC Project
P.O. Box 130
Manteo, NC 27954
(919) 473-1135

TASC Project
P.O. Box 1685
Nags Head, NC 27959
(919) 441-3366

Tideland Enhanced TASC
Tideland Mental Health Center
202 East Water Street
Plymouth, NC 27962
(919) 791-1010

SouthLight
2500 Blue Ridge Road, Suite 400
Raleigh, NC 27607
(919) 787-6131

Edgecombe-Nash TASC
500 Nash Medical Arts
Rocky Mount, NC 27804
(919) 937-8141

Substance Abuse Center
417 North Main
Salisbury, NC 28144
(704) 637-9301

Coastal Horizons Center (TASC)
801 Princess Street
Wilmington, NC 28401
(910) 343-0145

Step One Inc.
Substance Abuse Services
665 West Fourth Street
Winston-Salem, NC 27101
(910) 725-8389

Ohio Department of Alcohol and Drug Addiction
Services
Two Nationwide Plaza
280 North High Street, 12th Floor
Columbus, OH 43215-2537
(614) 752-8330

Clermont County TASC
4440 State Route 222
Batavia, OH 45103
(513) 732-7546

Stark County TASC
218 Second Street NW, Suite 105
Canton, OH 44703
(216) 588-7180

Cuyahoga County TASC
1276 West Third Street, Suite 525
Cleveland, OH 44115
(216) 443-8250

Preble County Juvenile TASC
204 North Barron Street
Eaton, OH 45320
(513) 456-3443

Ohio Department of Alcohol and Drug Addiction
Services
Two Nationwide Plaza
280 North High Street, 12th Floor
Columbus, OH 43215-2537
(614) 752-8330

Clermont County TASC
4440 State Route 222
Batavia, OH 45103
(513) 732-7546

Stark County TASC
218 Second Street NW, Suite 105
Canton, OH 44703
(216) 588-7180

Cuyahoga County TASC
1276 West Third Street, Suite 525
Cleveland, OH 44115
(216) 443-8250

Preble County Juvenile TASC
204 North Barron Street
Eaton, OH 45320
(513) 456-3443

TASC of Oregon, Inc.
1733 Northeast Seventh Street
Portland, OR 97212
(503) 281-0037

Marion County Department of Corrections
3060 Center Street NE
Salem, OR 97301
(503) 588-5289

Office of Drug and Alcohol Programs
Department of Health
929 Health & Welfare Building
P.O. Box 90
Harrisburg, PA 17108
(717) 787-2712

Lehigh County TASC
521 Court Street
Allentown, PA 18101
(610) 432-6760

Centre County TASC
Keystone Community Services
111 East High Street
Bellefonte, PA 16823
(814) 353-9450

Franklin-Fulton TASC
425 Franklin Farm Lane
Chambersburg, PA 17201
(717) 263-1256

Bucks County TASC
252 West Swamp Road, Unit 33
Doylestown, PA 18901
(215) 230-8715

Clearfied-Jefferson County TASC
c/o Gateway Institute & Clinic
100 Caldwell Drive
Dubois, PA 15801
(814) 371-1100

Northampton County TASC
Treatment Trends
158-160 South Third Street
Easton, PA 18042
(610) 250-3961

Erie County TASC
GECAC Drug and Alcohol Service
809 Peach Street
Erie, PA 16501
(814) 459-4581 ext. 528

Chester County TASC
Whiteland Business Park
930 East Lancaster Avenue
Exton, PA 19341
(610) 363-7709

Venango County TASC
1283 Liberty Street
P.O. Box 1130
Franklin, PA 16323
(814) 432-9744

York/Adams County TASC
Drug and Alcohol Treatment and Prevention Services
108 North Stratton Street
Gettysburg, PA 17325
(717) 334-8154

Westmoreland County TASC
Comprehensive Substance Abuse Services of
Southwestern Pennsylvania, Inc.
203 South Maple Avenue, Room 215
Greensburg, PA 15601
(412) 832-5880

Dauphin County TASC
25 South Front Street, Suite 825
Harrisburg, PA 17101
(717) 255-2984

C.M.P.
128 South First Street
Lehighton, PA 18235
(717) 421-1960

Mercer County TASC
Intermediate Punishment Program
403 Court Street
Mercer, PA 16137
(412) 862-3880

Montgomery County TASC
18 West Main Street
Norristown, PA 19401
(610) 279-4262

Allegheny County TASC
Ielase Institute of Forensic Psychology
232 First Avenue
Pittsburgh, PA 15222
(412) 261-2817

Berks County TASC
524 Washington Street
Reading, PA 19601
(610) 375-4426

Lackawanna County TASC
Drug and Alcohol Treatment Service
116 North Washington Avenue
Scranton, PA 18503
(717) 961-1997

Carbon County TASC
14 North Sixth Street
Stroudsburg, PA 18260
(717) 421-1960

Luzerne/Wyoming County TASC
Catholic Social
Services
33 Northampton Street
Wilkes-Barre, PA 18701
(717) 822-7118

Green Ridge Counseling Center
829 West Fourth Street
Williamsport, PA 17701
(717) 322-1216

York/Adams County TASC
Stepping Stone Counseling and Education Services
211 South George Street
York, PA 17403
(717) 854-0444

Department of Substance Abuse
P.O. Box 20363
Cranston, RI 02920
(401) 464-2381

South Carolina Department of Alcohol and Other Drug
Abuse Services
3700 Forest Drive, Suite 300
Columbia, SC 29204
(803) 734-9520

Treatment Alternatives to Incarceration Program (TAIP)
Texas Commission on Alcohol and Drug Abuse
710 Brazos
Austin, TX 78701-2506
(512) 867-8700

Treatment Alternatives to Incarceration Program (TAIP)
Travis County Community Supervision and Corrections
Department, Suite 700
411 West 13th Street
Executive Office Building
Austin, TX 78701
(512) 473-9540

Dallas County Treatment Alternatives to Incarceration Program (TAIP)
38 Trailview Drive
Carrollton, TX 75007

Treatment Alternatives to Incarceration Program (TAIP)
Rio Grande Council on Government
1100 North Stanton, Suite 1610
El Paso, TX 79902
(915) 533-0998

Treatment Alternatives to Incarceration Program (TAIP)
200 West Belknap Street
Fort Worth, TX 76196
(817) 884-2449

Central Texas Treatment Center/ Adult Probation
P.O. Box 662
Georgetown, TX 78627
(512) 869-0643

Treatment Alternatives to Incarceration Program (TAIP)
Houston Council on Alcohol and Drug Abuse
3333 Eastside, Suite 111
Houston, TX 77098
(713) 520-5502

Treatment Alternatives to Incarceration Program (TAIP)
200 Main Plaza, Suite 300
San Antonio, TX 78200
(210) 978-0443

Behavioral Education Associates and TASC Associates
1823 Stadium Road, #209
P.O. Box 213
Wharton, TX 77488-0213
(409) 282-2813

Richmond TASC
Richmond Mental Health
Mental Retardation/Substance Abuse Services
2930 West Broad Street, Suite 3
Richmond, VA 23230
(804) 780-4536

Snohomish County TASC/Pacific Treatment
Alternatives
1114 Pacific Avenue
Everett, WA 98201
(206) 259-7142

King County TASC/Drug Free Systems
811 First Avenue, Suite 610
Seattle, WA 98104
(206) 467-0338, ext. 111

NorthEast Washington Treatment Alternative/TASC
1224 North Ash
Spokane, WA 99201
(509) 326-7740

Tacoma TASC/Pierce County Alliance
510 Tacoma Avenue S
Tacoma, WA 98402-5416
(206) 572-4750

Pacific Crest Consortium/Clark County TASC
2402 Broadway
Vancouver, WA 98663
(206) 693-2243

Yakima County TASC
Yakima County Alcohol/Drug Assessment & Referral Center
Yakima County Courthouse
128 North Second Street, Room B-18
Yakima, WA 98901
(509) 575-4472

Rock Valley Treatment Alternatives Program
431 Olympian Boulevard
Beloit, WI 53511
(608) 362-5592

Treatment Alternatives Program
Triniteam, Inc.
202 Graham Avenue
Eau Claire, WI 54701
(715) 836-8114

Treatment Alternatives Program (TAP)
Wisconsin Department of Health & Social Services
1 West Wilson Street
P.O. Box 7851
Madison, WI 53707-7851
(608) 266-3145

Dane County Treatment Alternatives Program
702 West Main Street
Madison, WI 53715
(608) 256-4502

Department of Human Services
1206 North Port Drive
Madison, WI 53704
(608) 242-6474

Wisconsin Correctional Service
436 West Wisconsin Avenue
Milwaukee, WI 53203
(414) 271-2512

Programa TASC DSCA (NM)
Apartado 1190
Arecibo, PR 00613
(787) 879-2021

TASC Departamento de Servicios Contra La Adiccion
414 Barbosa Avenue
Hato Rey, PR 00912
(787) 763-7575

Ponce TASC (NM)
P.O. Box 7321
Ponce, PR 00732

STATE LISTINGS

State Office Functions

STATE POLICY OFFICES

Office of the Governor
Establishes policy priorities and issues executive orders; responsible for the implementation of legislation; responsible for designating the state agency that applies for federal drug law enforcement, education, treatment, and prevention funds.

State Legislature
Enacts enabling legislation and provides oversight of executive agency activities; sets funding levels for statewide drug law enforcement, treatment, and prevention.

State Drug Program Coordinator
Establishes a statewide drug abuse action plan and coordinates the activities of executive branch agencies; helps to establish program priorities.

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Establishes legal guidelines for the implementation of legislation and the prosecution of offenders; helps coordinate statewide drug task force activities.

Law Enforcement Planning Office
Executive branch agency responsible for coordinating statewide criminal justice initiatives.

Crime Prevention Office
Monitors statewide crime prevention efforts between law enforcement agencies and the community; disseminates drug and crime prevention literature to schools and the general public.

Statistical Analysis Centers
Assembles statewide criminal justice statistics and issues periodic reports; acts as a clearinghouse for statewide crime information and statistics.

Uniform Crime Reports
Assembles statewide UCR offense and arrest data and produces annual report; submits statewide arrest statistics to the FBI's National Uniform Crime Reports for inclusion in the annual *Crime in the United States*.

BJA Strategy Preparation Agency
Prepares and submits to the Bureau of Justice Assistance (BJA) a State drug strategy; distributes BJA grant funds in accordance with the strategy; performs other analyses of statewide drug problems and appropriate interventions.

Judicial Agency
The administrative office of the state court system coordinates the activities of the various judicial districts, gathers state court data, and issues periodic reports.

Corrections Agency
Operates the state prison system; establishes in-prison programs; collects statistics on correctional populations.

STATE HEALTH OFFICES

RADAR (Regional Alcohol and Drug Awareness Resource) Network Agency
State office responsible for distributing alcohol and drug abuse prevention and education materials. Established by the U.S. Department of Health and Human Services' Office of Substance Abuse Prevention, these activities are coordinated by the National Clearinghouse for Alcohol and Drug Information.

HIV-Prevention Program
Coordinates state AIDS prevention activity and oversees state AIDS prevention funding.

Drugs and Alcohol Agency
Sets prevention and treatment priorities and administers state and federal funds, particularly those from the U.S. Department of Health and Human Services' Office of Substance Abuse Prevention.

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Establishes school-based drug and alcohol prevention/education programs and administers federal Drug-Free Schools and Communities funds.

States

Alabama

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol
600 Dexter Avenue
Montgomery, AL 36104
(334) 242-7100
E-mail: govjames@asnmail.asc.edu

State Legislative Contact
Legislative Reference Service
State House
Room 613
11 South Union
Montgomery, AL 36130-6701
(334) 242-7560

State Drug Program Coordinator
Alabama Department of Public Safety
2720-A Gunter Park Drive West
Montgomery, AL 36109-1014
(334) 260-1100

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Attorney General's Office
State House
11 South Union Street
Montgomery, AL 36130-1801
(334) 242-7300
E-mail: alattgen@counsel.com

Law Enforcement Planning
Alabama Department of Economic and Community Affairs
Law Enforcement Planning
401 Adams Avenue
P.O. Box 5690
Montgomery, AL 36103-5690
(334) 242-5803

Statistical Analysis Center
Alabama Criminal Justice Information Center
770 Washington Avenue, Suite 350
Montgomery, AL 36130
(334) 242-4900

Uniform Crime Reports Contact
Uniform Crime Reports Program
Alabama Criminal Justice Information Center
770 Washington Avenue, Suite 350
Montgomery, AL 36130
(334) 242-4900

BJA Strategy Preparation Agency
Alabama Department of Economic and Community Affairs
Law Enforcement/Traffic Safety Division
P.O. Box 5690
401 Adams Avenue
Montgomery, AL 36103-5690
(334) 242-5891

Judicial Agency
Judicial Agency
Administrative Office of Courts
300 Dexter Avenue
Montgomery, AL 36104-3741
(334) 242-0300

Corrections Agency
Department of Corrections
Treatment Division
1400 Lloyd Street
Montgomery, AL 36130-1501
(334) 240-9586

STATE HEALTH OFFICES

RADAR Network Agency
Alabama Department of Mental Health/Mental Retardation
Division of Substance Abuse Services
100 North Union Street
P.O. Box 301410
Montgomery, AL 36130-1410
(334) 242-3966

HIV-Prevention Program
Department of Public Health
Disease Control Bureau
HIV/AIDS Division
434 Monroe Street
Montgomery, AL 36130-1410
(334) 613-5364

Drug and Alcohol Agency
Drug and Alcohol Agency
Alabama Department of Mental Health and Mental Retardation
Substance Services Division
P.O. Box 301410
Montgomery, AL 36130-1410
(334) 242-3961

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Drug Education Program
State Department of Education
50 North Ripley Street, Room 5348
Montgomery, AL 36130-3901
(334) 242-8199

Alaska

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 110001
Juneau, AK 99811-0001
(907) 465-3500

State Legislative Contact
Legislative Affairs Agency
130 Seward Street, Suite 313
Juneau, AK 99801-2197
(907) 465-4648
E-mail: juneau-lio@legis.state.ak.us

State Drug Program Coordinator
Alaska Division of Alcohol and Drug Abuse
P.O. Box 110607
Juneau, AK 99811-0607
(907) 465-2071

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Attorney General
Department of Law
P.O. Box 110300
Juneau, AK 99811-0300
(907) 465-2133
E-mail: bruce-botelho@law.state.ak.u

Law Enforcement Planning
Alaska Department of Public Safety
P.O. Box 111200
Juneau, AK 99811-1200
(907) 465-4322
E-mail: pestocka@psafety.state.ak.us

Crime Prevention Office
Alaska Crime Prevention Association
P.O. Box 210-127
Anchorage, AK 99521-0127
(907) 338-5548

Statistical Analysis Center
The Justice Center
University of Alaska Anchorage
3211 Providence Drive
Anchorage, AK 99508
(907) 786-1810
E-mail: ayjust@uaa.alaska.edu

Uniform Crime Reports Contact
Uniform Crime Reporting Section
Department of Public Safety Information System
5700 East Tudor Road
Anchorage, AK 99507
(907) 269-5708

BJA Strategy Preparation Agency
Department of Public Safety
Alaska State Troopers
5700 East Tudor Road
Anchorage, AK 99507
(907) 269-5082
E-mail: ackatsel@psafety.state.ak.us

Judicial Agency
Administrative Office of the Courts
Alaska Court System
303 K Street
Anchorage, AK 99501
(907) 264-0547

Corrections Agency
Department of Corrections
4500 Diplomacy Drive, Suite 207
Anchorage, AK 99502
(907) 269-7350

STATE HEALTH OFFICES

RADAR Network Agency
Alaska Council on Prevention of Alcohol and Drug Abuse
3333 Denali Street, Suite 201
Anchorage, AK 99503
(907) 258-6021

HIV-Prevention Program
AIDS/STD Program Section of Epidemiology
Division of Public Health
P.O. Box 240249
Anchorage, AK 99524-0249
(907) 269-8000

Drug and Alcohol Agency
Division of Alcoholism and Drug Abuse
P.O. Box 110607
Juneau, AK 99811-0607
(907) 465-2071
E-mail: ljones%health@state.ak.us

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Alaska Department of Education
Drug-Free Schools Program
801 West 10th Street, Suite 200
Juneau, AK 99801-1894
(907) 465-8730

Arizona

STATE POLICY OFFICES

Governor's Office
Office of the Governor
1700 West Washington Street
Phoenix, AZ 85007
(602) 542-4331

State Legislative Contact
Legislative Council
State Capitol
Legislative Services Wing
Room 100
1700 West Washington Street
Phoenix, AZ 85007
(602) 542-4236

State Drug Program Coordinator
Governor's Division of Drug Policy
Suite 101-G
1700 West Washington Street
Phoenix, AZ 85007
(602) 542-3456
E-mail: gvboehl@ad.state.as.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
1275 West Washington Street
Phoenix, AZ 85007
(602) 542-4266

Law Enforcement Planning
Office of the Attorney General
1275 West Washington Street
Phoenix, AZ 85007
(602) 542-4266

Statistical Analysis Center
Arizona Criminal Justice Commission, Suite 207
1501 West Washington Street
Phoenix, AZ 85007
(602) 542-1928
E-mail: acjc@goodnet.com

Uniform Crime Reports Contact
Uniform Crime Reports Program
Arizona Department of Public Safety
P.O. Box 6638
Phoenix, AZ 85005
(602) 223-6638

BJA Strategy Preparation Agency
Arizona Criminal Justice Commission, Suite 207
1501 West Washington Street
Phoenix, AZ 85007
(602) 542-1928

Judicial Agency
Administrative Office of the Courts
Supreme Court
1501 West Washington Street,
Suite 411
Phoenix, AZ 85007
(602) 542-9301

Corrections Agency
Department of Corrections
1601 West Jefferson
Phoenix, AZ 85007
(602) 542-5497

STATE HEALTH OFFICES

RADAR Network Agency
Arizona Prevention Resource
Center
641 East Van Buren, Suite B-2
Phoenix, AZ 85004
(602) 727-2772

HIV-Prevention Program
Office of HIV/STD Services
Bureau of Epidemiology and
Disease Control Services
Arizona Department of Health
Services
3815 North African American
Canyon Highway
Phoenix, AZ 85015-5351
(602) 230-5819
E-mail: acjc@goodnet.com

Drug and Alcohol Agency
Bureau of Substance Abuse and
Mental Health
Arizona Department of Health
Services
2122 East Highland Avenue
Phoenix, AZ 85016
(602) 381-8999

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Arizona Department of Education
Title IV - Safe & Drug Free
Schools
1535 West Jefferson
Phoenix, AZ 85007
(602) 542-8728
E-mail:
colson@macpo.ade.state.az.us

Arkansas

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, Room 250
Little Rock, AR 72201
(501) 682-2345

State Legislative Contact
Bureau of Legislative Research
Legislative Council
State Capitol, Room 315
Fifth and Woodlane
Little Rock, AR 72201
(501) 682-1937

State Drug Program Coordinator
State Drug Director
State Capitol, Suite 250
Little Rock, AR 72201
(501) 682-2345

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
200 Tower Building
323 Center Street
Little Rock, AR 72201
(501) 682-2007

Law Enforcement Planning
Law Enforcement Standards and
Training Commission
P. O. Box 3106
East Camden, AR 71701
(501) 574-1810

Crime Prevention Offices
Arkansas Crime Information
Center
Office of Crime Prevention
One Capitol Mall 4D-200
Little Rock, AR 72201
(501) 682-2222
E-mail: acic@acic.org

Statistical Analysis Center
Special Services Section
Arkansas Crime Information
Center
One Capitol Mall, 4D-200
Little Rock, AR 72201
(501) 682-2222
E-mail: rthomas@acic.org

Uniform Crime Reports Contact
Arkansas Crime Information
Center
One Capitol Mall, 4D-200
Little Rock, AR 72201
(501) 682-2222

BJA Strategy Preparation Agency
Department of Finance and
Administration
Office of Intergovernmental
Services
1515 Building, Suite 417
Little Rock, AR 72203
(501) 682-1074

Judicial Agency
Administrative Office of the Courts
Supreme Court of Arkansas
Justice Building
Little Rock, AR 72201
(501) 682-9400

Corrections Agency
Department of Corrections
P.O. Box 8707
Pine Bluff, AR 71611
(501) 247-6200

STATE HEALTH OFFICES

RADAR Network Agency
Bureau of Alcohol and Drug Abuse
Prevention
Freeway Medical Center
5800 West 10th Street, Suite 907
Little Rock, AR 72204
(501) 280-4506
E-mail: adap@aristotle.net

HIV-Prevention Program
Arkansas Department of Health
Division of AIDS/STD
4815 West Markham, Slot #33
Little Rock, AR 72205
(501) 661-2408

Drug and Alcohol Agency
Bureau of Alcohol and Drug Abuse Prevention
Department of Health
Freeway Medical Center
5800 West 10th Street, Suite 907
Little Rock, AR 72204
(501) 280-4505

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Arkansas Department of Education
Drug Education Program
#4 Capitol Mall, Room 202B
Little Rock, AR 72201-1071
(501) 682-5170
E-mail: osmith@lokik12.ar.us

California

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol Building
Sacramento, CA 95814
(916) 445-2841

State Legislative Contact
Legislative Analyst's Office
925 L Street, Suite 1000
Sacramento, CA 95819
(916) 445-4660
E-mail: craig.cornett@lao.ca.gov

State Drug Program Coordinator
State Department of Alcohol and Drug Programs
1700 K Street, Fifth Floor
Sacramento, CA 95814-4037
(916) 445-0834

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
California Attorney General's Office
Division of Law Enforcement
Bureau of Narcotic Enforcement
P.O. Box 161089
Sacramento, CA 95816-1089
(916) 227-4044

Law Enforcement Planning
Division of Law Enforcement
4949 Broadway
Sacramento, CA 95820
(916) 227-2222

Crime Prevention Offices
Crime and Violence Prevention Center
California Department of Justice
Office of the Attorney General
P.O. Box 944255
Sacramento, CA 94244-2550
E-mail: agcvpc@ns.net

Statistical Analysis Center
Criminal Justice Statistics Center
4949 Broadway, Room E-231
P.O. Box 903427
Sacramento, CA 94203-4270
(916) 227-3382
E-mail: dlesac@ns.net

Uniform Crime Reports Contact
Uniform Crime Reports Program
Law Enforcement Information Center
Department of Justice
P.O. Box 903427
Sacramento, CA 94203-4270
(916) 227-3473

BJA Strategy Preparation Agency
Office of Criminal Justice Planning
Anti-Drug Abuse Branch
1130 K Street, Suite 300
Sacramento, CA 95814
(916) 324-9163

Judicial Agency
Administrative Office of the Courts
303 Second Street, South Tower
San Francisco, CA 94107
(415) 396-9100

Corrections Agency
Department of Corrections
P.O. Box 942883
Sacramento, CA 94283-0001
(916) 445-7688

STATE HEALTH OFFICES

RADAR Network Agency
Department of Alcohol and Drug Programs
1700 K Street, First Floor
Sacramento, CA 95814-4022
(916) 327-3728
http://www.adp.cahwnet.gov
E-mail:
adp.drepace@hwl.cahwnet.gov

HIV-Prevention Program
Director of Office of AIDS Programs and Policy
Los Angeles County Department of Health Services
600 South Commonwealth Avenue, Sixth Floor
Los Angeles, CA 90005
(213) 351-8000

Drug and Alcohol Agency
Department of Alcohol and Drug Programs
1700 K Street
Sacramento, CA 95814
(916) 445-0834

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
California Department of Education
Healthy Kids Programs Office
721 Capitol Mall, Third Floor
Sacramento, CA 95814
(916) 657-2810
E-mail: gkilbert@cde.ca.gov

Colorado

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, Room 136
Denver, CO 80203
(303) 866-2471
E-mail:
romer@governor.state.co.us

State Legislative Contact
Legislative Council
State Capitol, Room 029
200 East Colfax Avenue
Denver, CO 80203
(303) 866-3521

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Law
1525 Sherman Street, Fifth Floor
Denver, CO 80203
(303) 866-4500

Law Enforcement Planning
Division of Criminal Justice
Department of Public Safety
700 Kipling Street, Suite 3000
Lakewood, CO 80215
(303) 239-4442
E-mail: william.woodward@safety.state.co.us

Statistical Analysis Center
Colorado Division of Criminal Justice
700 Kipling Street, Suite 1000
Denver, CO 80215
(303) 239-4453
E-mail: kenglis8@aol.com

Uniform Crime Reports Contact
Uniform Crime Reports Section
Colorado Bureau of Investigation
690 Kipling Street
Denver, CO 80215
(303) 239-4300

BJA Strategy/Preparation Agency
Division of Criminal Justice
700 Kipling Street, Suite 3000
Denver, CO 80215
(303) 239-4442
E-mail: tregn@aol.com

Judicial Agency
Administrative Office of the Courts
Judicial Department
1301 Pennsylvania, Suite 300
Denver, CO 80203-2416
(303) 861-1111 ext. 125

Corrections Agency
Department of Corrections
2862 South Circle Drive, Suite 400
Colorado Springs, CO 80906
(719) 579-9580

STATE HEALTH OFFICES

RADAR Network Agency
Colorado Department of Human Services
Alcohol and Drug Abuse Division
Prevention-Intervention Section
4300 Cherry Creek Drive South
Denver, CO 80222-1530
(303) 692-2956
E-mail: linda.garrett@state.co.us

HIV-Prevention Program
Department of Health
STD/AIDS Section
4300 Cherry Creek Drive South
Denver, CO 80222-1530
(303) 692-2500

Drug and Alcohol Agency
Alcohol and Drug Abuse Division
Health Office
4300 Cherry Creek Drive South
Denver, CO 80222-1530
(303) 692-2930

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Colorado Department of Education
Prevention Initiatives Unit
201 East Colfax Avenue
Denver, CO 80203
(303) 866-6869
E-mail: jackson_k@ade.state.co.us

Connecticut

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Executive Chambers
210 Capitol Avenue
Hartford, CT 06106
(860) 566-4840

State Legislative Contact
Office of Legislative Research
Room 5300
Legislative Office Building
Hartford, CT 06106
(860) 240-8400

State Drug Program Coordinator
Office of Policy and Management
Policy Development Planning Division
P.O. Box 341441
450 Capitol Avenue, MS 52-CPD
Hartford, CT 06106
(860) 418-6394

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
55 Elm Street
Hartford, CT 06106
(860) 566-6026

Law Enforcement Planning
Policy Development and Planning Division
Office of Policy Management
450 Capitol Avenue, MS 52-CPD
P.O. Box 341441
Hartford, CT 06134-1441
(860) 418-6249

Crime Prevention Office
Crime Prevention Association of Connecticut
120 Main Street
Danbury, CT 06810
(203) 797-4577

Statistical Analysis Center
Policy Development and Planning Division
450 Capitol Avenue, MS 52-CPD
P.O. Box 341441
Hartford, CT 06134-1441
(860) 418-6376
E-mail: dolly.reed@po.state.ct.us

Uniform Crime Reports Contact
Uniform Crime Reporting Program
1111 Country Club Road
P.O. Box 2794
Middletown, CT 06457-9294
(860) 685-8030

BJA Strategy Preparation Agency
Office of Policy and Management
450 Capitol Avenue, MS 52-CPD
P.O. Box 341441
Hartford, CT 06134-1441
(860) 418-6210

Judicial Agency
Connecticut Judicial Branch
Office of the Chief Court Administrator
Supreme Court
231 Capitol Avenue
P.O. Drawer N, Station A
Hartford, CT 06106
(203) 566-4461

Corrections Agency
Department of Corrections
340 Capitol Avenue
Hartford, CT 06106
(860) 566-4457

STATE HEALTH OFFICES

RADAR Network Agency
Connecticut Clearinghouse
334 Farmington Avenue
Plainville, CT 06062
(860) 793-9791
E-mail: dolly.reed@po.state.ct.us

HIV-Prevention Program
Department of Public Health
AIDS Prevention & Intervention Programs
P.O. Box 340308
410 Capitol Avenue, MS 11APV
Hartford, CT 06134-0308
(860) 509-7801

Drug and Alcohol Agency
Division of Community Based Regulation
P.O. Box 340308
410 Capitol Avenue
Hartford, CT 06134-0308
(860) 509-8045

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Connecticut Department of Education
P.O. Box 2219, Room 215
Hartford, CT 06145
(860) 566-6645

Delaware

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Tatnall Building
Dover, DE 19901
(302) 739-4101
E-mail: gcarter@state.de.us

State Legislative Contact
Legislative Council
Legislative Hall
Legislative Avenue
PO Box 1401
Dover, DE 19901
(302) 736-4114

State Drug Program Coordinator
Chairman
Department of Public Safety
P.O. Box 818
Dover, DE 19901
(302) 739-4321

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Justice
820 North French Street
Wilmington, DE 19801
(302) 577-3838

Law Enforcement Planning
Criminal Justice Council
Elbert N. Carvel State Office Building, Fourth Floor
820 North French Street
Wilmington, DE 19801
(302) 577-3466 (beeper)

Statistical Analysis Center
60 The Plaza
Dover, DE 19901
(302) 739-4626

Uniform Crime Reports Contact
Uniform Crime Reports Program
State Bureau of Identification
P.O. Box 430
Dover, DE 19903-0430
(302) 739-5875

BJA Strategy Preparation Agency
Criminal Justice Council
Elbert N. Carvel State Office Building
820 North French Street, 4th Floor
Wilmington, DE 19801
(302) 577-3466

Judicial Agency
Administrative Office of the Courts
Elbert N. Carvel State Office Building
820 North French Street, 11th Floor
Wilmington, DE 19801
(302) 577-2480
E-mail: eboulden@state.de.us

Corrections Agency
Department of Corrections
80 Monrovia Avenue
Smyrna, DE 19977
(302) 739-5601

STATE HEALTH OFFICES

RADAR Network Agency
Office of Prevention Resource Clearinghouse
Delaware Youth and Family Center
1825 Faulkland Road
Wilmington, DE 19805-1195
(302) 892-4500

HIV-Prevention Program
Division of Public Health
AIDS/HIV Program Office
Jesse Cooper Building
P.O. Box 637
Dover, DE 19903
(302) 739-3032

Drug and Alcohol Agency
Division of Alcoholism, Drug Abuse and Mental Health
Department of Health and Social Services
1901 North DuPont Highway
New Castle, DE 19720
(302) 577-4461

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Public Instruction
Health Education and Services
P.O. Box 1402
Dover, DE 19903
(302) 739-4676
E-mail: evincent@state.de.us

District of Columbia

POLICY OFFICES

Mayor's Office
Executive Office of the Mayor
Office of Communications
One Judiciary Square
441 Fourth Street NW, Suite 1100
Washington, DC 20001
(202) 727-6224

Legislative Contact
Office of the Corporation Council
441 Fourth Street NW, Suite 1060N
Washington, DC 20001
(202) 727-6248

Drug Program Coordinator
Office of Criminal Justice Plans and Analysis
717 14th Street NW, Suite 500
Washington, DC 20005
(202) 727-9472

CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Corporation Counsel, D.C.
One Judiciary Square
441 Fourth Street NW, Suite 1060N
Washington, DC 20001
(202) 727-6248

Law Enforcement Planning
Office of Grants Management and Development
717 14th Street NW, Suite 400
Washington, DC 20001
(202) 727-6537

Statistical Analysis Center
Office of Grants Management & Development
717 14th Street NW, Suite 400
Washington, DC 20005
(202) 727-6537

Uniform Crime Reports Contact
Uniform Crime Reports Program
Information Services Division
Metropolitan Police Department, Room 5054
300 Indiana Avenue NW
Washington, DC 20001
(202) 727-4301

BJA Strategy Preparation Agency
Office of Grants Management and Development
717 14th Street NW, Suite 400
Washington, DC 20001
(202) 727-6554

Judicial Agency
Administrative Office of the Courts
District of Columbia Courts Room 1500
500 Indiana Avenue NW
Washington, DC 20001
(202) 879-1700

Corrections Agency
Department of Corrections
1923 Vermont Avenue NW
Washington, DC 20001
(202) 673-7316

HEALTH OFFICES

RADAR Network Agency
Addiction Prevention and Recovery Administration
(APRA)
Office of Prevention and Youth Services
1300 First Street NE, Third Floor
Washington, DC 20002
(202) 727-0716

HIV-Prevention Program
HIV/AIDS Agency
717 14th Street NW
Washington, DC 20005
(202) 727-2500

Drug and Alcohol Agency
Department of Human Services
Addiction Prevention and Recovery Administration
1300 First Street NE
Washington, DC 20002
(202) 727-9393

EDUCATION OFFICE

Coordinator For Drug-Free Schools
District of Columbia Public Schools
Substance Abuse Prevention Education Program
Giddings Administrative Unit
315 G Street SE
Washington, DC 20003
(202) 724-3610

Florida

STATE POLICY OFFICES

Governor's Office
Office of the Governor
The Capitol
Tallahassee, FL 32399-0001
(904) 488-4441
http://www.eog.state.fl.us

State Legislative Contact
Division of Legislative Library Services
State Legislature
The Capitol, Room 701
Tallahassee, FL 32399
(904) 488-2812

State Drug Program Coordinator
Policy Coordinator
Governor's Drug Policy Office
1501 The Capitol
Tallahassee, FL 32399-0001
(904) 922-4020

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Legal Affairs
The Capitol, Plaza Level 01
Tallahassee, FL 32399-1050
(904) 487-1963

Law Enforcement Agency
Florida Department of Law Enforcement
P.O. Box 1489
Tallahassee, FL 32302-1489
(904) 488-8771

Law Enforcement Planning
Office of Planning and Budgeting
Carlton Building
Room 415
Calhoun Street
Tallahassee, FL 32301
(904) 488-0090

Crime Prevention Offices
Attorney General's Office
Bureau of Criminal Justice Programs
The Capitol
Tallahassee, FL 32399-1050
(904) 487-3712
http://legal.firn.edu

Statistical Analysis Center
Florida Department of Law Enforcement
2331 Phillips Road, 32208
Tallahassee, FL 32208
(904) 487-4808
E-mail: fsac@freenet.fsu.edu

Uniform Crime Reports Contact
Uniform Crime Reports Section
Florida Crime Information Center
P.O. Box 1489
Tallahassee, FL 32302-1489
(904) 487-1179

BJA Strategy Preparation Agency
Florida Department of Community Affairs
Bureau of Community Assistance
Criminal Justice Section
2555 Shumard Oak Boulevard
Tallahassee, FL 32399-2100
(904) 488-8016
E-mail: wilderc@dca.state.fl.us

Judicial Agency
State Courts Administrator
Supreme Court Building
500 South Duval Street
Tallahassee, FL 32399-1900
(904) 922-5082

Corrections Agency
Department of Corrections
2601 Blairstone Road
Tallahassee, FL 32399-2500
(904) 488-7480

STATE HEALTH OFFICES

RADAR Network Agency
Florida Alcohol and Drug Abuse Association
1030 East Lafayette Street, Suite 100
Tallahassee, FL 32301-4547
(904) 878-2196
E-mail: fadaa@polaris.net

HIV-Prevention Program
Office of Disease Intervention
HIV Patient Care
Building 6, Suite 403
1317 Winewood Boulevard
Tallahassee, FL 32399-0700
(904) 413-0674

Drug and Alcohol Agency
Alcohol and Drug Abuse Program
Alcohol, Drug Abuse and Mental Health Office
Florida Department of HRS
1317 Winewood Boulevard
Tallahassee, FL 32399-0700
(904) 487-2920

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Florida Department of Education
Florida Drug-Free Schools Program
325 West Gaines Street, Suite 322
Tallahassee, FL 32399-0400
(904) 488-6304

Georgia

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, Room 203
Atlanta, GA 30334
(404) 656-1776

State Legislative Contact
House Research
18 Capitol Square, Suite 205A
Atlanta, GA 30334
(404) 656-3206

State Drug Program Coordinator
State Director for Substance Abuse Services
Department of Human Resources
Division of MHMRSA
2 Peachtree Street, Suite 4-550
Atlanta, GA 30303
(404) 657-6400
E-mail: rmurf@dmh.dhr.state.ga.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Law
40 Capitol Square SW
Atlanta, GA 30334-1300
(404) 656-4585

Law Enforcement Planning
Office of the Attorney General
Department of Law
40 Capitol Square SW
Atlanta, GA 30303-1300
(404) 656-4585

Crime Prevention Offices
Georgia Crime Prevention Program
40 Marietta Street NW
Suite 800
Atlanta, GA 30303
(404) 679-4950

Georgia Crime Prevention Association
4400 Memorial Drive
Decatur, GA 30032
(404) 294-2574

Statistical Analysis Center
Statistical Analysis Center
Georgia Criminal Justice Coordinating Council
503 Oak Place, Suite 540
Atlanta, GA 30349
(404) 559-4949

Uniform Crime Reports Contact
Uniform Crime Reports
Georgia Crime Information Center
Georgia Bureau of Investigation
P.O. Box 370748
Decatur, GA 30037
(404) 244-2840

BJA Strategy Preparation Agency
Georgia Criminal Justice Coordinating Council
503 Oak Place, Suite 540
Atlanta, GA 30349
(404) 559-4949

Judicial Agency
Administrative Office of the Courts
State Office Building Annex, Room 550
224 Washington Street SW
Atlanta, GA 30334
(404) 656-5171

Corrections Agency
Department of Corrections
2 Martin Luther King Jr. Drive SE
East Tower
Atlanta, GA 30334
(404) 656-6002

STATE HEALTH OFFICES

RADAR Network Agency
Georgia Prevention Resource Center
Substance Abuse Services Suite 320
2 Peachtree Street, Fourth Floor
Atlanta, GA 30303
(404) 657-21364

HIV-Prevention Program
Epidemiology and Prevention Branch
Division of Public Health
Georgia Department of Human Resources
2 Peachtree Street NW
Atlanta, GA 30303
(404) 657-2700

Drug and Alcohol Agency
Division of Mental Health, Mental Retardation and
Substance Abuse
2 Peachtree Street NW
Atlanta, GA 30303
(404) 657-2135

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Georgia Department of Education
Policy and Communications
1854 Twin Towers East
Atlanta, GA 30334-5040
(404) 651-9406

Hawaii

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol
415 Beretania Street
Honolulu, HI 96813
(808) 586-0034

State Legislative Contact
Department of the Attorney General
425 Queen Street
Honolulu, HI 96813
(808) 548-1282

State Drug Program Coordinator
Department of the Attorney General
425 Queen Street
Honolulu, HI 96813
(808) 548-1282

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Attorney General's Office
Office of the Attorney General
425 Queen Street
Honolulu, HI 96813
(808) 586-1500

Law Enforcement Planning
Crime Prevention and Justice Assistance Division
Department of the Attorney General
425 Queen Street
Honolulu, HI 96813
(808) 586-1150
E-mail: 1koga@lava.net

Statistical Analysis Center
Crime Prevention Division
Department of the Attorney General
City Center Building
810 Richards Street, Suite 701
Honolulu, HI 96813
(808) 586-1416

Uniform Crime Reports Contact
Uniform Crime Reports
Crime Prevention and Justice Assistance Division
Department of the Attorney General
425 Queen Street
Honolulu, HI 96813
(808) 586-1416
E-mail: lkoga@lava.net

BJA Strategy Preparation Agency
Department of the Attorney General
Crime Prevention and Justice Assistance Division
425 Queen Street
Honolulu, HI 96813
(808) 586-1150
E-mail: lkoga@lava.net

Judicial Agency
Hawaii Drug Court Program
Circuit Court of the First Circuit
850 Richards Street
Honolulu, HI 96813
(808) 599-3700

Corrections Agency
Corrections Program Services Division
Department of Public Safety
919 Ala Moana Boulevard, Fourth Floor
Honolulu, HI 96814
(808) 587-1266

STATE HEALTH OFFICES

RADAR Network Agency
Drug-Free Hawaii Prevention Resource Center
425 Queen Street
Honolulu, HI 96813
(808) 545-3228

HIV-Prevention Program
Department of Health
STD/AIDS Branch
3627 Kilawea Avenue, #306
Honolulu, HI 96816
(808) 733-9010

Drug and Alcohol Agency
Health Department
Alcohol and Drug Abuse Division, Room 706
1270 Queen Emma Street
Honolulu, HI 96813
(808) 586-3962

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Hawaii Department of Education
Safe and Drug-Free Schools and Communities Program
Special Programs Management Section, Second Floor
3430 Leahi Avenue, Building D
Honolulu, HI 96815
(808) 733-4496

Idaho

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 83720
Boise, ID 83720-0034
(208) 334-2100
E-mail: governor@40gov.state.id.us

State Legislative Contact
Legislative Services
State Capitol Building
P.O. Box 83720
700 West Jefferson Street
Boise, ID 83720-0054
(208) 334-2475
http://www.state.id.us/legislat/legislat.html

State Drug Program Coordinator
Department of Law Enforcement
P.O. Box 700
Meridian, ID 883680-0700
(208) 884-7000

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 83720
Boise, ID 83720-0010
(208) 334-2400

Law Enforcement Planning
Peace Officer Standards and Training
Department of Law Enforcement
P.O. Box 700
Meridian, ID 83680-0700
(208) 884-7250
E-mail: mbecar@dle.state.id.us

Crime Prevention Office
Idaho Crime Prevention Association
7200 Barrister Driver
Boise, ID 83704
(208) 377-6622

Statistical Analysis Center
Idaho Department of Law Enforcement
Support Services Bureau
700 South Stratford
P.O. Box 700
Meridian, ID 83680-0700
(208) 884-7044
E-mail: ruhlenko@dle.state.id.us

Uniform Crime Reports Contact
Uniform Crime Reports
Idaho Department of Law Enforcement
Bureau of Criminal Identification
P.O. Box 700
Meridian, ID 83680
(208) 884-7156

BJA Strategy Preparation Agency
Idaho Department of Law Enforcement
P.O. Box 700
Meridian, ID 83680-0700
(208) 884-7040
E-mail: rsilva@dle.state.id.us

Judicial Agency
Administrative Director
Office of the Courts
Supreme Court Building
451 West State Street
Boise, ID 83720-0101
(208) 334-2246
E-mail: ptobias@jsc.state.id.us

Corrections Agency
Department of Corrections
P.O. Box 83720
Boise, ID 83720-0018
(208) 334-2318

STATE HEALTH OFFICES

RADAR Network Agency
Idaho RADAR Network Center
Boise State University
1910 University Drive
Boise, ID 83725
(208) 385-3471
E-mail: psawyer@bsu.idbsu.edu

HIV-Prevention Program
Department of Health and Welfare
Bureau of Clinical & Preventive Services
P.O. Box 83720
Boise, ID 83720-0036
(208) 334-6526

Drug and Alcohol Agency
Department of Health and Welfare
Division of Family and Community Services, Fifth Floor
P.O. Box 83720
Boise, ID 83720-0036
(208) 334-5700

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Drug Education Coordinator
Idaho Department of Education
P.O. Box 83720
Boise, ID 83720-0027
(208) 332-6960
E-mail: tbgetty@sde.state.id.us

Illinois

STATE POLICY OFFICES

Governor's Office
Office of the Governor
207 Statehouse
Springfield, IL 62706
(217) 782-7355
E-mail: governor@state.il.us

State Legislative Contact
Legislative Information Bureau
705 Stratton Building
Springfield, IL 62706
(217) 782-3944

State Drug Program Coordinator
Illinois Department of Alcoholism and Substance
Abuse
100 West Randolph Street, Suite 5-600
Chicago, IL 60601
(312) 814-2291
http://www.state.il.us/agency/dhs

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
500 South Second Street
Springfield, IL 62706
(217) 782-1090

Law Enforcement Planning
Illinois Criminal Justice Information Authority
Suite 1016
120 South Riverside Plaza
Chicago, IL 60606
(312) 793-8550

Statistical Analysis Center
Illinois Criminal Justice Information
Authority, Suite 1016
120 South Riverside Plaza
Chicago, IL 60606
(312) 793-8550

Uniform Crime Reports Contact
Uniform Crime Reports
Crime Studies, Illinois State Police
100 Iles Park Place
Springfield, IL 62708
(217) 782-5791
http://www.state.il.us/isp/isphpage.htm

BJA Strategy Preparation Agency
Illinois Criminal Justice Information Authority
Suite 1016
120 South Riverside Plaza
Chicago, IL 60606-3997
(312) 793-8550

Judicial Agency
Administrative Office of the Illinois Courts
160 North LaSalle Street, 18th Floor
Chicago, IL 60601
(312) 793-8191

Corrections Agency
Department of Corrections
P.O. Box 19277
Springfield, IL 62794-9277
(217) 522-2666

STATE HEALTH OFFICES

RADAR Network Agency
Prevention First Inc. Library
720 North Franklin, Suite 500
Chicago, IL 60610
(312) 988-4646
(800) 572-5385 (IL only)

HIV-Prevention Program
Illinois Department of Public Health
AIDS Activity Section
525 West Jefferson Street
Springfield, IL 62761
(217) 524-5983

Drug and Alcohol Agency
Illinois Department of Alcoholism and Substance Abuse
James R. Thompson Center Room 5-600
100 West Randolph Street
Chicago, IL 60601
(312) 814-3840

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Illinois State Board of Education
Grants Management Division
100 North First Street
Springfield, IL 62777
(217) 782-3810

Indiana

STATE POLICY OFFICES

Governor's Office
Office of the Governor
206 State House
Indianapolis, IN 46204
(317) 232-4567
E-mail:
evanbayh@ideanet.doe.state.in.us

State Legislative Contact
Legislative Services Agency
State House, Room 302
Indianapolis, IN 46204
(317) 232-9856

State Drug Program Coordinator
Governor's Commission for a Drug-Free Indiana
Ista Building, Suite 320
150 West Market Street
Indianapolis, IN 46204
(317) 232-4219

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Indiana Government South, Fifth Floor
402 West Washington Street
Indianapolis, IN 46204-2770
(317) 232-6201

Law Enforcement Planning
Indiana Criminal Justice Institute
302 West Washington Street, Room E209
Indianapolis, IN 46204
(317) 232-1233
E-mail: cjilan@2ma.isd.state.in.us

Statistical Analysis Center
Indiana Criminal Justice Institute
302 West Washington Street, Room E209
Indianapolis, IN 46204-2767
(317) 232-1233
E-mail:
smeagher@ideanet.doe.state.in.us

BJA Strategy Preparation Agency
Indiana Criminal Justice Institute
302 West Washington Street, Room E209
Indianapolis, IN 46204
(317) 232-1233
E-mail: cjilan@2ma.isd.state.in.us

Judicial Agency
Administrative Office of the Courts
Supreme Court
115 West Washington, Suite 1080
Indianapolis, IN 46204-3417
(317) 232-2542

Corrections Agency
Department of Correction
E334 Indiana Government Center South
302 West Washington Street
Indianapolis, IN 46204
(317) 232-5766

STATE HEALTH OFFICES

RADAR Network Agency
Indiana Prevention Resource Center for Substance Abuse
Indiana University, Room 110
840 State Road, 46 Bypass
Bloomington, IN 47405
(812) 855-1237
E-mail: drugs@indiana.edu

HIV-Prevention Program
Department of Health
HIV/AIDS Program
1330 West Michigan Street
P.O. Box 1964
Indianapolis, IN 46202-1964
(317) 383-6851

Drug and Alcohol Agency
Bureau for Chemical Addictions
Division of Mental Health
Family and Social Services Administration, Room W353
402 West Washington Street
Indianapolis, IN 46204-2739
(317) 232-7800
E-mail: jmeegan@fssa.state.in.us

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Education
Office of Student Services
State House, Room 229
Indianapolis, IN 46204-2798
(317) 232-9111
E-mail: sdavis@doe.state.in.us

Iowa

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol Building
Des Moines, IA 50319
(515) 281-5211

State Legislative Contact
Legislative Information Office
Legislative Service Bureau
State Capitol Building
Des Moines, IA 50319
(515) 281-4961

State Drug Program Coordinator
Governor's Alliance on Substance Abuse
Lucas State Office Building, Fourth Floor
Des Moines, IA 50319
(515) 281-3784

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Iowa Department of Justice
Second Floor
Hoover State Office Building
Des Moines, IA 50319
(515) 281-5164

Law Enforcement Planning
Department of Public Safety
Division of Criminal Investigation
Wallace State Office Building
Des Moines, IA 50319
(515) 281-6203

Statistical Analysis Center
Division of Criminal Justice and Juvenile Planning
Lucas State Office Building
Des Moines, IA 50319
(515) 242-5816
E-mail: cjjp@max.state.ia.us

Uniform Crime Reports Contact
Uniform Crime Reports
Iowa Department of Public Safety
Wallace State Office Building
Des Moines, IA 50319
(515) 281-8494
E-mail: dps@state.ia.us

BJA Strategy Preparation Agency
Governor's Alliance on Substance Abuse
Lucas State Office Building
Des Moines, IA 50309
(515) 242-6379

Judicial Agency
Administrative Office of the Courts
Supreme Court of Iowa
State House
Des Moines, IA 50319
(515) 281-5241

Corrections Agency
Department of Corrections
Capitol Annex
523 East 12th Street
Des Moines, IA 50319
(515) 281-4811

STATE HEALTH OFFICES

RADAR Network Agency
Iowa Substance Abuse Information Center
Cedar Rapids Public Library
500 First Street SE
Cedar Rapids, IA 52401
(319) 398-5133
E-mail: isaic@crpl.cedar-rapids.lib.ia.us

HIV-Prevention Program
Department of Public Health
Division of Health Protection
Lucas State Office Building
321 East 12th Street
Des Moines, IA 50319
(515) 242-5838

Drug and Alcohol Agency
Department of Public Health
Division of Substance Abuse and Health Promotion
Lucas State Office Building Third Floor
321 East 12th Street
Des Moines, IA 50319
(515) 281-4417

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Substance Education Consultant
Iowa Department of Education
Grimes State Office Building
Des Moines, IA 50319
(515) 281-3021

Kansas

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, Second Floor
Topeka, KS 66612-1590
(913) 296-3232

State Legislative Contact
Legislative Research Department
State House, Room 545-N
Topeka, KS 66612
(913) 296-23181
E-mail: kslegres@lr.01.wpo.state.ks.us

State Drug Program Coordinator
Kansas Criminal Justice Coordinating Council
Jayhawk Tower
700 Southwest Jackson, Suite 501
Topeka, KS 66603
(913) 296-2584

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Kansas Judicial Center
301 Southwest 10th Street
Topeka, KS 66612
(913) 296-2215

Crime Prevention Office
Kansas Bureau of Investigation
Crime Prevention Unit
1620 Southwest Tyler Street
Topeka, KS 66612
(913) 296-8239

Statistical Analysis Center
Kansas Criminal Justice Coordinating Council
Kansas Sentencing Commission
Jayhawk Tower, Suite 501
700 Southwest Jackson
Topeka, KS 66603
(913) 296-0923

Uniform Crime Reports Contact
Crime Data Information Center
Kansas Bureau of Investigation
1620 Tyler Street
Topeka, KS 66612-1837
(913) 296-8200

BJA Strategy Preparation Agency
Kansas Criminal Justice Coordinating Council
700 Southwest Jackson, Room 501
Topeka, KS 66603
(913) 296-0923

Judicial Agency
Administrative Office of the Courts
Kansas Judicial Center
301 West 10th Street
Topeka, KS 66612
(913) 296-4873

Corrections Agency
Department of Corrections
900 Southwest Jackson Street, #451
Topeka, KS 66612-1284
(913) 296-3998

STATE HEALTH OFFICES

RADAR Network Agency
Department of Social and Rehabilitation Services
Biddle Building, Second Floor
300 Southwest Oakley
Topeka, KS 66606-1861
(913) 296-3925

HIV-Prevention Program
AIDS Program
Kansas Department of Health and Environment
109 Southwest Ninth, Suite 605
Topeka, KS 66612-1271
(913) 296-6173

Drug and Alcohol Agency
Kansas Alcohol and Drug Abuse Services
Biddle Building, Second Floor
300 Southwest Oakley
Topeka, KS 66606
(913) 296-3925

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Kansas Department of Education
120 East 10th Street
Topeka, KS 66612
(913) 296-6714

Kentucky

STATE POLICY OFFICES

Governor's Office
Office of the Governor
The Capitol
700 Capitol Avenue
Frankfort, KY 40601
(502) 564-2611

State Legislative Contact
Legislative Research Commission
State Capitol, Room 300
Frankfort, KY 40601
(502) 564-8100

State Drug Program Coordinator
Champions for a Drug Free Kentucky
Capitol City Airport
90 Airport Road, Suite 3
Frankfort, KY 40601
(502) 564-7889
E-mail: lcarrico@mail.state.ky.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 2000
Frankfort, KY 40602-2000
(502) 564-7600

Law Enforcement Planning
Kentucky Justice Cabinet
Law Enforcement Council
403 Wapping Street
Frankfort, KY 40601
(502) 564-3251

Statistical Analysis Center
Office of the Attorney General
Capitol Building
700 Capitol Avenue, Suite 116
Frankfort, KY 40601
(502) 564-7600

Uniform Crime Reports Contact
Uniform Crime Reports
Information Services Branch
Kentucky State Police
1250 Louisville Road
Frankfort, KY 40601
(502) 227-8783

BJA Strategy Preparation Agency
Kentucky Justice Cabinet
Division of Grants Management
Bush Building, Second Floor
403 Wapping Street
Frankfort, KY 40601
(502) 564-7554

Judicial Agency
Administrative Office of the Courts
100 Millcreek Park
Frankfort, KY 40601-9230
(502) 573-2350

Corrections Agency
Department of Corrections
State Office Building, Fifth Floor
Frankfort, KY 40601
(502) 222-9441

STATE HEALTH OFFICES

RADAR Network Agency
Drug Information Service for Kentucky
Division of Substance Abuse
275 East Main Street
Frankfort, KY 40621
(502) 564-2880
(800) 432-9337 (KY only)

HIV-Prevention Program
Cabinet for Health Services
Division of State and Local Administration
STD Control (CTS)
275 East Main Street
Frankfort, KY 40621
(502) 564-4990

Drug and Alcohol Agency
Division of Substance Abuse
Department of Mental Health and Mental Retardation
275 East Main Street
Frankfort, KY 40621
(502) 564-2880

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Department of Education
Division of Program Resources
Title Programs
825 Capitol Plaza Tower
500 Mero Street
Frankfort, KY 40601
(502) 564-3791

Louisiana

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 94004
Baton Rouge, LA 70804-9004
(504) 342-7015

State Legislative Contact
Legislative Research Library
P.O. Box 94012
Baton Rouge, LA 70804
(504) 342-2456
http://www.house.state.la.us

State Drug Program Coordinator
Office of the Attorney General
P.O. Box 94005
Baton Rouge, LA 70804-9005
(504) 339-5192

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Justice
P.O. Box 94005
Baton Rouge, LA 70804-9005
(504) 342-7013

Law Enforcement Planning
Law Enforcement Commission
1885 Wooddale Boulevard, Suite 708
Baton Rouge, LA 70806
(504) 925-4418

Statistical Analysis Center
Louisiana Commission on Law Enforcement and Administration of Criminal Justice Room 708
1885 Wooddale Boulevard
Baton Rouge, LA 70806-1511
(504) 925-4429

Uniform Crime Reports Contact
Uniform Crime Reports
Louisiana Commission on Law Enforcement
1885 Wooddale Boulevard, Seventh Floor
Baton Rouge, LA 70806
(504) 925-4847

BJA Strategy Preparation Agency
Louisiana Commission on Law Enforcement, Room 708
1885 Wooddale Boulevard
Baton Rouge, LA 70806
(504) 925-3513

Judicial Agency
Judicial Administrator
Supreme Court Building
301 Loyola Avenue, Room 109
New Orleans, LA 70112
(504) 568-5747

Corrections Agency
Department of Public Safety and Corrections
P.O. Box 94304
Baton Rouge, LA 70804-9304
(504) 342-6741

STATE HEALTH OFFICES

RADAR Network Agency
Louisiana Office of Alcohol and Drug Abuse
P.O. Box 3868
Baton Rouge, LA 70821-3868
(504) 342-9354

HIV-Prevention Program
HIV/AIDS Services Program
P.O. Box 60630
325 Loyola Avenue
New Orleans, LA 70160
(504) 568-5050

Drug and Alcohol Agency
Office of Alcohol and Drug Abuse
P.O. Box 2790 - Bin 18
Baton Rouge, LA 70821
(504) 342-6717

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Louisiana Department of Education
Bureau of Student Services
P.O. Box 94064
Baton Rouge, LA 70804-9064
(504) 342-3480
E-mail: dfrost@mail.doe.state.la.us

Maine

STATE POLICY OFFICES

Governor's Office
Office of the Governor
1 State House Station
Augusta, ME 04333
(207) 287-3531

State Legislative Contact
Legislative Information
100 State House Station
Augusta, ME 04333
(207) 287-1692

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
6 State House Station
Augusta, ME 04333
(207) 626-8800

Crime Prevention Office
Maine Criminal Justice Academy
Maine Department of Public Safety
93 Silver Street
Waterville, ME 04901
(207) 877-8000

Statistical Analysis Center
Maine Criminal Justice Data Center
Department of Corrections
State House Station #111, Fourth Floor
Augusta, ME 04333
(207) 287-4343
E-mail: colcunn@state.me.us

Uniform Crime Reports Contact
Uniform Crime Reporting Division
Maine State Police
36 Hospital Street, Station #42
Augusta, ME 04333-0042
(207) 624-7004

BJA Strategy Preparation Agency
Department of Public Safety
State House Station #42
Augusta, ME 04333
(207) 624-8758

Judicial Agency
Administrative Office of the Courts
P.O. Box 4820
Portland, ME 04112-4820
(207) 822-0792

Corrections Agency
Department of Corrections
State House Station #111
Augusta, ME 04333
(207) 287-2711
E-mail: joseph.lehman@state.me.us

STATE HEALTH OFFICES

RADAR Network Agency
Office of Substance Abuse
Information Resource Center
State House Station #159
A.M.H.I. Complex
Marguardt Building
Augusta, ME 04333
(207) 287-8900

HIV-Prevention Program
Department of Human Services
State House Station #11
Augusta, ME 04333-0011
(207) 287-37470

Drug and Alcohol Agency
Office of Substance Abuse
State House Station #159
A.M.H.I. Complex
Marguardt Building
Augusta, ME 04333
(207) 287-2595

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Education
Station #161
24 Stone Street
Augusta, ME 04333
(207) 287-4729
E-mail: roger.richards@state.me.us

Maryland

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State House
Annapolis, MD 21404
(410) 974-3901
E-mail: governor@Gov.state.md.us

State Legislative Contact
Department of Legislative Reference
Legislative Services Building
90 State Circle
Annapolis, MD 21401
(410) 841-3886

State Drug Program Coordinator
Governor's Office of Crime and Prevention Control
300 East Joppa Road, Suite 1105
Baltimore, MD 21286-3016
(410) 321-3521

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
200 Saint Paul Place
Baltimore, MD 21202-2020
(410) 576-6300

Crime Prevention Offices
Maryland Community Crime Prevention Institute
Police Training Commission
3085 Hernwood Road
Woodstock, MD 21163
(410) 442-2706
(800) 303-8802

Maryland Crime Prevention Association
PO Box 20397
Baltimore, MD 21284-0397

Statistical Analysis Center
Maryland Justice Analysis Center
Institute of Criminal Justice and Criminology
College of Behavioral and Social Sciences
2220 Samuel J. LeFrak Hall
University of Maryland
College Park, MD 20742-8235
(301) 405-4701
E-mail: cwellford@bss2.umd.edu

Uniform Crime Reports Contact
Uniform Crime Reporting Section
Central Records Division
Maryland State Police Department
1711 Belmont Avenue
Baltimore, MD 21244
(410) 298-3883

BJA Strategy Preparation Agency
Governor's Office of Crime Control and Prevention
300 East Joppa Road, Suite 1105
Baltimore, MD 21286
(410) 321-3521

Judicial Agency
Administrative Office of the Courts
Courts of Appeal Building
361 Rowe Boulevard
Annapolis, MD 21401
(410) 974-2141

Corrections Agency
Division of Correction
Department of Public Safety and Correctional Services
6776 Reisterstown Road, Suite 310
Baltimore, MD 21215-2341
(410) 764-4100

STATE HEALTH OFFICES

RADAR Network Agency
Alcohol and Drug Abuse Administration
Department of Health and Mental Hygiene, Fourth Floor
201 West Preston Street
Baltimore, MD 21201
(410) 225-6916
E-mail: frjones@prevline.health.org

HIV-Prevention Program
AIDS Administration
Department of Health and Mental Hygiene
500 North Calvert Street, Fifth Floor
Baltimore, MD 21202
(410) 767-5132

Drug and Alcohol Agency
Governor's Crime Control and Prevention Commission
300 East Joppa Road, Suite 1105
Baltimore, MD 21286-3016
(410) 321-3521

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Department of Education
Drug-Free Schools Program
200 West Baltimore Street
Baltimore, MD 21201
(410) 767-0301

Massachusetts

STATE POLICY OFFICES

Governor's Office
Executive Office
State House, Room 360
Boston, MA 02133
(617) 727-3600

State Drug Program Coordinator
Governor's Alliance Against Drugs
John W. McCormack State Office Building
One Ashburton Place, Room 611
Boston, MA 02108
(617) 727-0786

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Narcotics and Special Investigations Division
One Ashburton Place, Room 1910
Boston, MA 02108
(617) 727-2200

Law Enforcement Planning
Division of Programs
100 Cambridge Street, Room 2100
Boston, MA 02202
(617) 727-6300

Crime Prevention Offices
Massachusetts Criminal Justice Training Council
Massachusetts Crime Watch
411 Waverly Oaks Road, Suite 325
Waltham, MA 02154
(617) 727-7827

Statistical Analysis Center
Director of Research & Development
Executive Office of Public Safety
100 Cambridge Street, Room 2100
Boston, MA 02202
(617) 727-6300
E-mail: rkohl@state.ma.us

Uniform Crime Reports Contact
Massachusetts State Police
Crime Reporting Unit
470 Worcester Road
Framingham, MA 01701
(508) 820-2110

BJA Strategy Preparation Agency
Massachusetts Committee on Criminal Justice
Executive Office of Public Safety
100 Cambridge Street, Room 2100
Boston, MA 02202
(617) 727-6300
E-mail: jpetuchowski@state.ma.us

Judicial Agency
The Commonwealth of Massachusetts
Administrative Justice of Trial Court
Two Center Plaza, Room 540
Boston, MA 02108
(617) 742-8575

Corrections Agency
Department of Corrections
100 Cambridge Street
Boston, MA 02202
(617) 727-3300

STATE HEALTH OFFICES

RADAR Network Agency
Prevention Support Services
The Medical Foundation
95 Berkeley Street, Suite 201
Boston, MA 02116
(617) 451-0049

HIV-Prevention Program
AIDS Office
Massachusetts Department of Public Health
250 Washington Street
Boston, MA 02108-4619
(617) 624-6000

Drug and Alcohol Agency
Bureau of Substance Abuse
Department of Public Health
250 Washington Street
Boston, MA 02108
(617) 624-5111

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Massachusetts Department of Education
Learning Support Services
350 Main Street
Malden, MA 02148-5023
(617) 388-3300 ext. 415
E-mail: jbynoe@doe.mass.edu

Michigan

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 30013
Lansing, MI 48909
(517) 373-3400
E-mail: migov@aol.com

State Legislative Contact
Legislative Service Bureau
Michigan National Tower Fourth Floor
P.O. Box 30036
Lansing, MI 48909-7536
(517) 373-0170

State Drug Program Coordinator
Office of Drug Control Policy
124 West Allegan
Lansing, MI 48913
(517) 373-4700

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 30212
Lansing, MI 48909
(517) 373-1110

Law Enforcement Planning
Investigative Services Bureau
Michigan State Police
714 South Harrison Road
East Lansing, MI 48823
(517) 336-6531

Crime Prevention Office
Detroit Police Department
Crime Prevention Association
2110 Park Avenue, Suite 332
Detroit, MI 48201
(313) 596-2520

Statistical Analysis Center
Michigan State University School of Criminal Justice
560 Baker Hall
East Lansing, MI 48824-1118
(517) 355-2197
E-mail: tim.bynum@ssc.msu.edu

Uniform Crime Reports Contact
Uniform Crime Reporting Section
Michigan State Police
7150 Harris Drive
Lansing, MI 48913
(517) 322-1150

BJA Strategy Preparation Agency
Office of Drug Control Policy
1200 Michigan National Tower
124 West Allegan
Lansing, MI 48913
(517) 373-4700

Judicial Agency
State Court Administrative Office
309 North Washington Square
P.O. Box 30048
Lansing, MI 48909
(517) 373-0130

Corrections Agency
Michigan Department of Corrections
Grandview Plaza Building
P.O. Box 30003
Lansing, MI 48909
(517) 373-0720

STATE HEALTH OFFICES

RADAR Network Agency
Michigan Resource Center
111 West Edgewood Boulevard, Suite 11
Lansing, MI 48911
(517) 882-9955
E-mail: mrc@voyager.net

HIV-Prevention Program
HIV/AIDS Prevention and Intervention Section
Michigan Department of Public Health
P.O. Box 30035
3500 North Martin Luther King Boulevard
Lansing, MI 48909
(517) 335-8371

Drug and Alcohol Agency
Center for Substance Abuse Services
Michigan Department of Public Health
3423 North Martin Luther King Boulevard
P.O. Box 30195
Lansing, MI 48909
(517) 335-8810

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Community Health
Office of Drug Control Policy
Drug Education Division
124 West Allegan, Suite 1200
Lansing, MI 48913
(517) 373-4700

Minnesota

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Room 130, State Capitol
75 Constitution Avenue
St. Paul, MN 55155-1099
(612) 296-3391
E-mail: governor@state.mn.u

State Legislative Contact
Legislative Reference Library
State Office Building, Room 645
100 Constitution Avenue
St. Paul, MN 55155
(612) 296-3398

State Drug Program Coordinator
Minnesota Department of Public Safety
Office of Drug Policy
444 Cedar Street, 100-D
St. Paul, MN 56101
(612) 297-7311

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
State Capitol, Room 102
St. Paul, MN 55155
(612) 296-6196
E-mail:
attorney.general@state.mn.us

Crime Prevention Offices
Minnesota Crime Watch
Minnesota Department of Public Safety
Bureau of Criminal Apprehension
1246 University Avenue
St. Paul, MN 55104
(612) 643-2576

Statistical Analysis Center
Minnesota Planning Agency
Centennial Office Building
658 Cedar Street, Room 300
St. Paul, MN 55155
(612) 297-7518
E-mail:
susan.roth@mnplan.state.mn.us

Uniform Crime Reports Contact
Uniform Crime Reports
Minnesota Department of Public Safety
Criminal Justice Information Systems 1246
University Avenue
St. Paul, MN 55104
(612) 642-0610

BJA Strategy Preparation Agency
Minnesota Department of Children, Families and Learning
Office of Drug Policy and Violence Prevention
550 Cedar Street, #409
St. Paul, MN 55101
(612) 297-7311
E-mail: jeri.bolsvert@state.mn.us

Judicial Agency
Administrative Office of the Courts
Supreme Court
25 Constitution Avenue
St. Paul, MN 55155
(612) 296-2474

Corrections Agency
Department of Corrections
1450 Energy Park Drive, Suite 200
St. Paul, MN 55108-5219
(612) 642-0282

STATE HEALTH OFFICES

RADAR Network Agency
Minnesota Prevention Resource Center
2829 Verndale Avenue
Anoka, MN 55303
(612) 427-5310
E-mail: mprc@niph.org

HIV-Prevention Program
AIDS/STD Prevention Services Section
Minnesota Department of Health
P.O. Box 9441
717 Southeast Delaware Street
Minneapolis, MN 55440-944
(612) 623-5698

Drug and Alcohol Agency
Chemical Dependency Program Division
Department of Human Services
444 Lafayette Road
St. Paul, MN 55155-3823
(612) 296-4610

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Drug Abuse Program
State Department of Education
550 Cedar Street, Room 976
St. Paul, MN 55101
(612) 296-8023
E-mail: carol.thomas@State.mn.us

Mississippi

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 139
Jackson, MS 39215
(601) 359-3100

State Legislative Contact
Legislative Reference Bureau
P.O. Box 1018
Jackson, MS 39215-1018
(601) 359-3135

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 220
Jackson, MS 39205
(601) 359-3680
E-mail: mmoore@ago.state.ms.us

Statistical Analysis Center
Department of Criminal Justice Planning
401 North West Street, Eighth Floor
P.O. Box 23039
Jackson, MS 39225-3039
(601) 359-7896

BJA Strategy Preparation Agency
Division of Public Safety Planning
Department of Public Safety
401 North West Street, Eighth Floor
P.O Box 23039
Jackson, MS 39201
(601) 359-7880

Judicial Agency
Administrative Office of the Courts
Supreme Court
P.O. Box 117
Jackson, MS 39205
(601) 354-7408

Corrections Agency
Department of Corrections
723 North President Street
Jackson, MS 39202-3097
(601) 359-5600

STATE HEALTH OFFICES

RADAR Network Agency
Mississippi Department of Mental Health
Division of Alcoholism and Drug Abuse
1101 Robert E. Lee Building, 9th Floor
239 North Lamar Street
Jackson, MS 39207
(601) 359-1288

HIV-Prevention Program
Mississippi Department of Health
Division of STD/HIV
P.O. Box 1700
Jackson, MS 39215-1700
(601) 960-7723

Drug and Alcohol Agency
Department of Mental Health
Division of Alcohol and Drug Abuse Services
1101 Robert E. Lee Building
Jackson, MS 39201
(601) 359-1288

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Drug-Free Schools Programs
Mississippi Department of Education
P.O. Box 771, Suite 205
637 North President
Jackson, MS 39205
(601) 359-3793

Missouri

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 720
Jefferson City, MO 65102
(573) 751-3222
E-mail: constit@services.state.mo.u

State Legislative Contact
Committee on Legislative Research
State Capitol, Room 117A
Jefferson City, MO 65101
(573) 751-4223

State Drug Program Coordinator
Department of Public Safety
Truman State Office Building
P.O. Box 749
Jefferson City, MO 65102
(573) 751-5432
E-mail: khiggins@mail.state.mo.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 899
Jefferson City, MO 65102
(573) 751-3321

Law Enforcement Planning
Missouri Department of Public Safety, Room 870
Truman State Office Building
Jefferson City, MO 65102-5432
(573) 751-4905
E-mail: khiggins@mail.state.mo.us

Crime Prevention Offices
Crime Prevention/DARE Unit
Springfield Police Department
2825 South Glenstone F-1
Springfield, MO 65804
(417) 891-1500

Missouri Department of Public Safety
Statewide Crime Prevention Resource Center, Room 870
Truman State Office Building
301 West High Street, P.O. Box 749
Jefferson City, MO 65102
(573) 751-4905
http://www.dps.state.mo.us

Statistical Analysis Center
Information Systems Division
Missouri Highway Patrol
1510 East Elm Street
Jefferson City, MO 65102
(573) 751-4026

BJA Strategy Preparation Agency
Missouri Department of Public Safety, Room 870
Truman State Office Building
P.O. Box 749
Jefferson City, MO 65102-0749
(573) 751-4905
E-mail: khiggins@mail.state.mo.us

Judicial Agency
Office of the State Courts Administrator
Supreme Court
P.O. Box 104480
Jefferson City, MO 65110
(573) 751-3585

Corrections Agency
Department of Corrections
P.O. Box 236
Jefferson City, MO 65102
(573) 751-2389

STATE HEALTH OFFICES

RADAR Network Agency
Missouri Division of Alcohol and Drug Abuse
1706 East Elm Street
P.O. Box 687
Jefferson City, MO 65102
(573) 751-4942

HIV-Prevention Program
Missouri Department of Health
Bureau of STD/AIDS Care
P.O. Box 570
Jefferson City, MO 65102
(573) 751-6107

Drug and Alcohol Agency
Missouri Division of Alcohol and Drug Abuse
Department of Mental Health
1706 East Elm Street
P.O. Box 687
Jefferson City, MO 65102
(573) 751-4942

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Department of Elementary and Secondary Education
P.O. Box 480
Jefferson City, MO 65102
(573) 751-9053
E-mail: sbarr@mail.dese.state.mo.us

Montana

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Capitol Station
Helena, MT 59620-0801
(406) 444-3111

State Legislative Contact
Legislative Services Division
State Capitol, Room 138
Helena, MT 59620-1706
(406) 444-3064
E-mail: sfox@mt.gov

State Drug Program Coordinator
Department of Public Health and Human Services
Addictive & Mental Disorders Division
P.O. Box 202951
1400 Broadway
Helena, MT 59620-2951
(406) 444-3964

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Justice
Justice Building
215 North Sanders Street
Helena, MT 59620
(406) 444-2026

Law Enforcement Planning
Division of Law Enforcement Services
P.O. Box 201417
Helena, MT 59620-1417
(406) 444-3874

Statistical Analysis Center
Board of Crime Control
Montana Department of Justice
303 North Roberts Street, Fourth Floor
Helena, MT 59620
(406) 444-4298
E-mail: tmurphy@mt.gov

Uniform Crime Reports Contact
Uniform Crime Reports
Montana Board of Crime Control
303 North Roberts Street
Helena, MT 59620
(406) 444-2077

BJA Strategy Preparation Agency
Montana Board of Crime Control
Scott Hart Building
303 North Roberts Street
Helena, MT 59620
(406) 444-3604

Judicial Agency
Administrative Office of the Courts
Supreme Court
Justice Building, Room 315
215 North Sanders Street
Helena, MT 59620
(406) 444-2621

Corrections Agency
Department of Corrections
1539 11th Avenue
P.O. Box 201301
Helena, MT 59620-1301
(406) 444-3930

STATE HEALTH OFFICES

RADAR Network Agency
Department of Public Health and Human Services
Addictive & Mental Disorders Division
P.O. Box 202951
1400 Broadway
Helena, MT 59620-2951
(406) 444-3964

HIV-Prevention Program
Montana Department of Public Health and Human
Services - STD/HIV Section
Cogswell Building
P.O. Box 202951
Helena, MT 59620-2951
(406) 444-3565

Drug and Alcohol Agency
Department of Public Health and Human Services
Addictive & Mental Disorders Division
P.O. Box 202951
1400 Broadway
Helena, MT 59620-2951
(406) 444-3964

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Office of Public Instruction
Capitol Building
P.O. Box 202501
Helena, MT 59620-2501
(406) 444-4434
E-mail: jbirch@opi.mt.gov

Nebraska

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 94848
Lincoln, NE 68509-4848
(402) 471-2244

State Legislative Contact
Legislative Research Division
State Capitol
P.O. Box 94945
Lincoln, NE 68509
(402) 471-2221

State Drug Program Coordinator
Governor's Policy Research Office
P.O. Box 94601
Lincoln, NE 68509-4601
(402) 471-2414
E-mail: pro1@pro:state.ne.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Attorney General's Office
Office of the Attorney General
Drug and Violent Crime Unit
2115 State Capitol Building
Lincoln, NE 68509
(402) 471-2682

Law Enforcement Planning
Nebraska Commission on Crime
P.O. Box 94946
Lincoln, NE 68509-4946
(402) 471-2194

Statistical Analysis Center
Nebraska Commission on Law Enforcement and Criminal Justice
301 Centennial Mall South, Third Floor
P.O. Box 94946
Lincoln, NE 68509-4946
(402) 471-2194
E-mail: crime01@vmhost.cdp.state.ne.us

Uniform Crime Reports Contact
Uniform Crime Reporting Section
Nebraska Commission of Law Enforcement and Criminal Justice
P.O. Box 94946
Lincoln, NE 68509
(402) 471-3982

BJA Strategy Preparation Agency
Nebraska Commission on Law Enforcement and Criminal Justice
P.O. Box 94946
Lincoln, NE 68509
(402) 471-3416

Judicial Agency
Administrative Office of the Courts
Supreme Court
State Capitol, Room 1220
Lincoln, NE 68509
(402) 471-3730

Corrections Agency
Department of Correctional Services
P.O. Box 94661
Lincoln, NE 68509-4661
(402) 471-2654

STATE HEALTH OFFICES

RADAR Network Agency
State RADAR Network Center
Nebraska Council to Prevent Alcohol and Drug Abuse
650 J Street, Suite 215
Lincoln, NE 68510
(402) 474-1992
E-mail: nebraskacouncil@ltec.net

HIV-Prevention Program
Department of Health
P.O. Box 95007
Lincoln, NE 68509-5007
(402) 471-2937

Drug and Alcohol Agency
Division on Alcoholism and Drug Abuse
Department of Public Institutions
P.O. Box 94728
Lincoln, NE 68509-4728
(402) 471-2851

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Nebraska State Department of Education
Drug Free Programs
P.O. Box 94987
301 Centenial Mall South
Lincoln, NE 68509-4987
(402) 471-2448

Nevada

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Capitol Complex
Carson City, NV 89710
(702) 687-5670

State Legislative Contact
Legislative Counsel Bureau
401 South Carson Street
Carson City, NV 89710
(702) 687-6800

State Drug Program Coordinator
Chief Legal Counsel
Office of the Governor
Capitol Complex
Las Vegas, NV 89710
(702) 687-6602

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Capitol Complex
198 South Carson Street
Carson City, NV 89710
(702) 687-4170

Law Enforcement Planning
Department of Motor Vehicles and Public Safety
555 Wright Way
Carson City, NV 89711-0900
(702) 687-4412

Crime Prevention Offices
Nevada Crime Prevention Association
P.O. Box 578
Las Vegas, NV 89101
(702) 229-3507

Attorney General's Office
Community Crime Prevention
Capitol Complex
Carson City, NV 89710
(702) 687-4170

Statistical Analysis Center
Records and Identification Services
Nevada Highway Patrol
555 Wright Way
Carson City, NV 89711-0525
(702) 687-5713

Uniform Crime Reports Contact
Criminal Information Services
Nevada Highway Patrol
555 Wright Way
Carson City, NV 89711
(702) 687-5713

BJA Strategy Preparation Agency
Department of Motor Vehicles and Public Safety
Office of Criminal Justice Assistance
107 Jacobsen Way/Stewart Facility
Carson City, NV 89711-0910
(702) 687-5282

Judicial Agency
Administrative Office of the Courts
Capitol Complex
Carson City, NV 89710
(702) 687-5076

Corrections Agency
Department of Prisons
P.O. Box 7011
Carson City, NV 89702
(702) 887-3216

STATE HEALTH OFFICES

RADAR Network Agency
Bureau of Alcohol and Drug Abuse
505 East King Street, Suite 500
Carson City, NV 89710
(702) 687-4790
E-mail: mlwalker@prevline.health.org

HIV-Prevention Program
Nevada State Health Division
505 East King Street, Room 304
Carson City, NV 89710
(702) 687-4800

Nevada AIDS Hotline
505 East King Street, Room 304
Carson City, NV 89710
(800) 842-AIDS
E-mail: nvhotline@aol.com

Drug and Alcohol Agency
Bureau of Alcohol and Drug Abuse
Department of Employment, Training & Rehabilitation
505 East Third Street
Carson City, NV 89713
(702) 687-4790
E-mail: mlwalker@prevline.health.or

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Department of Education
Office of Public Instruction
Capitol Complex
400 West King Street
Carson City, NV 89710
(702) 687-3187

New Hampshire

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State House
107 North Main, Room 208
Concord, NH 03301
(603) 271-2121

State Legislative Contact
Office of Legislative Services
State House, Room 109
107 North Main Street
Concord, NH 03301
(603) 271-3435

State Drug Program Coordinator
Bureau of Substance Abuse Services
State Office Park South
105 Pleasant Street
Concord, NH 03301
(603) 271-6104

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Department of Justice
33 Capitol Street
Concord, NH 03301-6397
(603) 271-365

Law Enforcement Planning
Judicial Council
State House Annex
25 Capitol Street, Room 424
Concord, NH 03301
(603) 271-3592

Statistical Analysis Center
Office of the Attorney General
33 Capitol Street
Concord, NH 03301
(603) 271-3658

Uniform Crime Reports Contact
New Hampshire Department of Public Safety
Division of State Police
Uniform Crime Report Unit
10 Hazen Drive
Concord, NH 03305
(603) 271-2509

BJA Strategy Preparation Agency
Department of Justice
33 Capitol Street
Concord, NH 03301
(603) 271-1297

Judicial Agency
Administrative Office of the Courts
Supreme Court Building
Noble Drive
Concord, NH 03301
(603) 271-2521

Corrections Agency
Department of Corrections
P.O. Box 1806
105 Pleasant Street
Concord, NH 03302

STATE HEALTH OFFICES

RADAR Network Agency
New Hampshire Bureau of Substance Abuse Services
State Office Park South
105 Pleasant Street
Concord, NH 03301
(603) 271-6100
E-mail: mdube@prevline.health.org

HIV-Prevention Program
STD/HIV Program
Division of Public Health Services
Bureau of Disease Control
6 Hazen Drive
Concord, NH 03301
(603) 271-4576

Drug and Alcohol Agency
New Hampshire Bureau of Substance Abuse Services
Department of Health and Human Services
Division of Mental Health & Developmental Services
State Office Park South
105 Pleasant Street
Concord, NH 03301

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Education
State Office Park South
101 Pleasant Street
Concord, NH 03301
(603) 271-2717

New Jersey

STATE POLICY OFFICES

Governor's Office
Office of the Governor
125 State Street, CN 001
Trenton, NJ 08625-0001
(609) 292-6000

State Legislative Contact
Office of Legislative Services
Legislative Information and Bill Room
State House Annex, CN 068
Trenton, NJ 08625
(609) 292-4840
(800) 792-8630 (NJ only)
http://www.njleg.state.nj.us

State Drug Program Coordinator
Office of the Attorney General
Department of Law and Public Safety
CN 080
Trenton, NJ 08625
(609) 292-4925

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Law and Public Safety
Justice Complex, CN 080
Trenton, NJ 08625
(609) 292-4925

Law Enforcement Planning
State Law Enforcement Planning Agency and Coordination Section
State Police
P.O. Box 7068
Trenton, NJ 08628
(609) 882-2000

Crime Prevention Offices
New Jersey Crime Prevention Officers Association
3515 Bargaintown Road
Egg Harbor Two, NJ 08234-8321
(609) 926-4039

Statistical Analysis Center
Research and Evaluation Section
Criminal Justice Division
25 Market Street, CN 085
Trenton, NJ 08625
(609) 984-2737

Uniform Crime Reports Contact
Uniform Crime Reporting
Division of State Police
Box 7068
West Trenton, NJ 08628-0068
(609) 882-2000, ext. 2392

BJA Strategy Preparation Agency
Department of Law and Public Safety
Division of Criminal Justice
25 Market Street, CN 085
Trenton, NJ 08625-0085
(609) 292-5939

Judicial Agency
Administrative Office of the Courts
Hughes Justice Complex, CN 037
Trenton, NJ 08625
(609) 984-0275
E-mail: aoc@ix.netcom.com

Corrections Agency
Department of Corrections
Whittlesey Road
CN 863
Trenton, NJ 08625-0863
(609) 292-4036

STATE HEALTH OFFICES

RADAR Network Agency
New Jersey Department of Health and Senior Services
Division of Addiction Services
129 East Hanover Street, CN-362
Trenton, NJ 08625-0362
(609) 984-6961
E-mail: dadaas@prevline.health.org

HIV-Prevention Program
Department of Health
AIDS Program
50 East State Street, CN369
Trenton, NJ 08625-0369
(609) 984-5874

Drug and Alcohol Agency
Division of Addiction Services
129 East Hanover Street, CN 362
Trenton, NJ 08625-0362
(609) 292-5760

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Manager
New Jersey State Departrment of Education
Division of Student Services
Office of Safe and Drug-Free Schools
100 Riverview Plaza, CN500
Trenton, NJ 08625
(609) 292-0321

New Mexico

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, Room 400
Santa Fe, NM 87501
(505) 827-3000
E-mail: gov@gov.state.nm.us

State Legislative Contact
Legislative Council Service
State Capitol, Room 311
Santa Fe, NM 87503
(505) 986-4600

State Drug Program Coordinator
Cabinet Secretary
Department of Public Safety
P.O. Box 1628
Santa Fe, NM 87504-1628
(505) 827-3370

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Drawer 1508
Santa Fe, NM 87504-1508
(505) 827-6000

Statistical Analysis Center
Institute for Social Research
University of New Mexico
2808 Central Avenue SE
Albuquerque, NM 87106
(505) 277-2501
E-mail: lafree@unm.edu

BJA Strategy Preparation Agency
Office of Grants Management
Department of Public Safety
P.O. Box 1628
Santa Fe, NM 87504-1628
(505) 827-3338
E-mail: dfarrell@ops.state.nm.us

Judicial Agency
Administrative Office of the Courts
Supreme Court Building, Room 25
Santa Fe, NM 87501
(505) 827-4800

Corrections Agency
Department of Corrections
P.O. Box 27116
Santa Fe, NM 87502-0116
(505) 827-8645

STATE HEALTH OFFICES

RADAR Network Agency
Department of Health
Division of Substance Abuse
1190 St. Francis Drive, Room N3200
Santa Fe, NM 87502
(505) 827-2601

HIV-Prevention Program
Department of Health
Public Health Division
AIDS Prevention Program
525 Camino de los Marquez, Suite 1
Santa Fe, NM 87502-6110
(505) 476-8475

Drug and Alcohol Agency
Department of Health
Division of Substance Abuse
P.O. Box 26110
1190 St. Francis Drive
Santa Fe, NM 87502-6110
(505) 827-2601
E-mail: tie@nmbhsdl

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Department of Education
Director of Safe and Drug-Free Schools
120 South Federal Place, Room 206
Santa Fe, NM 87501
(505) 827-1827
E-mail: xfzv65a@prodigy.com

New York

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Executive Chambers, State Capitol
Albany, NY 12224
(518) 474-8390

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Law
State Capitol, Room 220
Albany, NY 12224
(518) 474-7330

Law Enforcement Planning
Commissioner
Division of Criminal Justice Services
Executive Park Tower
Stuyvesant Plaza
Albany, NY 12203-3764
(518) 457-1260

Crime Prevention Offices
New York State Crime Prevention Coalition
563 New Scotland Avenue
P.O. Box 8633
Albany, NY 12208-0633
(518) 344-3748
(800) NYS-CPCC

Statistical Analysis Center
Bureau of Statistical Services
New York State Division of Criminal Justice Services
Executive Park Tower, Eighth Floor
Stuyvesant Plaza
Albany, NY 12203
(518) 457-8381
E-mail: elyr@crisny.org

Uniform Crime Reports Contact
Uniform Crime Reports
Bureau of Statistical Services
New York State Division of Criminal Justice Services
Executive Park Tower Building, Eighth Floor
Stuyvesant Plaza
Albany, NY 12203
(518) 457-8381

BJA Strategy Preparation Agency
New York State Division of Criminal Justice Services
Office of Funding and Program Assistance
Executive Park Tower
Stuyvesant Plaza
Albany, NY 12203-3764
(518) 457-8462

Judicial Agency
Administrative Office of the Courts
270 Broadway, Room 1400
New York, NY 10007
(212) 417-2007

Corrections Agency
Commission on Corrections
Stuyvesant Plaza
Executive Park Tower, Second Floor
Albany, NY 12203-3764
(518) 485-2346

STATE HEALTH OFFICES

HIV-Prevention Program
Department of Health
AIDS Institute
Corning Tower, Room 308
Empire State Plaza
Albany, NY 12237
(518) 473-4229

Drug and Alcohol Agency
Office of Alcohol and Substance Abuse Services
1450 Western Avenue
Albany, NY 12203-8200
(518) 457-2061

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Education Department
Drug-Free Schools & Communiy ACT Program, Room 318-MEB
Washington Avenue
Albany, NY 12234
(518) 486-6090

North Carolina

STATE POLICY OFFICES

Governor's Office
Office of the Governor
116 West Jones Street
Raleigh, NC 27603-8001
(919) 733-5811

State Legislative Contact
Department of Administration
116 West Jones
Raleigh, NC 27603-8003
(919) 733-6887

State Drug Program Coordinator
Department of Crime Control and Public Safety
P.O. Box 29591
Raleigh, NC 27626-0591
(919) 733-2126

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Justice
P.O. Box 629
Raleigh, NC 27602-0629
(919) 733-3377

Law Enforcement Planning
Governor's Crime Commission
3824 Barrett Drive, Room 100
Raleigh, NC 27609
(919) 571-4736
E-mail: markj@gcc.dcc.state.nc.us

Crime Prevention Offices
North Carolina Crime Prevention Division
P.O. Box 29591
Raleigh, NC 27626-0591
(919) 733-5522

Statistical Analysis Center
Criminal Justice Analysis Center
Governor's Crime Commission
3824 Barrett Drive, Suite 100
Raleigh, NC 27609-7220
(919) 571-4736
E-mail: jimk@gcc.dcc.state.nc.us

Uniform Crime Reports Contact
Crime Reporting and Field Services
State Bureau of Investigation
Division of Criminal Information
407 North Blount Street
Raleigh, NC 27601
(919) 733-3171
E-mail: jnipper@mail.jus.state.nc.us

BJA Strategy Preparation Agency
Governor's Crime Commission
3824 Barrett Drive, Suite 100
Raleigh, NC 27609
(919) 571-4736
E-mail: markj@gcc.dcc.state.nc.us

Judicial Agency
Administrative Office of the Courts
P.O. Box 2448
Raleigh, NC 27602
(919) 733-7107

Corrections Agency
Department of Corrections
P.O. Box 29540
Raleigh, NC 27626-0540
(919) 733-4926

STATE HEALTH OFFICES

RADAR Network Agency
North Carolina Alcohol/Drug Resource Center
3109-A University Drive
Durham, NC 27707-3703
(919) 493-2881

HIV-Prevention Program
HIV/STD Control Section Chief
Department of Environment, Health and Natural Resources
Communicable Disease Control
HIV/STD Prevention Branch
P.O. Box 27687
Raleigh, NC 27611-7687
(919) 733-7301

Drug and Alcohol Agency
Department of Public Instruction
Division of School Improvement
Safe and Drug-Free Schools Section
301 North Wilmington Street
Raleigh, NC 27601-2825
(919) 715-1635

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Director
Department of Public Instruction
Division of Alcohol & Drug Defense
210 North Dawson Street
Raleigh, NC 27603-1712
(919) 733-6615

North Dakota

STATE POLICY OFFICES

Governor's Office
Office of the Governor
600 East Boulevard Avenue
Bismarck, ND 58505-0001
(701) 328-2200

State Legislative Contact
Legislative Council
State Capitol
600 East Boulevard Avenue
Bismarck, ND 58505-0360
(701) 328-2916

State Drug Program Coordinator
Division of Mental Health & Alcohol and Drug Abuse
600 South Second Street, Suite 1E
Bismarck, ND 58504-5729
(701) 328-8920

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
600 East Boulevard Avenue
Bismarck, ND 58505-0040
(701) 328-2210

Law Enforcement Planning
Criminal Justice Training and Statistics
Office of the Attorney General
State Capitol
Bismarck, ND 58505
(701) 224-2594

Statistical Analysis Center
Information Services Section
Bureau of Criminal Investigation
4205 State Street
P.O. Box 1054
Bismarck, ND 58502-1054
(701) 328-5514
E-mail: c01125as.judyv@ranch.state.nd.us

Uniform Crime Reports Contact
Uniform Crime Reports
Attorney General's Office
Bureau of Criminal Investigation
P.O. Box 1054
Bismarck, ND 58502-1054
(701) 328-5500

BJA Strategy Preparation Agency
Attorney General's Office
Bureau of Criminal Investigation
P.O. Box 1054
Bismarck, ND 58502
(701) 328-5500

Judicial Agency
Administrative Office of the Courts
Supreme Court
600 East Boulevard Avenue
Bismarck, ND 58505
(701) 328-4216

Corrections Agency
Department of Corrections and Rehabilitation
P.O. Box 1898
Bismarck, ND 58502-1898
(701) 328-6390
E-mail: elittle@pioneer.state.nd.us

STATE HEALTH OFFICES

RADAR Network Agency
North Dakota Prevention Resource Center
Division of Alcohol and Drug Abuse
600 South Second Street, Suite 1-E
Bismarck, ND 58504-5729
(701) 328-8919
E-mail: charolso@sendit.nodak.edu

HIV-Prevention Program
HIV/AIDS Program Manager
Division of Disease Control
North Dakota Department of Health
600 East Boulevard Avenue
Bismarck, ND 58505-0200
(701) 328-2378
(800) 472-2180
E-mail: msmail.pamv@ranch.state.nd.us

Drug and Alcohol Agency
Department of Public Instruction
Drug-Free Schools
State Capitol, Ninth Floor
Bismarck, ND 58505-0440
(701) 328-2254

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Public Instruction
Drug-Free Schools
State Capitol, Ninth Floor
Bismarck, ND 58505-0440
(701) 328-2254

Ohio

STATE POLICY OFFICES

Governor's Office
Office of the Governor
77 South High Street, 30th Floor
Columbus, OH 43266-0601
(614) 644-0813

State Legislative Contact
Legislative Information Office
State House
Columbus, OH 43215
(614) 466-8842

State Drug Program Coordinator
Department of Alcohol and Drug Addiction Services
Two Nationwide Plaza, 12th Floor
280 North High Street
Columbus, OH 43215
(614) 466-3445

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
30 East Broad Street
Columbus, OH 43215-3428
(614) 466-4320

Law Enforcement Planning
Criminal Justice Services Office
400 East Town Street, Suite 120
Columbus, OH 43215
(614) 466-0280

Crime Prevention Office
Ohio Crime Prevention Association
6543 Commerce Parkway, Suite R
Dublin, OH 43017
(614) 761-0500

Statistical Analysis Center
Research and Statistics
Office of Criminal Justice Services
400 East Town Street, Suite 120
Columbus, OH 43215
(614) 466-5126
E-mail: knowles@ocjs.state.oh.us

BJA Strategy Preparation Agency
Governor's Office of Criminal Justice Services
400 East Town Street, Suite 120
Columbus, OH 43215
(614) 466-7782
E-mail: info@ocjs.state.oh.us

Judicial Agency
Administrative Office of the Courts
Supreme Court
30 East Broad Street
Columbus, OH 43266-0419
(614) 466-2653
E-mail: stovers@sconet.ohio.gov

Corrections Agency
Department of Rehabilitation and Correction
1050 Freeway Drive North
Columbus, OH 43229
(614) 752-1162

STATE HEALTH OFFICES

RADAR Network Agency
Department of Alcohol and Drug Addiction Services
Two Nationwide Plaza, 12th Floor
280 North High Street
Columbus, OH 43215-2537
(614) 466-6379

HIV-Prevention Program
Prevention Division
AIDS/STD Prevention Program
Ohio Department of Health
35 East Chestnut Street, Seventh Floor
P.O. Box 118
Columbus, OH 43266-0118
(614) 466-5480

Drug and Alcohol Agency
Department of Alcohol and Drug Addiction Services
280 North High Street, 12th Floor
Columbus, OH 43215-2537
(614) 466-3445
E-mail: ada@state.oh.us

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Ohio Department of Education
Student Development Division
65 South Front Street, Room 611
Columbus, OH 43215-4183
(614) 466-2471
E-mail: sd_airhart@ode.ohio.gov

Oklahoma

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, Room 212
Oklahoma City, OK 73105
(405) 521-2342
E-mail: governor@oklaoss.ok.us

State Drug Program Coordinator
Oklahoma Health Care Authority
4545 North Lincoln Boulevard, Suite 124
Oklahoma City, OK 73105
(405) 530-3439

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
112 State Capitol Building
Oklahoma City, OK 73105
(405) 521-3921

Law Enforcement Planning
Department of Public Safety
P.O. Box 11415
Oklahoma City, OK 73136-1415
(405) 425-2001

Statistical Analysis Center
Oklahoma Criminal Justice Resource Center
5500 North Western, Suite 245
Oklahoma City, OK 73118
(405) 858-7025
E-mail: fferrari@oklaosf.state.ok.us

Uniform Crime Reports Contact
Uniform Crime Reporting Unit
Oklahoma Bureau of Investigation
6600 North Harvey, Suite 300
Oklahoma City, OK 73116
(405) 848-6724

BJA Strategy Preparation Agency
District Attorney's Training and Coordination Council
2200 Classen Boulevard, Suite 1800
Oklahoma City, OK 73106-5811
(405) 557-6707

Judicial Agency
Administrative Office of the Courts
1915 North Stiles Avenue, Suite 305
Oklahoma City, OK 73105
(405) 521-2450

Corrections Agency
Department of Corrections
P.O. Box 11400
Oklahoma City, OK 73136
(405) 425-2505

STATE HEALTH OFFICES

RADAR Network Agency
Oklahoma State Department of Mental Health and Substance Abuse Services, Second Floor
1200 Northeast 13th Street
P.O. Box 53277
Oklahoma City, OK 73117
(405) 522-3810

HIV-Prevention Program
Department of Health
Personal Health Services
HIV/STD Services
1000 Northeast 10th Street
Oklahoma City, OK 73117-1299
(405) 271-4636

Drug and Alcohol Agency
Department of Mental Health and Substance Abuse Services
P.O. Box 53277
Oklahoma City, OK 73152-3277
(405) 522-3908

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Comprehensive Health
Oklahoma Department of Education
2500 North Lincoln Boulevard
Oklahoma City, OK 73105-4599
(405) 521-4507

Oregon

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol Building, Room 254
Salem, OR 97310
(503) 378-3111

State Legislative Contact
Legislative Library
State Capitol, Room 347
Salem, OR 97310
(503) 986-1668

State Drug Program Coordinator
Drug Program Coordinator
Criminal Justice Services Division
Department of State Police
400 Public Service Building
Salem, OR 97310
(503) 378-3720

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Department of Justice
1162 Court Street NE
Salem, OR 97310
(503) 378-6002

Law Enforcement Planning
Division of Civil Enforcement
Office of the Attorney General
Department of Justice
1162 Court Street NE
Salem, OR 97310
(503) 378-4732

Crime Prevention Offices
Oregon Board on Police Standards and Training
Oregon Crime Watch
550 North Monmouth Avenue
Monmouth, OR 97361-0070
(503) 378-2100

Statistical Analysis Center
Oregon Criminal Justice Commission
Statistical Analysis Center
155 Cottage Street NE
Salem, OR 97310
(503) 378-2053
E-mail: phil.m.lemman@state.or.us

Uniform Crime Reports Contact
Law Enforcement Data System Section
Oregon State Police
400 Public Service Building
Salem, OR 97310
(503) 378-3057

BJA Strategy Preparation Agency
Criminal Justice Services Division
Department of State Police
400 Public Service Building
Salem, OR 97310-0310
(503) 378-3720

Judicial Agency
Office of the State Court Administrator
Supreme Court Building
Salem, OR 97310
(503) 986-5500

Corrections Agency
Department of Corrections
2575 Center Street NE
Salem, OR 97310
(503) 945-0920

STATE HEALTH OFFICES

RADAR Network Agency
RADAR Network Agency
Oregon Prevention Resource Center
Office of Alcohol and Drug Abuse Programs
Department of Human Resources
555 24th Place NE
Salem, OR 97310
(503) 378-8000

HIV-Prevention Program
HIV Program Manager
Oregon Department of Human Resources
Health Division
800 Northeast Oregon Street, Suite 745
Portland, OR 97232
(503) 731-4029
E-mail: robert.o.mcalister@state.or.us

Drug and Alcohol Agency
Office of Alcohol and Drug Abuse Programs
500 Summer Street NE, Third Floor
Salem, OR 97310-1016
(503) 945-5763

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Coordinator for Drug-Free Schools
State Department of Education
255 Capitol Street NE
Salem, OR 97310
(503) 378-5585

Pennsylvania

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Main Capitol, Room 225
Harrisburg, PA 17120
(717) 787-2500
E-mail: tridge@gois.state.pa.us

State Legislative Contact
Legislative Reference Bureau
Main Capitol Building, Room 641
Harrisburg, PA 17120
(717) 787-422323

State Drug Program Coordinator
Director of Criminal Justice Policy
Office of the Governor
506 Finance Building
Harrisburg, PA 17120
(717) 787-1854
E-mail: mwoolley@gois.state.pa.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Strawberry Square, 16th Floor
Harrisburg, PA 17120
(717) 787-3391

Law Enforcement Planning
Pennsylvania State Police
Bureau of Drug Law Enforcement
Strawberry Square, 16th Floor
Harrisburg, PA 17120
(717) 783-8514

Crime Prevention Offices
Pennsylvania Commission on Crime and Delinquency
Crime Prevention Division
P.O. Box 1167
Harrisburg, PA 17108-1167
(717) 787-1777
E-mail: willough@pccd.state.pa.us

Statistical Analysis Center
Bureau of Statistics and Policy Research
Pennsylvania Commission on Crime and Delinquency
P.O. Box 1167
Harrisburg, PA 17108
(717) 787-5152
E-mail: renninge@pccd.state.pa.us

Uniform Crime Reports Contact
Uniform Crime Reports
Bureau of Research and Development
Pennsylvania State Police
1800 Elmerton Avenue
Harrisburg, PA 17110
(717) 783-5536

BJA Strategy Preparation Agency
Pennsylvania Commission on Crime and Delinquency
P.O. Box 1167
Harrisburg, PA 17108-1167
(717) 787-2040
E-mail: Thomas@pccd.state.pa.us

Judicial Agency
Administrative Office of Pennsylvania
Supreme Court of Pennsylvania
1515 Market Street, Suite 1414
Philadelphia, PA 19102
(215) 560-6300

Corrections Agency
Department of Corrections
P.O. Box 598
Camp Hill, PA 17001-0598
(717) 975-4860

STATE HEALTH OFFICES

RADAR Network Agency
PennSAHIC
652 West 17th Street
Erie, PA 16502
(814) 459-0245
(800) 582-7746
http://www.pennsahic.org
E-mail: pensahic@moose.erie.net

HIV-Prevention Program
Pennsylvania Department of Health
Bureau of HIV/AIDS
Division of Education and Training
P.O. Box 90, Room 912
Harrisburg, PA 17108
(717) 783-0572

Drug and Alcohol Agency
Office of Drug and Alcohol Programs
Room 933
P.O. Box 90
Harrisburg, PA 17108
(717) 787-8200

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Division of Student Services
Pennsylvania Department of Education
333 Market Street
Harrisburg, PA 17126-0333
(717) 772-2429

Rhode Island

STATE POLICY OFFICES

Governor's Office
Office of the Governor
143 State House
Providence, RI 02903
(401) 277-2080
E-mail: linc01a@prodigy.com

State Legislative Contact
Legislative Council
State House, Room 101
82 Smith Street
Providence, RI 02903
(401) 277-3757

State Drug Program Coordinator
Department of Health
Division of Substance Abuse
Cannon Building, Room 105
3 Capitol Hill
Providence, RI 02908-5097
(401) 277-4680

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
150 South Main Street
Providence, RI 02903
(401) 274-4400

Law Enforcement Planning
Rhode Island State Police
Headquarters
311 Danielson Pike
North Scituate, RI 02850
(401) 444-1000

Crime Prevention Office
Warwick Police Department
99 Veterans Memorial Drive
Warwick, RI 02886
(401) 737-2244

Statistical Analysis Center
Governor's Justice Commission
One Capitol Hill, Fourth Floor
Providence, RI 02908-5803
(401) 277-4499

Uniform Crime Reports Contact
Uniform Crime Reports
Rhode Island State Police
P.O. Box 185
North Scituate, RI 02857
(401) 444-1120

BJA Strategy Preparation Agency
Rhode Island Governor's Justice Commission
222 Quaker Lane
Suite 100
West Warwick, RI 02893
(401) 277-2620

Judicial Agency
Office of the State Court Administrator
Providence County Courthouse
250 Benefit Street
Providence, RI 02903
(401) 277-3263

Corrections Agency
Department of Corrections
40 Howard Avenue
Cranston, RI 02920
(401) 464-2611

STATE HEALTH OFFICES

RADAR Network Agency
Office of Substance Abuse
Cannon Building, Suite 105
Capitol Hill
Providence, RI 02908-5097
(401) 277-4680

HIV-Prevention Program
Department of Health
Disease Prevention and Control
3 Capitol Hill
Providence, RI 02908-5097
(401) 277-1171

Drug and Alcohol Agency
Office of Substance Abuse
Cannon Building, Suite 105
Capitol Hill
Providence, RI 02908-5097
(401) 277-4680

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Rhode Island Department of Education
Safe & Drug-Free Schools and Communities Act
Program
225 Westminster Street, Sixth Floor
Providence, RI 02903-3400
(401) 277-4600 ext.2372
E-mail: ride0039@ride.ri.net

South Carolina

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 11369
Columbia, SC 29211
(803) 734-9818
E-mail: governor@state.sc.us

State Legislative Contact
Code Commissioner and Director
Legislative Council
State House
Columbia, SC 29211
(803) 734-2145

State Drug Program Coordinator
DESIP Project Administrator
South Carolina Department of Public Safety
5400 Broad River Road
Columbia, SC 29210-4088
(803) 896-8708
E-mail: lan@scdps.state.sc.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 11549
Columbia, SC 29211
(803) 734-3970
E-mail:
abcmcondon@ag.state.sc.us

Law Enforcement Planning
South Carolina Law Enforcement Division
P.O. Box 21398
Columbia, SC 29221
(803) 737-9000

Statistical Analysis Center
Department of Public Safety
5400 Broad River Road
Columbia, SC 29210
(803) 896-8717
E-mail:
rfm@mail06.scdps.state.sc.us

Uniform Crime Reports Contact
Uniform Crime Reports
South Carolina Law Enforcement Division
P.O. Box 21398
Columbia, SC 29221-1398
(803) 896-7162

BJA Strategy Preparation Agency
Office of Safety and Grants
Department of Public Safety
5400 Broad River Road
Columbia, SC 29201-4088
(803) 896-8708

Judicial Agency
South Carolina Court Administration
1015 Sumter Street, Second Floor
Columbia, SC 29201
(803) 734-1800

Corrections Agency
Department of Corrections
P.O. Box 21787
Columbia, SC 29221-1787
(803) 896-8555

STATE HEALTH OFFICES

RADAR Network Agency
South Carolina Commission on Alcohol and Drug Abuse
The Drugstore Information Clearinghouse
3700 Forest Drive, Suite 300
Columbia, SC 29204
(803) 734-9559
E-Mail: epeters@prevline.health.org

HIV-Prevention Program
Health and Environmental Control
STD/HIV Division
Jarrett Complex, Box 101106
2600 Bull Street
Columbia, SC 29201
(803) 734-4110

Drug and Alcohol Agency
Department of Alcohol and Other Drug Abuse Services
3700 Forest Drive, Suite 300
Columbia, SC 29204
(803) 734-9520
E-mail: epeters@prevline.health.org

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Safe & Drug-Free Schools and Communities
South Carolina Department of Education
1429 Senate Street, Room 1108
Columbia, SC 29201
(803) 734-8566

South Dakota

STATE POLICY OFFICES

Governor's Office
Office of the Governor
500 East Capitol Avenue
Pierre, SD 57501
(605) 773-3212
E-mail: cathys@gov.state.sd.us

State Legislative Contact
Legislative Research Council
State Capitol Annex
500 East Capitol Avenue
Pierre, SD 57501
(605) 773-3251
E-mail: clarec@lrc.state.sd.us

State Drug Program Coordinator
Office of the Attorney General
500 East Capitol
Pierre, SD 57501
(605) 773-3212

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
State Capitol Building
500 East Capitol Avenue
Pierre, SD 57501-5070
(605) 773-3215

Law Enforcement Planning
Division of Criminal Investigation
500 East Capitol Avenue
Pierre, SD 57501-5070
(605) 773-3331

Statistical Analysis Center
South Dakota Statistical Analysis Center
500 East Capitol Avenue
Pierre, SD 57501-5070
(605) 773-6310
E-mail: wandaf@atg.state.sd.us

Uniform Crime Reports Contact
State Statistical Analysis Center
500 East Capitol Avenue
Pierre, SD 57501
(605) 773-6310

BJA Strategy Preparation Agency
Office of Operations
State Capitol Building
500 East Capitol Avenue
Pierre, SD 57501-5070
(605) 773-6313

Judicial Agency
Administrative Office of the Courts
Unified Judicial System of South Dakota
500 East Capitol Avenue
Pierre, SD 57501
(605) 773-3474

Corrections Agency
Department of Corrections
115 East Dakota
Pierre, SD 57501
(605) 773-3478

STATE HEALTH OFFICES

RADAR Network Agency
Division of Alcohol and Drug Abuse
Hillsview Plaza
500 East Capitol Avenue
Pierre, SD 57501-5070
(605) 773-3123

HIV-Prevention Program
Department of Health
615 East Fourth Street
Pierre, SD 57501
(605) 773-3737
E-mail: davem@doh.state.sd.us

Drug and Alcohol Agency
Division of Alcohol and Drug Abuse
Department of Human Services
Hillsview Plaza
500 East Capitol Avenue
Pierre, SD 57501-5070
(605) 773-4828

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Department of Education
700 Governors Drive
Pierre, SD 57501-3182
(605) 773-4670

Tennessee

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, First Floor
Nashville, TN 37243-0001
(615) 741-2001
E-mail:
dsundquist@mail.state.tn.us

State Legislative Contact
Office of Legislative Information Services
General Assembly
Rachel Jackson Building, First Floor
Nashville, TN 37243
(615) 741-3511

State Drug Program Coordinator
Safe & Drug Free Schools Community Program
Andrew Johnson Tower, Sixth Floor
710 James Robertson Parkway
Nashville, TN 37243-0375
(615) 741-3248

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Enforcement Division
Nashville, TN 20494
(615) 741-4081

Law Enforcement Planning
Department of Safety
1150 Foster Avenue, Room 292
Nashville, TN 37249-1000
(615) 251-5166
E-mail: akimbrou@mail.state.tn.us

Statistical Analysis Center
Tennessee Bureau of Investigation
1148 Foster Avenue
Nashville, TN 37210-4406
(615) 726-7970
E-mail:
jvandercook@mail.state.tn.us

BJA Strategy Preparation Agency
Office of Criminal Justice Programs
Department of Finance and Administration
1400 Andrew Jackson Building
500 Deaderick Street
Nashville, TN 37243-1700
(615) 741-3784

Judicial Agency
Administrative Office of the Courts
Nashville City Center
511 Union Street, Suite 600
Nashville, TN 37243-0607
(615) 741-2687

Corrections Agency
Department of Corrections
320 Sixth Avenue North, Fourth Floor
Nashville, TN 37243-0465
(615) 741-2071
http://www.state.tn.us

STATE HEALTH OFFICES

RADAR Network Agency
Tennessee Alcohol and Drug Association
Statewide Clearinghouse
545 Mainstream Drive, Suite 404
Nashville, TN 37228
(615) 244-7066
1-800-889-9789

HIV-Prevention Program
Department of Health
STD/HIV Program
Health Services Bureau
426 Fifth Avenue North, Fourth Floor
Nashville, TN 37247-4501
(615) 741-7247

Drug and Alcohol Agency
Tennessee Bureau of Alcohol and Drug Abuse Services
Third Floor Cordell Hull
426 Fifth Avenue North
Nashville, TN 37247-0101
(615) 741-1921
E-mail: sperry@mail.state.tn.us

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Tennessee Department of Education
Safe and Drug-Free Schools Program
Andrew Johnson Tower, Sixth Floor
710 James Robertson Parkway
Nashville, TN 37243-0375
(615) 741-3248

Texas

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Capitol Station
P.O. Box 12428
Austin, TX 78711
(512) 463-2000
http://www.governor.state.tx.us

State Legislative Contact
Legislative Council
State Capitol, 1W15
Austin, TX 78711
(512) 463-1151

State Drug Program Coordinator
Texas War on Drugs
7600 Chevy Chase Drive, Suite 115
Austin, TX 78752
(512) 452-0141
E-mail: twod@cris.com

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 12548
Austin, TX 78711-2548
(512) 463-2100
(800) 252-8011 (TX only)
http://www.oag.state.tx.us

Law Enforcement Planning
Criminal Justice Division
Office of the Attorney General
P.O. Box 12548
Austin, TX 78711-2548
(512) 463-2080

Crime Prevention Office
Office of Court Administration of the Texas
Judicial System
P.O. Box 12066
Austin, TX 78711-2066
(512) 463-1625

Statistical Analysis Center
Criminal Justice Policy Council
P.O. Box 13332
Austin, TX 78711-3332
(512) 463-1810
E-mail: cjpc@access.texas.gov

Uniform Crime Reports Contact
Uniform Crime Reporting
Texas Department of Public Safety
P.O. Box 4143
Austin, TX 78765
(512) 424-2091

BJA Strategy Preparation Agency
Criminal Justice Division
Office of the Governor
P.O. Box 12428
Austin, TX 78711
(512) 463-1952
E-mail: rbodisch@governor.texas.gov

Judicial Agency
Administrative Office of the Courts
Tom C. Clark State Courts Building
205 West 14th Street, Suite 600
Austin, TX 78701
(512) 463-1625

Corrections Agency
Criminal Justice Agency
Department of Criminal Justice
P.O. Box 99
Huntsville, TX 77342-0099
(409) 295-6371
E-mail: wscott.@access.texas.gov

STATE HEALTH OFFICES

RADAR Network Agency
Texas Commission on Alcohol and Drug Abuse
9001 North IH 35, Suite 105
Austin, TX 78753-5233
(512) 349-6644
E-mail: mstanfie@tcada.state.tx.us

HIV-Prevention Program
Texas Department of Health
Disease Control and Prevention
HIV/STD Prevention Bureau
1100 West 49th Street
Austin, TX 78756-3199
(512) 490-2505

Drug and Alcohol Agency
Texas Commission on Alcohol and Drug Abuse
9001 North IH 35, Suite 105
Austin, TX 78753-5233
(512) 349-6600

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Safe and Drug-Free Schools and Communities
Coordinator
Texas Education Agency
Division of Accelerated Instruction
1701 North Congress Avenue
Austin, TX 78701-1494
(512) 463-9374

Utah

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol, Room 210
Salt Lake City, UT 84114
(801) 538-1000
E-mail: governor@state.ut.us

State Legislative Contact
Office of Legislative Research and General Counsel
State Capitol, Room 436
Salt Lake City, UT 84114
(801) 538-1032

State Drug Program Coordinator
Commission on Criminal and Juvenile Justice
State Capitol, Room 101
Salt Lake City, UT 84114
(801) 538-1031

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
State Capitol
Room 236
Salt Lake City, UT 84114
(801) 533-5261

Law Enforcement Planning
Utah Department of Public Safety
5272 South College Drive, Room 200
Murray, UT 84123
(801) 284-6240
E-mail: psdomain.psudi.bmunson@state.ut.us

Statistical Analysis Center
101 State Capitol
Salt Lake City, UT 84114
(801) 538-1031
E-mail: jhemenwa@state.ut.us

Uniform Crime Reports Contact
Uniform Crime Reports
Utah Department of Public Safety
4501 South 2700 West
Salt Lake City, UT 84119
(801) 965-4445

BJA Strategy Preparation Agency
Commission on Criminal and Juvenile Justice
State Capitol, Room 101
Salt Lake City, UT 84114
(801) 538-1031
E-mail: jhemenwa@state.ut.us

Judicial Agency
Office of Court Administrator
230 South 500 East, Suite 300
Salt Lake City, UT 84102
(801) 578-3800
E-mail: ericl@aoc.utcourts.gov

Corrections Agency
Department of Corrections
6100 South Fashion Boulevard
Murray, UT 84107
(801) 265-5500
E-mail: crdept.jford@state.ut.us

STATE HEALTH OFFICES

RADAR Network Agency
Utah State Division of Substance Abuse
120 North 200 West, Second Floor
Salt Lake City, UT 84103
(801) 538-3939

HIV-Prevention Program
HIV/AIDS Drug Therapy Program
Utah Department of Health
Bureau of HIV/AIDS
P.O. Box 142867
Salt Lake City, UT 84114-2867
(801) 538-6096

Drug and Alcohol Agency
Division of Substance Abuse
Department of Human Services
P.O. Box 45500
Salt Lake City, UT 84145-0500
(801) 538-3938

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Drug-Free School Coordinator
Utah State Office of Education
Drug-Free Schools Program
250 East 500 South
Salt Lake City, UT 84111
(801) 538-7713

Vermont

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Pavilion Office Building
109 State Street
Montpelier, VT 05609-0101
(802) 828-3333
E-mail: jbagalio@state.vt.us

State Legislative Contact
Legislative Council
State House
115 State Street, Drawer 33
Montpelier, VT 05633-5301
(802) 828-2231

State Drug Program Coordinator
Director of State Police
Department of Public Safety
103 South Main Street
Waterbury, VT 05671-2101
(802) 244-8718
E-mail: jwalton@dps.vt.us

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
109 State Street
Montpelier, VT 05609-1001
(802) 828-3171
E-mail: syoung@ag10.atg.state.vt.us

Law Enforcement Planning
Department of Public Safety
103 South Main Street
Waterbury, VT 05671-2101
(802) 244-8718
E-mail: jwalton@dps.vt.us

Statistical Analysis Center
Vermont Center for Justice Research
33 College Street
Northfield, VT 05602
(802) 828-8511
E-mail: clemmey@norwich.edu

Uniform Crime Reports Contact
Uniform Crime Reports
Support Services
Department of Public Safety
103 South Main Street
Waterbury, VT 05671-2101
(802) 244-8786

BJA Strategy Preparation Agency
Department of Public Safety
Waterbury State Complex
103 South Main Street
Waterbury, VT 05676-0850
(802) 244-8781
E-mail: jwalton@dps.vt.u

Judicial Agency
Office of the Court Administrator
Supreme Court
109 State Street
Montpelier, VT 05609-0701
(802) 828-3278
E-mail: supreme.crt@state.vt.us

Corrections Agency
Department of Corrections
Agency of Human Services
State Complex
103 South Main Street
Waterbury, VT 05671-0201
(802) 241-2263

STATE HEALTH OFFICES

RADAR Network Agency
Office of Alcohol and Drug Abuse Programs
P.O. Box 70
108 Cherry Street
Burlington, VT 05402-0070
(802) 651-1550

HIV-Prevention Program
Department of Health
VD Control Program
P.O. Box 70
108 Cherry Street
Burlington, VT 05402
(802) 863-7245

Drug and Alcohol Agency
Office of Alcohol and Drug Abuse Programs
P.O. Box 70
108 Cherry Street
Burlington, VT 05402-0070
(802) 651-1550

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Education
Safe and Drug-Free Schools & Communities Program
120 State Street
Montpelier, VT 05620-2703
(802) 828-3125
E-mail: smahoney@doe.state.ut.us

Virginia

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Capitol Building, Third Floor
Richmond, VA 23219
(804) 786-2211
E-mail: jerry.kilgore@lms.state.va.us

State Legislative Contact
Division of Legislative Services
General Assembly Building
910 Capitol Street, Second Floor
Richmond, VA 23208
(804) 786-3591

State Drug Program Coordinator
Office of the Secretary of Public Safety
202 North Ninth Street, Suite 613
Richmond, VA 23219
(804) 786-5351

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Commonwealth of Virginia
900 East Main Street
Richmond, VA 23219
(804) 786-2071
E-mail: vaattygen@aol.com

Law Enforcement Planning
State Police
P.O. Box 27472
Richmond, VA 23261-7472
(804) 674-2087

Crime Prevention Offices
Department of Criminal Justice Services
Virginia Crime Prevention Center
805 East Broad Street
Richmond, VA 23219
(804) 786-8467

Virginia Crime Prevention Association, Inc.
4914 Redford Avenue, Suite 306
Richmond, VA 23230
(804) 359-8120

Statistical Analysis Center
Department of Criminal Justice Services
805 East Broad Street
Richmond, VA 23219
(804) 371-0532
E-mail: jmcdonough.dcjs@state.va.us

Uniform Crime Reports Contact
Uniform Crime Reports
Records Management Division
Department of State Police
P.O. Box 27472
Richmond, VA 23261-7472
(804) 674-2023

BJA Strategy Preparation Agency
Department of Criminal Justice Services
805 East Broad Street, 10th Floor
Richmond, VA 23219
(804) 786-1577
E-mail: jmarshall.dcjs@state.va.us

Judicial Agency
Administrative Office of the Courts
Supreme Court
100 North Ninth Street, Third Floor
Richmond, VA 23219
(804) 786-6455

Corrections Agency
Department of Corrections
P.O. Box 26963
Richmond, VA 23261-6963
(804) 674-3119

STATE HEALTH OFFICES

RADAR Network Agency
Office of Prevention
Department of Mental Health
P.O. Box 1797
Richmond, VA 23214
(804) 371-75649

HIV-Prevention Program
Office of Health & Human Resources
Health Department
P.O. Box 2448
Richmond, VA 23218
(804) 786-6267
E-mail: elam.vdh@state.va.us

Drug and Alcohol Agency
Division of Substance Abuse Services
Department of Mental Health, Mental Retardation,
and Substance Abuse Services
P.O. Box 1797
Richmond, VA 23218
(804) 786-3906

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Coordinator for Drug-Free Schools
Virginia Department of Education
Youth Risk Prevention Program
P.O. Box 2120
Richmond, VA 23216-2120
(804) 225-2871

Washington

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Legislative Building, Room AS-13
Olympia, WA 98504-0002
(360) 753-6780
http://www.wa.gov/governor

State Legislative Contact
Office of Program Research
House of Representatives
House Office Building, Room 230
Olympia, WA 98504
(360) 786-7102

State Drug Program Coordinator
Executive Policy Legal Counsel
Office of the Governor
P.O. Box 43113
Olympia, WA 98504-3113
(360) 753-1022

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
P.O. Box 40100
Olympia, WA 98504-0100
(360) 753-6200

Law Enforcement Planning
Research & Planning Section
Budget & Fiscal Services
Washington State Patrol
P.O. Box 42602
Olympia, WA 98504-2602

Statistical Analysis Center
Office of Financial Management
Information and Forecasting Services
Insurance Building
P.O. Box 43113
Olympia, WA 98504-3113
(360) 586-2501
E-mail: glenn@ofm.wa.gov

Uniform Crime Reports Contact
Uniform Crime Reporting Program
Washington Association of Sheriffs and Police Chiefs
P.O. Box 826
Olympia, WA 98507
(360) 586-3221

BJA Strategy Preparation Agency
State Department of Community, Trade and Economic Development
906 Columbia Street SW
P.O. Box 48300
Olympia, WA 98504-8300
(360) 586-0665

Judicial Agency
Administrative Office of the Courts
Supreme Court - Temple of Justice
P.O. Box 41174
Olympia, WA 98504-1174
(360) 357-2121

Corrections Agency
Department of Corrections
P.O. Box 41100
Olympia, WA 98504-1100
(360) 753-2500

STATE HEALTH OFFICES

RADAR Network Agency
Washington State Substance Abuse Coalition, Suite 18
12729 Northeast 20th Street
Bellevue, WA 98005
(206) 637-7011
E-mail: wssac@halcyon.com

HIV-Prevention Program
HIV-AIDS Office of Prevention and Education Services
Airdustrial Park, Building 9
P.O. Box 47890
Olympia, WA 98504-7840
(360) 586-0426

Drug and Alcohol Agency
Division of Alcohol and Substance Abuse
Department of Social and Health Services
Health and Rehabilitative Services
P.O. Box 45330
Olympia, WA 98504-5060
(360) 438-820

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Safe & Drug-Free Schools Program, OSPI
P.O. Box 47200
Old Capitol Building
Olympia, WA 98504-7200
(360) 753-5595

West Virginia

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Capitol Building
1900 Kanawha Boulevard East
Charleston, WV 25305-0370
(304) 558-2000

State Legislative Contact
Legislative Services
State Capitol, Room E-132
Charleston, WV 25305
(304) 347-4830

State Drug Program Coordinator
Department of Public Safety
State Capitol Complex
P.O. Box 50155
Charleston, WV 25305
(304) 348-2930

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Building 1, Room E-26
1900 Kanawha Boulevard East
Charleston, WV 25305-0220
(304) 558-2021

Law Enforcement Planning
Division of Criminal Justice and Highway Safety
Department of Military Affairs and Public Safety
1204 Kanawha Boulevard East
Charleston, WV 25304-0311
(304) 348-8814
E-mail: wvcjhs@citynet.net

Statistical Analysis Center
Criminal Justice & Highway Safety Division
1204 Kanawha Boulevard East
Charleston, WV 25301
(304) 558-8814

Uniform Crime Reports Contact
Uniform Crime Reporting Program
West Virginia State Police
725 Jefferson Road
South Charleston, WV 25309
(304) 746-2159

BJA Strategy Preparation Agency
Criminal Justice and Highway Safety Division
Department of Military Affairs and Public Safety
1204 Kanawha Boulevard East
Charleston, WV 25301
(304) 558-8814
E-mail: wvcjhs@citynet.net

Judicial Agency
Administrative Office of the Courts
Supreme Court of Appeals
E-400 State Capitol Building
1900 Kanawha Boulevard East
Charleston, WV 25305-0833
(304) 558-0145

Corrections Agency
Division of Corrections
State Office Building 4, Room 300
112 California Avenue
Charleston, WV 25305
(304) 558-2036

STATE HEALTH OFFICES

RADAR Network Agency
West Virginia Library Commission
Cultural Center
1900 Kanawha Boulevard East
Charleston, WV 25305-0620
(304) 558-2041

HIV-Prevention Program
Department of Health and Human Resources
Bureau for Public Health
AIDS Program
1422 Washington Street East
Charleston, WV 25301
(304) 558-2195

Drug and Alcohol Agency
Division on Alcoholism and Drug Abuse
Bureau of Community Support
State Office Building 6, Room 717
Charleston, WV 25305
(304) 558-2276

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
State Department of Education
Student Services and Assessment
Capitol Complex, Building 6, B-057
1900 Kanawha Boulevard East
Charleston, WV 25305-0330
(304) 558-2546

Wisconsin

STATE POLICY OFFICES

Governor's Office
Office of the Governor
P.O. Box 7863
Madison, WI 53707-7863
(608) 266-1212
http://www.wisgov.state.wi.us

State Legislative Contact
Reference Staff
Legislative Reference Bureau
P.O. Box 2037
Madison, WI 53701-2037
(608) 266-0341

State Drug Program Coordinator
Alliance for a Drug-Free Wisconsin
1 West Wilson Street, Room 851
Madison, WI 53702
(608) 266-9354

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
114 East, State Capitol
Madison, WI 53707-7857
(608) 266-1221

Law Enforcement Planning
Division of Law Enforcement Services
Department of Justice
P.O. Box 7857
Madison, WI 53707-7858
(608) 266-7751

Crime Prevention Office
Attorney General's Crime Prevention Resource Center
2 East Mifflin, Suite 100
Madison, WI 53703
(608) 267-6736
E-mail: perretzda@doj.state.wi.us

Statistical Analysis Center
Office of Justice Assistance
222 State Street, Second Floor
Madison, WI 53702
(608) 266-7185

Uniform Crime Reports Contact
Uniform Crime Reports
Office of Justice Assistance
222 State Street, Second Floor
Madison, WI 53703
(608) 266-3323

BJA Strategy Preparation Agency
Office of Justice Assistance
222 State Street, Second Floor
Madison, WI 53702
(608) 266-7282
(608) 266-7282

Judicial Agency
Director of State Courts
State Capitol, Room 213 NE
P.O. Box 1688
Madison, WI 53701-1688
(608) 266-6828

Corrections Agency
Department of Corrections
P.O. Box 7925
Madison, WI 53707-7925
(608) 266-4548

STATE HEALTH OFFICES

RADAR Network Agency
Wisconsin Clearinghouse for Prevention Resources
1552 University Avenue
Madison, WI 53705
(608) 262-9157
http://www.uhs.wisc.edu/wch
E-mail: wchpr@www.uhs.wisc.edu

HIV-Prevention Program
AIDS/HIV Program
Bureau of Public Health
Division of Health, Room 96
1414 East Washington Avenue
Madison, WI 53703-3044
(608) 267-5287

Drug and Alcohol Agency
Bureau of Substance Abuse Services
Supportive Living Division
P.O. Box 7851
Madison, WI 53707-7851
(608) 266-3719
E-mail: mcculps@dhfs.state.wi.us

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Department of Public Instruction
Student Services/Prevention and Wellness Team
125 South Webster Street
P.O. Box 7841
Madison, WI 53707-7841
(608) 266-3390
E-mail: trudebk@mail.state.wi.us

Wyoming

STATE POLICY OFFICES

Governor's Office
Office of the Governor
State Capitol Building, Room 124
200 West 24th Street
Cheyenne, WY 82002-0010
(307) 777-7434
E-mail: governor@missc.state.wy.us

State Legislative Contact
Legislative Service Office
State Capitol, Room 213
200 West 24th Street
Cheyenne, WY 82002
(307) 777-788

State Drug Program Coordinator
Governor's State Drug Policy Board
316 West 22nd Street
Cheyenne, WY 82002-0001
(307) 777-7181

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
123 Capitol Building
Cheyenne, WY 82002
(307) 777-7841

Law Enforcement Planning
Office of the Attorney General
123 Capitol Building
200 West 24th Street
Cheyenne, WY 82002
(307) 777-7841

Crime Prevention Office
Wyoming Crime Prevention Coalition
45 West 12th Street
Sheridan, WY 82801
(307) 672-2413

Statistical Analysis Center
Division of Criminal Investigation
Office of the Attorney General
316 West 22nd Street
Cheyenne, WY 82002
(307) 777-7523

Uniform Crime Reports Contact
Uniform Crime Reports
Criminal Justice Information Section
Division of Criminal Investigation
316 West 22nd Street
Cheyenne, WY 82002
(307) 777-7625

BJA Strategy Preparation Agency
Division of Criminal Investigation
316 West 22nd Street
Cheyenne, WY 82002
(307) 777-7181

Judicial Agency
Court Administrator
Wyoming Supreme Court
Supreme Court Building
Cheyenne, WY 82002
(307) 777-7480
E-mail: ajohnson@courts.state.wy.us

Corrections Agency
Department of Corrections
1 East Herschler Building
Cheyenne, WY 82002
(307) 777-7405
E-mail: jlight@missc.state.wy.us

STATE HEALTH OFFICES

RADAR Network Agency
Wyoming CARE Program
Box 3374, University Station
Room 35, College of Education
Laramie, WY 82071-3374
(307) 766-4119

HIV-Prevention Program
Department of Health
Division of Public Health
Hathaway Building, Fourth Floor
Cheyenne, WY 82002-0480
(307) 777-6186

Drug and Alcohol Agency
Office of Substance Abuse
Division of Behavioral Health
451 Hathaway Building
2300 Capitol Avenue
Cheyenne, WY 82002-0480
(307) 777-7094
E-mail: jdefra@missc.state.wy.us

STATE EDUCATION OFFICE

State Coordinator For Drug-Free Schools
Wyoming Department of Education
Hathaway Building
2300 Capitol Avenue
Cheyenne, WY 82002
(307) 777-7168
E-mail: psoumoki@educ.state.wy.us

American Samoa

POLICY OFFICES

Governor's Office
Office of the Governor
Pago Pago, AS 96799
011-684-633-4116

CRIMINAL JUSTICE OFFICES

Attorney General's Office
Department of Legal Affairs
Fagatogo
Pago Pago, AS 96799
011-684-633-4163

Law Enforcement Planning
Criminal Justice Planning Agency
Utulei
Pago Pago, AS 96799
011-684-633-5221

Uniform Crime Reports Contact
Uniform Crime Reports
Department of Public Safety
PO Box 1086
Pago Pago, AS 96799
011-684-633-1111

BJA Strategy Preparation Agency
Government of American Samoa
Office of Legal Affairs
P.O. Box 7
Pago Pago, AS 96799
011-684-633-1838

Judicial Agency
High Court and District Court
Court House
Fagatogo
Pago Pago, AS 96799
011-684-633-4131

Corrections Agency
Department of Public Safety
PO Box 1086
Pago Pago, AS 96799
011-684-633-1111

HEALTH OFFICES

RADAR Network Agency
Department of Human Resources
Social Services Division
Drugs and Alcohol Program
Government of American Samoa
P.O. Box 5051
Pago Pago, AS 96799
011-684-633-2696

HIV-Prevention Program
Government of American Samoa
Department of Public Health
Pago Pago, AS 96799
011-684-633-4606
E-mail: dr_lorac@msn.com

Drug and Alcohol Agency
Division of Social Services
Department of Human Resources
American Samoa Government
Pago Pago, AS 96799
011-684-699-2696

EDUCATION OFFICE

Coordinator For Drug-Free Schools
Department of Education
Drug-Free Schools Program
American Samoa Government
P.O. Box 1923
Pago Pago, AS 96799
011-684-633-5244

Guam

POLICY OFFICES

Governor's Office
Office of the Governor
Executive Chambers
P.O. Box 2950
Agana, GU 96910
011-671-472-8931

CRIMINAL JUSTICE OFFICES

Attorney General's Office
Office of the Attorney General
Prosecution Division
2-200E Judicial Center
120 West O'Brien Drive
Agana, GU 96910
011-671-475-3406
E-mail: smaxwell@ns.gov.gu

Uniform Crime Reports Contact
Uniform Crime Reports
Guam Police Department
Planning, Research and Development
Pedro's Plaza
287 West O'Brien Drive
Agana, GU 96910
011-671-472-8911

BJA Strategy Preparation Agency
Bureau of Planning
The Ricardo J. Bordallo
Governor's Complex
P.O. Box 2950
Agana, GU 96932
011-671-472-4201

Judicial Agency
Administrative Office of the Courts
Superior Court of Guam
Guam Judicial Center
120 West O'Brien Drive
Agana, GU 96910
011-671-475-3544

HEALTH OFFICES

RADAR Network Agency
Department of Mental Health and Substance Abuse
709 Governor Carlos G. Camacho Road
P.O. Box 9400
Tamuning, GU 96911
011-671-647-5441

HIV-Prevention Program
STD/HIV Supervisor
Bureau of Communicable Disease Control Unit
Department of Public Health and Social Services
P.O. Box 2816
Agana, GU 96932
011-671-734-2437

Drug and Alcohol Agency
Drug & Alcohol Treatment Services Branch
Department of Mental Health and Substance Abuse
790 Governor Carlos G. Gamacho Road
Tamuning, GU 96911
011-671-647-5445/5440/5325

EDUCATION OFFICE

Coordinator For Drug-Free Schools
Department of Education
DFSC Coordinator
Guam Public School System
P.O. Box DE
Agana, GU 96910
011-671-472-8524 ext. 307

Northern Mariana Islands

STATE POLICY OFFICES

Governor's Office
Office of the Governor
Capitol Hill
Saipan, MP 96950
(670) 322-5191

STATE CRIMINAL JUSTICE OFFICES

Attorney General's Office
Attorney General
Administration Building
Capitol Hill
Saipan, MP 96950
(670) 322-4311

Law Enforcement Planning
Executive Director
Capitol Hill
P.O. Box 1133
Saipan, MP 96950
(670) 322-9350

Statistical Analysis Center
Criminal Justice Planning Agency
Commonwealth of the Northern Mariana Islands
Criminal Justice Statistical Analysis Center
P.O. Box 1133
Saipan, MP 96950
(670) 664-4550

Uniform Crime Reports Contact
Uniform Crime Report
Director of Public Safety
Civic Center
Saipan, MP 96950
(670) 234-6823

Judicial Agency
Commonwealth Supreme Court
Nauru Building, Second Floor
P.O. Box 2165
Saipan, MP 96950
(670) 234-5175

Corrections Agency
Director of Division of Corrections
Department of Public Safety
Commonwealth of the Northern Mariana Islands
P.O. Box 10007
Saipan, MP 96950
(670) 234-7254

Puerto Rico

POLICY OFFICES

Governor's Office
Office of the Governor
Public Safety Area
P.O. Box 902-0082
San Juan, PR 00902-0082
(787) 721-2840

Legislative Contact
Legislative Reference Library
Office of Legislative Services
P.O. Box 3986
San Juan, PR 00902-3986
(787) 723-4112

CRIMINAL JUSTICE OFFICES

Attorney General's Office
Attorney General
Department of Justice
P.O. Box 902192
San Juan, PR 00902-0192
(787) 721-7700

Law Enforcement Planning
Crime Commission
P.O. Box 82
San Juan, PR 00901
(787) 793-1234

Statistical Analysis Center
Criminal Justice Information Center
Statistical Analysis Center
Office of the Attorney General
601 Olimpo Street, Miramar
P.O. Box 192
San Juan, PR 00902
(787) 729-2445

Uniform Crime Reports Contact
Uniform Crime Reports
Statistics Division
Puerto Rico Police
P.O. Box 70166
San Juan, PR 00936-8166
(787) 793-1234

BJA Strategy Preparation Agency
Attorney General
Department of Justice
Commonwealth of Puerto Rico
P.O. Box 902192
San Juan, PR 00902
(787) 721-7700

Judicial Agency
Office of Court Administration
General Court of Justice
P.O. Box 190917, Hato Rey Station
Hato Rey, PR 00919
(787) 763-3358

HEALTH OFFICES

RADAR Network Agency
Department of Anti-Addiction Services
414 Barbosa Avenue
Hato Rey, PR 00918
(787) 767-5990

Drug and Alcohol Agency
Mental Health, Drug, and Alcohol Agency
Mental Health and Anti-addiction Services
Administration
P.O. Box 21414
San Juan, PR 00928-1414
(787) 764-3670

EDUCATION OFFICE

Coordinator For Drug-Free Schools
Department of Education
Office of Federal Affairs
P.O. Box 759
Hato Rey, PR 00919
(787) 759-8910

Virgin Islands

POLICY OFFICES

Governor's Office
Office of the Governor
Government House
St. Thomas, VI 00802
(340) 774-0001

Legislative Contact
Legislative Counsel's Office
Veterans Drive
Charlotte Amalie
St. Thomas, VI 00801
(340) 774-0739

Drug Program Coordinator
Law Enforcement Planning Commission
Office of the Governor
8172 Sub Base, Suite 3
St. Thomas, VI 00802-5803
(340) 774-6400

CRIMINAL JUSTICE OFFICES

Attorney General's Office
Department of Justice, 48B-50C
Gers Complex, 2nd Floor
St. Thomas, VI 00802
(340) 774-5666

Law Enforcement Planning
Law Enforcement Planning Commission
8172 Sub Base, Suite 3
St. Thomas, VI 00802-5803
(340) 774-6400

Statistical Analysis Center
Law Enforcement Planning Commission
8172 Sub Base, Suite 3
St. Thomas, VI 00802-5803
(340) 774-6400

Uniform Crime Reports Contact
Records Bureau Uniform Crime Reports
Virgin Islands Police Department
Criminal Justice Complex
Charlotte Amalie
St. Thomas, VI 00802
(340) 774-2211

BJA Strategy Preparation Agency
Law Enforcement Planning Commission
Office of the Governor
8172 Sub Base, Suite 3
St. Thomas, VI 00802-5803
(340) 774-6400

Judicial Agency
Administrative Office of the Courts
Territorial Court of the Virgin Islands
P.O. Box 70
Charlotte Amalie
St. Thomas, VI 00804
(340) 774-6680

Corrections Agency
Department of Justice
Bureau of Corrections
3008 Orange Grove
Christian Stead
St. Croix, VI 00820
(340) 773-6309

HEALTH OFFICES

RADAR Network Agency
Division of Mental Health
Prevention Unit
Charles Harwood Hospital Complex, Richmond
St. Croix, VI 00820
(340) 774-7700

HIV-Prevention Program
Department of Health
PO Box 1026
Christiansted
St. Croix, VI 00820
(340) 773-1059

Drug and Alcohol Agency
Division of Mental Health, Alcoholism and Drug
Dependency Services
Charles Harwood Memorial Hospital
Christian Stead
St. Croix, VI 00820
(340) 774-7400

EDUCATION OFFICE

Coordinator For Drug-Free Schools
Department of Education
44-46 Kongens Gade
Charlotte Amalie
St. Thomas, VI 00802
(340) 774-0100

APPENDIX III

State-by-State Directory of Drug Abuse and Alcoholism Treatment and Prevention Programs

INTRODUCTION

This directory is a compilation of U.S. public and private facilities responsible for providing alcoholism and drug-abuse treatment and prevention services. The information was collected by the Substance Abuse and Mental Health Administration (SAMHSA), Office of Applied Studies, and last published in print version in 1998. An online, searchable version is available at http://www.samhsa.gov

The directory is provided as a resource for program managers, treatment personnel, researchers, education officials, parents, and students interested in the location of such facilities. Licensed treatment centers in the Federated States of Micronesia, Guam, Puerto Rico, the Republic of Palau, and the Virgin Islands appear at the end of the list, starting on page 1796. Phone numbers have not been provided since they may change—check your local telephone directory for new listings or contact your information operator service for current listings.

ALABAMA

ALEXANDER CITY

Lighthouse of Tallapoosa County, Inc.
36 Franklin Street
Alexander City, AL 35010

ANNISTON

Anniston Fellowship House, Inc.
106 East 22nd Street
Anniston, AL 36201

Calhoun/Cleburne Mental Health Center New Directions
331 East 8th Street
Anniston, AL 36202

BIRMINGHAM

Alcoholism Recovery Services, Inc.
2701 Jefferson Avenue SW
Birmingham, AL 35211

Aletheia House, Inc.
201 Finley Avenue West
Birmingham, AL 35204

Birmingham Health Care for the Homeless
712 25th Street North
Birmingham, AL 35203

Fellowship House, Inc.
1625 12th Avenue South
Birmingham, AL 35205

Bradford Health Services
Birmingham Regional Office
Jefferson
631 Beacon Parkway West, Suite 211
Birmingham, AL 35209

Department of Veterans Affairs Medical Center
1717 11th Avenue South
Birmingham, AL 35205

Hill Crest Behavioral Health Services
Chemical Dependency Track
6869 5th Avenue South
Birmingham, AL 35212

Jefferson County Economic Opportunity Alcoholism Outreach/Aftercare Program
3040 Ensley Avenue
Birmingham, AL 35208

Oakmont Center
1915 Avenue H
Ensley
Birmingham, AL 35218

Saint Anne's Home, Inc.
2772 Hanover Circle
Birmingham, AL 35205

Tri-County Treatment Center
500 Gene Reed Road, Suite 220
Birmingham, AL 35215

University of Alabama Substance Abuse Programs
401 Beacon Parkway West
Birmingham, AL 35209

University of Alabama in Birmingham Hospital Center for Psychiatric Medicine
1713 6th Avenue South
Birmingham, AL 35294-0018

CALERA

Chilton/Shelby Mental Health Center Substance Abuse Division
1822 17th Street
Highway 25
Calera, AL 35040

CENTRE

Lifeline Services Inc.
Cherokee County Location
423 East Main Street
Centre, AL 35960

CLAYTON

Ventress Correctional Facility Substance Abuse Services
State Road 239
Clayton, AL 36016

DECATUR

Mental Health Center of North Central Alabama
Quest Recovery Center Substance Abuse Treatment
Highway 31 South
Decatur, AL 35601

DEMOPOLIS

West Alabama Mental Health Center Substance Abuse Program
1215 South Walnut Avenue
Demopolis, AL 36732

DOTHAN

Spectra Care
831 John D Odom Road
Dothan, AL 36303

FAIRHOPE

Baldwin County Mental Health Center
372 South Greeno Road
Fairhope, AL 36532

FLORENCE

Riverbend Center for Mental Health
635 West College Street
Florence, AL 35630

GADSDEN

Cherokee/Etowah/De Kalb Mental Health Center Substance Abuse Services
901 Goodyear Avenue
Gadsden, AL 35903

The Bridge, Inc.
3232 Lay Springs Road
Gadsden, AL 35901

GUNTERSVILLE

Marshall/Jackson Mental Health Authority
Mountain Lakes Behavioral Health Care
22165 U.S. Highway 431
Guntersville, AL 35976

HUNTSVILLE

Crestwood Medical Center of Huntsville Behavioral Services
1 Hospital Drive
Huntsville, AL 35801

Huntsville Metro Treatment Center
2227 Drake Avenue
Suite 10-D
Huntsville, AL 35805

Madison County Mental Health Center New Horizons Recovery Center
600 Saint Clair Street
Number 9 Suite 23
Huntsville, AL 3580l

The Pathfinder, Inc.
3104 Ivy Avenue SW
Huntsville, AL 35805

JASPER

Northwest Alabama Mental Health Center
1100 7 Avenue
Jasper, AL 35501

MAXWELL AFB

Maxwell Air Force Base Substance Abuse Program
42MDOS/SGOMH
330 Kirkpatrick Avenue E
Maxwell AFB, AL 36113-6334

MOBILE

Bradford Health Services Mobile Outreach
1000 Hillcrest Road, Suite 304
Mobile, AL 36695

Dauphin Way Lodge
1009 Dauphin Street
Mobile, AL 36604

ECD Program
2950 Springhill Avenue
Mobile, AL 36607

Oasis
4211 Government Blvd.
Mobile, AL 36693

MONTGOMERY

Bradford Health Services Montgomery Outreach
100 Mendel Parkway
Montgomery AL, 36117

Chemical Addictions Program, Inc.
1153 Air Base Boulevard
Montgomery, AL 36108

Jackson Hospital Psychiatric Unit
1235 Forest Avenue
Montgomery, AL 36106

Lighthouse Counseling Center, Inc. Intensive Outpatient Unit
1415 East South Boulevard
Montgomery, AL 36116

Meadhaven Addictive Disease Program
2105 East South Boulevard
Montgomery, AL 36116

Montgomery Metro Treatment Center
4303 Norman Bridge Road
Montgomery, AL 36105

MUSCLE SHOALS

Shoals Treatment Center
520 Louise Street
Muscle Shoals, AL 35661

PELHAM

Bradford Health Services
Oak Mountain Shelby
2280 Highway 35 South
Pelham, AL 35124

PHENIX CITY

Lifeline Services Inc.
Russell County Location
1602 Broad Street
Phenix City, AL 36867

ROANOKE

Self Discovery Inc.
59928 Highway 22
Roanoke AL 36274

ROGERSVILLE

The Freedom House
Route 4
Rogersville, AL 35652

RUSSELLVILLE

Sunrise Lodge Substance Abuse Treatment Center
1163 Washington Avenue SW
Russellville, AL 35653

SELMA

Cahaba Cares Substance Abuse Services
912 Jeff Davis Avenue
Selma, AL 36701

SPANISH FORT

The Shoulder
4901 Battleship Parkway
Spanish Fort, AL 36577

SYLACAUGA

Cheaha Regional Mental Health
Mental Retardation Board Inc.
1721 Old Birmingham Highway
Sylacauga, AL 35150

TUSCALOOSA

Indian Rivers Mental Health Center Alcohol and Drug Abuse Program
505 19 Avenue
Tuscaloosa, AL 35401

Phoenix House, Inc.
700 35 Avenue
Tuscaloosa, AL 35401

Tuscaloosa Treatment Center
535 River Road NE
Suite G3
Tuscaloosa, AL 35404

Veterans Affairs Primary Car Substance Abuse Clinic
3701 Loop Road East
Tuscaloosa, AL 35404

TUSKEGEE

Central Alabama Veteran Health Care System
2400 Hospital Road
Tuskegee, AL 36083

WARRIOR

Bradford Health Services
Warrior Lodge/Jefferson
1189 Allbritton Road
Warrior, AL 35180

WETUMPKA

Bradford Health Services at Elmore Community Hospital
500 Hospital Drive
Wetumpka, AL 36092

ALASKA

ANCHORAGE

Akeela Treatment Services
2805 Bering Street, Suite 4
Anchorage, AK 99503

Alaska Human Services Inc. Outpatient Alcohol/Substance Abuse Treatment Program
4050 Lake Otis Parkway
Suite 111
Anchorage, AK 99508

Alaska North Addictions Recovery Center
4330 Bragaw Street
Anchorage, AK 99508

Booth Memorial Youth and Family Services
3600 East 20th Avenue
Anchorage, AK 99508

Charter North Behavioral Health System
2530 Debarr Road
Anchorage, AK 99508

Genesis House Inc.
2825 West 42nd Place
Anchorage, AK 99517

Narcotic Drug Treatment Center, Inc. Center for Drug Problems
520 East 4th Avenue
Suite 102
Anchorage, AK 99501

Pacific Rim Counseling Inc.
4141 B Street, Suite 210
Anchorage, AK 99503

Providence Alaska Medical Center Project Breakthrough
2401 East 42nd Avenue, Suite 103
Anchorage, AK 99508

RITE Inc.
301 East Fireweed Lane
Suite 102
Anchorage, AK 99503

Salvation Army Clitheroe Center
1709 South Bragaw Street
Point Woronzof/West End Road, Suite B
Anchorage, AK 99503

Southcentral Foundation
Dena A Coy
3916 East 9th Avenue
Anchorage, AK 99508

Veterans Affairs and ROC Anchorage
2925 Debarr Road, Suite 116
Anchorage, AK 99508

Volunteers of America ARCH/ ASSIST
441 West 5th Street
Suite 301
Anchorage, AK 99501

ANIAK

Kuskokwim Native Association Community Counseling Program
P.O. Box 155
Aniak, AK 99557

BARROW

North Slope Borough Health Substance Abuse Treatment Services
579 Kingosak Street
Barrow, AK 99723

COPPER CENTER

Copper River Mental Health Center Substance Abuse Services
Mile 104 Old Richardson Highway
Copper Center, AK 99573

CORDOVA

Sound Alternatives
Cordova Community Medical Center
602 Chase Avenue
Cordova, AK 99574

DILLINGHAM

Bristol Bay Area Health Corporation Alcohol/Drug Abuse Program
Dillingham, AK 99576

DUTCH HARBOR

Aleutian Counseling Center
Dutch Harbor, AK 99692

EAGLE RIVER

Volunteers of America of Alaska ARCH
HC 85
Eagle River, AK 99577

EIELSON AFB

Eielson Air Force Base Substance Abuse Program
354 MDOS/SGOMH
3349 Central Avenue, Suite 1
Eielson AFB, AK 99702-2325

ELMENDORF AFB

Elmendorf AFB Substance Abuse Office
3 MDOS/SGOMH
24800 Hospital Drive
Elmendorf AFB, AK 99506

FAIRBANKS

Fairbanks Memorial Hospital Family Recovery Center
1650 Cowles Street
Fairbanks, AK 99701

Graf Rheeneenhaanjii Substance Abuse Services
2550 Lawlor Road
Fairbanks, AK 99701

Regional Center for Alcohol and other Addictions
3100 South Cushman Street
Fairbanks, AK 99707

Tanana Chiefs Conference Inc. Old Minto Recovery Camp
1221 1st Avenue, Suite 600
Fairbanks, AK 99707

Tanana Chiefs Conference, Inc. Ukon Tanana Counseling Servces
1302 21st Avenue
Fairbanks, AK 99701

FORT RICHARDSON

Community Counseling Center
600 Richardson Drive
Fort Richardson, AK 99505

FORT WAINWRIGHT

Fort Wainwright Alcohol and Drug Abuse Prevention Control Program (ADAPCP)
1060 Gaffney Road, Suite 6600
MCUC/CCC Building 1064
Fort Wainwright, AK 99703

HEALY

Railbelt Mental Health and Addiction
Dry Creek and Coal Street
Healy, AK 99743

JUNEAU

Gastineau Human Services
5597 Aisek Street
Juneau, AK 99801

Tongass Community Counseling Center
222 Seward Street, Suite 202
Juneau, AK 99801

KENAI

Cook Inlet Council on Alcohol and Drug Abuse
10200 Kenai Spur Highway
Kenai, AK 99611

Kenaitze Indian Tribe
Nakenu
150 North Willow Street
Kenai, AK 99611

KETCHIKAN

Gateway Center for Human Services Substance Abuse Services Division
3050 5th Avenue
Ketchikan, AK 99901

Ketchikan General Hospital Recovery Center
126 Washington Street
Ketchikan, AK 99901

Ketchikan Indian Corporation
355 Carlanna Lake Road
Ketchikan, AK 99901

KODIAK

Kodiak Council on Alcoholism, Inc.
115 Mill bay Road
Kodiak, AK 99615

KOTZEBUE

Maniilaq Addictions and Support
Maniilaq Association
Frank Ferguson Building
D and E Wings
Kotzebue, AK 99752

MCGRATH

Four Rivers Counseling Services
McGrath/Anvik Education MH Association
229 Joaquin Street
McGrath, AK 99627

METLAKATLA

Annette Island Service Unit
Family Services
Metlakatla, AK 99926

NOME

Northern Lights Recovery Center
5 Avenue and Division Streets
Community Health Services Building
Nome, AK 99762

PETERSBURG

Changing Tides Counseling Services
201 North Nordic Street
Suites 204 and 205
Petersburg, AK 99833

SAINT PAUL ISLAND

Pribilof Counseling Center
Saint Paul Island, AK 99660

SAND POINT

Eastern Aleutian Tirbes Inc.
Main Street
Sand Point, AK 99661

SELDOVIA

Seldovia Village Tribe/SKIAP
274 Main Street
Seldovia, AK 99663-0197

SEWARD

Seward Life Action Council
504 Adams Street
Seward, AK 99664

SITKA

Ravens Way/SEARHC Adolescent Residential Treatment Program
222 Tongass Drive
Sitka, AK 99835

Sitka Prevention and Treatment Services Inc.
509 Lincoln Street
Sitka, AK 99835

Southeast Alaska Regional Health Consortium Community Family Services Program
222 Tongass Avenue
Sitka, AK 99835

TOK

Upper Tanana Alcohol Program
Tok Clinic Building
Tok Cut-Off
Tok, AK 99780

VALDEZ

Valdez Counseling Center
337 Egan Avenue
Valdez, AK 99686

WASILLA

Alaska Addiction Rehab Services
Nugens Ranch
3701 Palmer-Wasilla Highway
Wasilla, AK 99687

MAT/SU Recovery Center Inc.
2801 Bogard Road
Wasilla, AK 99654

WRANGELL

Avenues
406 Alaska Avenue
Wrangell, AK 99929

AMERICAN SAMOA

PAGO PAGO

LBJ Tropical Medical Center Alcohol and Drug Program Human Services Clinic
Pago Pago, AS 96799

ARIZONA

APACHE JUNCTION

SMMHC Inc. Mental Health Center/Substance Abuse Services
564 North Idaho Road, Suite 9
Apache Junction, AZ 85220

BENSON

Southeastern Arizona Behavioral Health Services (SEABHS)
Administrative Unit
590 South Ocotillo Avenue
Benson, AZ 85602

BULLHEAD CITY

Mohave Mental Health Clinic Outpatient and Day Treatment
2135 Highway 95
Suites 125 and 241
Bullhead City, AZ 86442

CAMP VERDE

Camp Verde Yavapai/Apache Alcohol/Substance Abuse Program
Camp Verde, AZ 86322

Verde Valley Guidance Clinic Inc.
497 Main Street, Suite 4
Camp Verde, AZ 86322

CASA GRANDE

Against Abuse Inc.
Casa Grande, AZ 95230-0733

Behavioral Health Agency of Central Arizona
120 West Main Street
Casa Grande, AZ 85222-4820

PGBHA BHACA Casa Grande Outpatient
120 West Main Street
Casa Grande, AZ 85222

PGBHA Helping Associates Inc.
1901 North Trekell Road, Suite A
Casa Grande, AZ 85222

CHANDLER

Centro de Amistad Inc
100 West Boston Street, Suite 5
Chandler, AZ 85224

Chandler Valley Hope and Drug Treatment Center
501 North Washington Street
Chandler, AZ 85225

COTTONWOOD

Verde Valley Guidance Clinic, Inc.
19 East Beech Street
Cottonwood, AZ 86326

DILKON

Dilcon Agency
Department of Behavioral Health Services
Old Preschool Building
Next Chapter House
Dilkon, AZ 86047

ELOY

PGBHA Pinal County Hispanic Council
712 North Main Street
Eloy, AZ 85231

FLAGSTAFF

Aspen Hill Behavioral Health Systems
305 West Forest Avenue
Flagstaff, AZ 86001

Community Medical Services
2559 East 7th Avenue
Flagstaff, AZ 86004

The Guidance Center
2187 North Vickey Street
Flagstaff, AZ 86004

GLENDALE

Community Care Network (CCN) Jewish Family and Children's Services
6376 West Bell Road
Glendale, AZ 85308

Maverick House
7022 North 48th Avenue
Glendale, AZ 85301

Thunderbird Samaritan Behavioral Health
5555 West Thunderbird Road
Glendale, AZ 85306

GREEN VALLEY

La Frontera Center
BLNA
1151 South La Canada
Suite 105
Green Valley, AZ 85614

HOLBROOK

Community Counseling Centers, Inc.
105 North 5th Avenue
Holbrook, AZ 86025

KAYENTA

Kayenta outpatient Treatment Center
Kayenta, AZ 86033

KEARNY

Copper Community Resource and Development Inc.
1116 Tilbury Street
Kearny, AZ 85237

KINGMAN

Mohave Mental Health Clinic Substance Abuse Services
1750 Beverly Street
Kingman, AZ 86401

LAKE HAVASU CITY

Mohave Mental Health Clinic
Substance Abuse Services
2187 Swanson Street
Lake Havasu City, AZ 86403

LUKE AFB

Luke Air Force Base Substance Abuse Program
52 MDOS/SGOMH
7219 North Litchfield Road
Luke AFB, AZ 85309-1525

Mental Health Clinic Substance Abuse Control
56 MDOS/SGOMH
7219 North Litchfield Road
Luke AFB, AZ 85309-1525

MESA

Centro de Amistad, Inc. Mesa Office
734 East Broadway
Suite C
Mesa, AZ 85204

East Valley Addiction Council Inc.
554 South Bellview Street
Mesa, AZ 85204

East Valley Catholic Social Services
430 North Dobson Road
Mesa, AZ 85204

New Hope Behavioral Health Center, Inc.
6550 Broadway
Suite 101
Mesa, AZ 85208

Prehab of Arizona, Inc.
Center for Family Enrichment
1655 East University Drive
Mesa, AZ 85203

Helaman House
2613 South Power Road
Mesa, AZ 85206

Homestead Residence
1131 East University Drive
Mesa, AZ 85203

Samaritan Behavioral Health Desert
2225 West Southern Avenue
Mesa, AZ 85202

Women in New Recovery
540 West 1st Street
Mesa, AZ 85202

MORENCI

SEABHS
Administrative Office
Burro Alley and Coronado Boulevard
Morenci, AZ 85540

NOGALES

Santa Cruz Family Guidance Center Division Inc.
489 North Arroyo Boulevard
Nogales, AZ 85621

ORACLE

PGBHA Tri Community Resource Center
98 Mount Lemmon Road
Oracle, AZ 85623

PAGE

Kaibeto Outpatient Treatment Center
DBHS
337 North Navajo Street
Page, AZ 86040

PARKER

Colorado River Indian Tribes Behavioral Health Services
Route 1
Parker, AZ 85344

New Life Guidance Center
1200 Arizona Avenue
Parker, AZ 85344

Public Health Service
Indian Hospital Substance Abuse Services
Route 1
Parker, AZ 85344

PAYSON

Rim Guidance Center
404 West Aero Drive
Payson, AZ 85547

PEACH SPRINGS

Hualapai Health Department Alcoholism and Drug Abuse Program
960 Rodeo Way
Peach Springs, AZ 86434

PHOENIX

Behavioral Systems Southwest
2846 East Roosevelt Street
Phoenix, AZ 85008

Calvary Rehabilitation Center
720 East Montebello Avenue
Phoenix, AZ 85014

Carl T Hayden VA Medical Center Substance Abuse Treatment Program
650 East Indian School Road, Suite 11-A9
Phoenix, AZ 85012

Casa De Amigas
1648 West Cotler Street, Suite 8
Phoenix, AZ 85015

Chicanos por la Causa, Inc.
Centro de la Familia
4622 West Indian School Road, Suite D-12
Phoenix, AZ 85009

Community Medical Services
Larkspur Medical Center
12426 North 28th Drive
Phoenix, 85029

Corazon/CPLC
3639 West Lincoln Street
Phoenix, AZ 85009

Crossroads
1845 East Ocotillo Road
Phoenix, AZ 85016

Drug and Alcohol Treatment Institute Clarence Lawson Foundation
2230 North 24th Street
Phoenix, AZ 85008

Ebony House, Inc.
6222 South 13th Street
Phoenix, AZ 85040

Family Service Agency
1530 East Flower Street
Phoenix, AZ 85014

Hohokam Room
1501 East Washington Street
Phoenix, AZ 85034

Indian Rehabilitation, Inc.
650 North 2nd Avenue
Phoenix, AZ 85003

Intensive Treatment Systems Inc.
651 West Coolidge Street
Phoenix, AZ 85013

Jewish Family and Children's Service
2033 North 7th Street
Phoenix, AZ 85006

National Council on Alcohol and Drug Dependency/ Central and Northern Arizona
2701 North 16 Street
Suite 103
Phoenix, AZ 85006

New Arizona Family I
3301 East Pinchot Avenue
Phoenix, AZ 85018

New Arizona Family II SMI Dial Diagnosis Program
302 East Southern Avenue
Phoenix, AZ 85040

New Life for Girls
6216 North 27th Avenue
Phoenix, AZ 85017

Phoenix LARC Meta Center Public Inebriate Program
2770 East Van Buren Street
Phoenix, AZ 85008

Phoenix Indian Center
2601 North 3rd Street
Suite 100
Phoenix, AZ 85004

Progress Valley Phoenix
4430 North 23rd Avenue
Phoenix, AZ 85015

Saint Luke's Behavioral Health Center
1800 East Van Buren Street
Phoenix, AZ 85006

Salvation Army Recovery Center
2707 East Van Buren Street
Treatment Center
Phoenix, AZ 85008

Adult Rehabilitation Center
1625 South Central Avenue
Phoenix, AZ 85004

Southwest Behavioral Health Inc.
1714 East Broadway
Phoenix, AZ 85040

1424 South 7th Avenue
Phoenix, AZ 85007

5116 East Thomas Road
Phoenix, AZ 85018

Terros, Inc.
320 East Virginia Street
Phoenix, AZ 85004

Treatment Assessment Screening Center
TASC Inc.
2234 North 7th Street, Suite A
Phoenix, AZ 85006

Valle Del Sol, Inc. Behavioral Health Program
1209 South First Avenue
Phoenix, AZ 85003

PRESCOTT

Veterans' Affairs Medical Center
500 Highway 89 North, Room 208
Prescott, AZ 86313

West Yavapai Guidance Clinic
Hillside Center
642 Dameron Drive
Prescott, AZ 86301

Yavapai/Prescott Tribe Social Services Department
530 East Merritt Street
Prescott, AZ 86301

SACATON

Gila River Indian Community Alcohol and Drug Abuse Program
315 West Casa Blanca Road
Sacaton, AZ 85247

SAFFORD

SEABHS Graham/Greenlee Counseling Center
Safford Outpatient
620 Central Avenue
Safford, AZ 85546

SAINT JOHNS

Little Colorado Behavioral Health Center, Inc.
470 West Cleveland Street
Saint Johns, AZ 85936

SAN CARLOS

San Carlos Apache Tribe Alcohol Program
San Carlos, AZ 85550

SCOTTSDALE

Jewish Family and Children's Services
7770 East Roosevelt Street
Scottsdale, AZ 85257

New Foundation
Scottsdale, AZ 85271

Salt River Pima/Maricopa Behavioral Health Program
10005 East Osborn Road
Scottsdale, AZ 85256

Samaritan Behavioral Health
7575 East Earl Drive
Scottsdale, AZ 85251

Teen Ranch
5718 East Sharron Drive
Scottsdale, AZ 85254

SECOND MESA

Hopi Behavioral Health and Social Services Program
Second Mesa, AZ 86043

SEDONA

Verde Valley Guidance Clinic Inc.
2880 Hopi Drive
Sedona, AZ 86336

SELLS

Tohono Oodham Human Services Alcoholism and Substance Abuse Branch
Sells, AZ 85634

SHOW LOW

Community Counseling Centers
Outpatient Unit
2350 Show Low Lake Road
Show Low, AZ 85901

SIERRA VISTA

SEABHS Coronado Behavioral Health Division Sierra Vista Outpatient
185 South Moorman Street
Sierra Vista, AZ 85635

SOMERTON

Cocopah Alcohol and Drug Abuse Prevention Program
County 15th and Avenue G
Somerton, AZ 85350

SPRINGERVILLE

Little Colorado Behavioral Health Center, Inc.
50 North Hopi Street
Springerville, AZ 85938

TEMPE

Center for Behavioral Health
Special Services
2123 East Southern Avenue
Suite 2
Tempe, AZ 85282

Contact
1400 East Southern Street
Suite 301
Tempe, AZ 85282

Tempe Saint Luke's Hospital
1500 South Mill Avenue
Tempe, AZ 85281

Valle Del Sol, Inc. East Clinic
509 South Rockford Drive
Tempe, AZ 85281

TUBA CITY

Tuba City Outpatient Treatment Center Behavioral Health Services/DBHS
Main Street
Building 25
Tuba City, AZ 86045

TUCSON

Amity, Inc.
10500 East Tanque Verde Road
Tucson, AZ 85749

CODAC Behavioral Health Services of Pima County, Inc.
CODAC Counseling Center
333 West Fort Lowell Street
Tucson, AZ 85705

Wildflowers
700 North 7th Avenue
Tucson, AZ 85705

Compass Health Care
2475 North Jack Rabbit Street
Tucson, AZ 85745

Cope Behavioral Services
101 South Stone Street, Suite 200
Tucson, AZ 85745

Cottonwood de Tucson
4110 Sweetwater Drive
Tucson, AZ 85745

Davis Monthan Air Force Base ADPB
355 MDOS/SGOHA
Tucson, AZ 85707

Haven, Inc.
1107 East Adelaide Drive
Tucson, AZ 85719

La Frontera Center
East Clinic
2222 North Craycroft Road
Suite 120
Tucson, AZ 85712

Hope Center
260 South Scott Street
Tucson, AZ 85701

Substance Abuse Outpatient Service
502 West 29th Street
Tucson, AZ 85711

Mark Youth and Family Care Campus Inc.
4653 East Pima Street
Tucson, AZ 85712

Pascua Yaqui Chemical Dependency Program
7490 south Camino De Oeste
Tucson, AZ 85746

Portable Prep Behavioral Health Services Alcoholism Treatment Unit
806 East 46th Street
Tucson, AZ 85713

Saint Josephs Hospital O'Reilly Care Center
350 North Wilmont Road
Tucson, AZ 8711

Salvation Army Adult Rehabilitation Center
2717 South 6th Avenue
Tucson, AZ 85713

Sierra Tucson LLC
Lago Del Oro Parkway
Tucson, AZ 85739

Tucson Alcoholic Recovery Home, Inc.
1809 East 23 Street
Tucson, AZ 85713

Veterans Affairs Medical Center Substance Abuse Program
3601 South 6th Avenue
Tucson, AZ 85723

Westcenter
2105 East Allen Road
Tucson, AZ 85719

WHITERIVER

Rainbow Center
White Mountain Apache Tribe
White River, AZ 85941

WICKENBURG

Meadows Holdings
1655 North Tegner Road
Wickenburg, AZ 85390

WILLIAMS

The Guidance Center, Inc.
301 South 7th Street
Williams, AZ 86046

WINSLOW

Community Counseling Centers Outpatient Clinic
211 East 3rd Street
Winslow, AZ 86047

Navajo Nation Dilkon Outpatient Treatment Center
Dilkon Chapter House
Winslow, AZ 86047

ARKANSAS

ARKADELPHIA

Quapaw House, Inc.
401 Crittenden Street
Arkadelphia, AR 71923-6139

BENTON

Counseling Clinic, Inc. Outpatient Abuse Program
307 East Servier Street
Benton, AR 72015

Department of Corrections Benton Unit Substance Abuse Treatment Program
6701 Highway 67
Benton, AR 72015

BRICKEYS

East Arkansas Regional Unit
Route 1 Highway 131
Brickeys, AR 72320

CALICO ROCK

SATP North Central Unit
HC 62
Calico Rock, AR 72519

CAMDEN

Ouachita County Hospital Chemical Dependency Unit
638 California Street
Camden, AR 71701

CLARKSVILLE

Counseling Associates
1021 Poplar Street
Clarksville, AR 72830-4428

CONWAY

Counseling Associates, Inc.
350 Salem Road Suite 1
Conway, AR 72032-6135

DERMOTT

Department of Corrections Delta Reg Unit Substance Abuse Treatment Program
880 East Gaines Street
Dermott, AR 71638

EL DORADO

South Arkansas Regional Health Center Recovery Center
710 West Grove Street
El Dorado, AR 71730

FAYETTEVILLE

Charter Behavioral Health Systems of Northwest Arkansas
4253 Crossover Road
Fayetteville, AR 72703

Veterans Affairs Medical Center
1100 North College Street 116-A
Fayetteville, AR 72703

FORT SMITH

Gateway House, Inc. June Bailey Center
3900 North Armour Avenue
Fort Smith, AR 72904

Harbor House, Inc.
615 North 19th Street
Fort Smith, AR 72901

Harbor View Mercy Hospital Chemical Dependency Program
10301 Mayo Drive
Fort Smith, AR 72917

Horizon
3113 South 70th Street
Fort Smith, AR 72903

Sparks Care for Alcoholic and Drug Addiction
1311 South I Street
Fort Smith, AR 72901

Western Arkansas Counseling and Guidance Center/Horizon Adolescent Program
3113 South 70th Street
Fort Smith, AR 72903

GASSVILLE

Omart Inc
116 Snowball Drive
Gassville, AR 72635

GRADY

Dept. of Correction/Cummins Unit Substance Abuse Treatment Program (SATP)
Grady, AR 71644

Dept. of Correction/Cummins Unit Substance Abuse Treatment Program (SATP)
Grady, AR 71644

Dept. of Correction/Varner Unit Substance Abuse Treatment Program (SATP)
Grady, AR 71644

HARRISON

Omart
218 East Ridge Avenue
Harrison, AR 72601-4307

HOPE

Southwest Arkansas Counseling and Mental Health Center
201 North 20th Street
Hope, AR 71801

HOT SPRINGS

Quapaw House, Inc.
812 Mount Pine Road
Hot Springs, AR 71913

HOT SPRINGS NATIONAL PARK

Barbs Place
276 Linden Avenue
Hot Springs National Park, AR 71901-3308

JONESBORO

Crowleys Ridge Development Council Northeast Arkansas Womens Recovery
417 West Jefferson Street
Jonesboro, AR 72401

Mid/South Health Systems
2920 McClellan Drive
Jonesboro, AR 72401

Saint Bernards Behavioral Health
Substance Abuse Treatment Unit
2712 East Johnson Avenue
Jonesboro, AR 72401

LITTLE ROCK

Addiction Treatment Centers
2021 Main Street
Little Rock, AR 72206

BHC Pinnacle Pointe Hospital Substance Abuse Services
11501 Financial Center Parkway
Little Rock, AR 72211

Baptist Medicla Center
9601 Interstate 630 Exit 7
Little Rock, AR 72205

Catar Clinic
1401 South University Avenue
Little Rock, AR 72204

Gyst House
8101 Frenchman Lane
Little Rock, AR 72219

Living Hope Institute
600 South McKinley Street
Suite 400
Little Rock, AR 72205-5583

Serenity Park, Inc.
2801 Roosevelt Road
Little Rock, AR 72204

Mid Arkansas Substance Abuse Services
4601 West 7th Street
Little Rock, AR 72205

Recovery 2005, Inc.
1920 South Broadway
Little Rock, AR 72204

Serenity Park, Inc.
2801 Roosevelt Road
Little Rock, AR 72204

Supervised Treatment and Education Program
715 West 2nd Street
Little Rock, AR 72201

UAMS/Substance Abuse Treatment Clinic
4313 West Markham Street Unit 3
Lower North Side
Little Rock, AR 72205

Univ of Arkansas for Medical Sciences Arkansas Cares
5821 West 20th Street
Little Rock, AR 72204

Women and Childrens Recovery Center Arkansas CARES
2002 South Fillmore Street
Cottage 6
Little Rock, AR 72204-4909

LUXURA

Department of Corrections Mississippi County Work Release
Luxura, AR 72358

MAUMELLE

Charter Behavioral Health Systems
1601 Murphy Drive
Maumelle, AR 72113

The Bridgeway
21 Bridgeway Road
Maumelle, AR 72113

MOUNTAIN HOME

Ozark Counseling Services
8 Medical Plaza
Mountain Home, AR 72653

NORTH LITTLE ROCK

Central Arkansas Veterans Healthcare Special Treatment Section
2300 Fort Roots Drive
North Little Rock Division
North Little Rock, AR 72214

Family Service Agency of Central Arkansas
628 West Broadway, Suite 300
North Little Rock, AR 72114

Riverbend Recovery Center
1201 River Road
North Little Rock, AR 72114

Central Arkansas Veterans Healthcare Special Treatment Section
2300 Fort Roots Drive
North Little Rock Division
North Little Rock, AR 72114

PARAGOULD

Crowley's Ridge Development Council Northeast Arkansas Regional Recovery Center
5882 Highway 135 South
Paragould, AR 72401

PARIS

Western Arkansas Counseling
415 South 6th Street
Paris, AR 72855-4511

PINE BLUFF

Dept. of Corrections
8001 West 7th Street
Pine Bluff, AR 71603

Human Development and Research Services, Inc.
6841 West 13th Street
Pine Bluff, AR 71602

Human Development and Research Services Pregnant Parenting Women Living Center
3100 West 34th Avenue
Pine Bluff, AR 71603-5504

Human Development and Research Services
2801 Olive Street, Suite 23
Pine Bluff, AR 71611

Southeast Arkansas Behavioral Health Care, Inc.
2500 Rike Drive
Pine Bluff, AR 71613

POCAHONTAS

Black River Area Development Corp. Substance Abuse Treatment Program
1403 Hospital Drive
Pocahontas, AR 72455

RUSSELLVILLE

Arkansas River Valley Area Council Freedom House
400 Lake Front Drive
Russellville, AR 72801

Counseling Associates/ Russellville
110 Skyline Drive
Russellville, AR 72802

SEARCY

Wilbur D. Mills Center
3204 East Moore Avenue
Searcy, AR 72143

SPRINGDALE

A Little Bit of Recovery
640 North Mill Street
Springdale, AR 72764

Decision Point, Inc.
301 Holcomb Street
Springdale, AR 72764

Ozark Guidance Center
219 South Thompson Street
Springdale, AR 72766-6430

TEXARKANA

Southwest Arkansas Counseling Mental Health Center, Inc
2904 Arkansas Boulevard
Texarkana, AR 71854

TUCKER

Arkansas Department of Corrections Tucker Maximum Security Unit
2501 State Farm Road
Tucker, AR 72168

WRIGHTSVILLE

Arkansas Department of Correction Boot Camp
Wrightsville, AR 72183

CALIFORNIA

ACTON

Department of Health Services Acton Rehabilitation Center
30500 Arrastre Canyon Road
Acton, CA 93510

ALAMEDA

Xanthos
1335 Park Avenue
Alameda, CA 94501

ALHAMBRA

San Gabriel Valley Driver Improvement
25 South Raymond Avenue
Suite 301
Alhambra, CA 91801

ALISO VIEJO

Orange County Health Care Agency Aliso Viejo Alcohol/ Drug Abuse Servicess
5 Mareblu Street Suite 100
Aliso Viejo, CA 92656

ALTURAS

Modoc County alcohol and Drug Services
128 Henderson Street
Alturas, CA 96101

ANAHEIM

California Hispanic Commission on Alcohol/Drug Abuse
Casa Elena
832 South Anaheim Boulevard
Anaheim, CA 92805

Counseling Concepts
1815 East Center Street
Anaheim, CA 92805

Hope House
707 North Anaheim Boulevard
Anaheim, CA 92805

Oasis Counseling Centers
500 Melissa Street
Barstow, CA 92311

West Coast Detox
956 South Flore Street
Anaheim, CA 92805

Western Medical Center
1025 South Anaheim Boulevard
Anaheim, CA 92805

ANGELS CAMP

Changing Echoes
7632 Pool Station Road
Angels Camp, CA 95222

ANTIOCH

Criminal Justice Service, East Location
2400 Sycamore Drive
Suite 36
Antioch, CA 94509

Reach Project
1915 D Street
Antioch, CA 94509

APPLE VALLEY

Starting Point
11726 Deep Creek Road
Apple Valley, CA 92308

APTOS

Acacia Associates
9057 Soquel Drive
Suite E
Aptos, CA 95003

ARCATA

Mad River Community Hospital/ Chemical Dependency
3800 Janes Road
Arcata, CA 95521

ARROYO GRANDE

Life Steps Pasos De Vida
1431 Pomeroy Road
Arroyo Grande, CA 93420

San Luis Obispo County Drug and Alcohol Services
1106 Grand Avenue
Arroyo Grande, CA 93420

ARVIN

Traffic and Alcohol Awareness School of Kern (TAASK)
525 Bear Mountain Boulevard
Arvin, CA 93203

ATASCADERO

Aegis Medical Systems, Inc.
6500 Morro Road
Suite D
Atascadero, CA 93422

San Luis Obispo County Drug and Alcohol Services
3556 El Camino Real
Atascadero, CA 93422

ATWATER

Community and Social Model Advocates Tranquility Village
599 Mendocino Court
Atwater, CA 95301

AUBURN

Eagle Recovery Programs
12183 Locksley Lane
Auburn, CA 95602

Pacific Educational Services
11795 Education Street, Suite 220
Auburn, CA 95603

Sierra Council on Alcohol and Drug Dependency Auburn Service Center
610 Auburn Ravine Road
Suite A
Auburn, CA 95603

South Placer Residential Treatment Program
11417 D Avenue
Auburn, CA 95603

Sierra Family Services/Auburn
991 Lincoln Way
Auburn, CA 95603

AZUSA

Social Model Recovery Systems River Community
23701 East Fork Road
Azusa, CA 91702

Stepping Stones Home Colby House II
18417 Orkney Street
Azusa, CA 91702

BAKERSFIELD

Aegis Medical Systems, Inc.
1018 21st Street
Bakersfield, CA 93301

Citizens for the Betterment of Community and Country
Capistrano Women
3316 Laverne Street
Bakersfield, CA 93309

Jasons Retreat
504 Bernard Street
Bakersfield, CA 93305

Community Service Organization (CSO) Brotherhood
715 Lake Street
Bakersfield, CA 93305

Desert Counseling
Teen Recovery Program
1617 30th Street
Bakersfield, CA 93301

Womens Services
2913 South H Street
Bakersfield, CA 93304

Ebony Counseling Center
1301 California Avenue
Bakersfield, CA 93304

Family and Substance Abuse Counseling Agency
1009 Chester Avenue
Bakersfield, CA 93301

Kern County Hispanic Commission on Alcohol/Drug Abuse
Casa Serena
2300 18th Street
Bakersfield, CA 93303

Kern County Dept. of Mental Health Services Judicial Services
1401 L Street
Bakersfield, CA 93301

Recovery Network Biofeedback Center
2100 24th Street Suite 4
Bakersfield, CA 93301

Salvation Army Adult Rehabilitation Center
200 19th Street
Bakersfield, CA 93301

Sierra Tribal Consortium
1527 19th Street Suite 418
Bakersfield, CA 93301

Substance Abuse Alternatives
1101 Union Avenue Suite 100
Bakersfield, CA 93304

Traffic and Alcohol Awareness School of Kern (TAASK)
324 Oak Street, Suite A
Bakersfield, CA 93304

Vinesman Ponderosa Christian Recovery Ranch
3231 East Panoma Lane
Bakersfield, CA 93307

BALDWIN PARK

Aegis Medical Systems, Inc.
14418 East Pacific Avenue
Baldwin Park, CA 91706

Ettie Lee Homes
4100 Baldwin Park Boulevard
Baldwin Park, CA 91706

Industry Community Interface Enterprises
13922 East Ramona Street
Suite B
Baldwin Park, CA 91706

BANNING

Soroptomist House of Hope
628 8th Street
Banning, CA 92220

Koalacare of California Ace Program
455 1st Street
Banning, CA 92220

Riverside San Bernadino County Indian Health
11555 1/2 Potrero Road
Banning, CA 92220

Riverside County Substance Abuse Program Second Chance
1626 Hargrave Street
Banning, CA 92220

BARSTOW

Civilian Employee Assistance Programs Family Services Center B-170
Marine Corps Logistics Base
Barstow, CA 92311

Jackson/Bibby Awareness Group
222 Main Street
Suite 218
Barstow, CA 92311

Oasis Counseling Centers
500 Melissa Street
Barstow, CA 92311

BELL GARDENS

Southern California Alcohol/ Drug Program Casa Libre
6635 Florence Avenue
Suites 101 and 102
Bell Gardens, CA 90201

BERKELEY

Berkeley Addiction Treatment Services
2975 Sacramento Street
Berkeley, CA 94702

Berkeley Mental Health Court Program
2640 Martin Luther King Jr Way
Berkeley, CA 94704

Bonita House Resident Treatment Facility for Dual Diagnosis
1410 Bonita Avenue
Berkeley, CA 94709

New Bridge Foundation
1820 Scenic Avenue
Berkeley, CA 94709

BEVERLY HILLS

A Los Angeles Driver Education Center of Beverly Hills
147 North San Vicinte Avenue
Beverly Hills, CA 90211

BIG BEAR LAKE

Operation Breakthrough
40880 Pedder Road
Big Bear Lake, CA 92315

BISHOP

Inyo County Substance Abuse Services
162 H Grove Street
Suite J
Bishop, CA 93514

Toiyabe Indian Health Project Family Service Department
52 Tu Su Lane
Bishop, CA 93514

BLOOMINGTON

Cedar House Rehabilitation Center
18612 Santa Ana Avenue
Bloomington, CA 92316

BLYTHE

Riverside County Substance Abuse Program
1267 West Hobson Way
Blythe, CA 92225

Veterans Alcoholic Rehabilitation Program
9826 18 Street
Blythe, CA 92225

BOULEVARD

La Posta Substance Abuse Center
8 Crestwood Place
Boulevard, CA 91905

BRISBANE

Latino Commission on Alcohol and Drug Abuse Services of San Mateo County/Casa Maria
105 McLain Avenue
Brisbane, CA 94005

BURBANK

New Way Foundation
Aware Program
207 North Victory Boulevard
Burbank, CA 91502

Padre, Inc.
2410 West Olive Avenue
Burbank, CA 91506

BURLINGAME

Insights Youth and Family Assistance
1860 El Camino Real, Suite 400
Burlingame, CA 94010

Mills Peninsula Hospital Chemical Dependency Center
1783 El Camino Real
Burlingame, CA 94010

Radiant Recovery
530 El Camino Real, Suite B
Burlingame, CA 94010

Women's Recovery Association Residential and Outpatient
1450 Chapin Street
1st Floor
Burlingame, CA 94010

BURNEY

Crossroads Clinic
20597 Commerce Way
Burney, CA 96013

Pit River Health Services Substance Abuse Services
36977 Park Avenue
Burney, CA 96013

CALEXICO

Imperial Valley Methadone Clinic
535 Cesar Chavez Boulevard
Calexico, CA 92231

CALISTOGA

Duffy's Myrtledale, Inc. Alcohol Recovery Facility
3076 Myrtledale Road
Calistoga, CA 94515

CAMARILLO

Gateway Recovery and Intervention Program
200 Horizon Circle
Camarillo, CA 93010

Palmer Drug Abuse Program of Ventura County
155 Granada Street, Suite K
Camarillo, CA 93010

CAMPBELL

Camp Recovery Centers Outpatient Services
65 West Hamilton Avenue
Campbell, CA 95008

Office of Children Adolescent and Family Services (OCAFS)
595 Millich Drive Suite 100
Campbell, CA 95008

Support Systems Homes Inc
Support Systems Homes I
2000-A White Oaks Drive
Campbell, CA 95008

Support Systems Homes II
2015 White Oaks Drive
Campbell, CA 95008

CAMPO

San Diego Freedom Ranch
1777 Buckman Springs Road
Campo, CA 91906

CAMP PENDLETON

Consolidated Substance Abuse Counseling Center
Marine Corps Base Building 16105
Camp Pendleton, CA 92055-5016

Naval Hospital Naval Addictions Rehab/Education Department
Building H-49
Camp Pendleton, CA 92055

CANOGA PARK

Cabrito Foundation
Cabrito House
7552 Remmet Avenue
Canoga Park, CA 91303

Pine Grove Hospital
7011 Shoup Avenue
Canoga Park, CA 91307

CANYON COUNTRY

I-ADARP
27225 Camp Plenty Road, Suite 4
Canyon Country, CA 91351-2654

CAPISTRANO BEACH

Community Counseling Center of San Juan Capistrano/Casa Del Cerro I
26882-26884 Avenida Las Palmas South
Capistrano Beach, CA 92624

CARMICHAEL

Associated Rehab Program for Women
8400 Fair Oaks Boulevard
Carmichael, CA 95608

Bivalley Medical Clinic Carmichael
6127 Fair Oaks Boulevard
Carmichael, CA 95608

CARPINTERIA

Salvation Army Adult Rehabilitation Center
6410 Cindy Lane
Carpinteria, CA 93013

CARSON

Fred Brown Recovery Services
Carson House
329 West 218th Street
Carson, CA 90745

Kaiser Permanente Chemical Dependency Recovery Program/Carson
23621 South Main Street
Carson, CA 90745

CASTAIC

Antelope Valley Rehabilitation Centers Warm Springs Rehabilitation Center
38200 North Lake Hughes Road
Castaic, CA 91310

CASTRO VALLEY

HAART/Castro Valley
2457 Grove Way Suite 103A
Castro Valley, CA 94546

CATHEDRAL CITY

Charter Behavioral Health System of Southern California/Palm Springs
69696 Ramon Road
Cathedral City, CA 92234

Riverside County Substance Abuse Program Cathedral Canyon Clinic
68-615 Perez Road Suite 8
Cathedral City, CA 92234

CERRITOS

Southeast California Alcoholism and Drug Programs, Inc.
13205 South Street
Cerritos, CA 90701

CHICO

Aegis Medical Systems
1166 Esplanade Street, Suite 1
Chico, CA 95926

Butte County Department of Behavioral Health
Adult Services
584 Rio Lindo Avenue
Chico, CA 95926

Youth Services
564 Rio Lindo Avenue Suite 103
Chico, CA 95926

Chico Recovery Center
2565 Zanella Way Suite E
Chico, CA 95928

EAP Addiction Recovery
1224 Mangrove Avenue Suite 7
Chico, CA 95926

Solutions
2095 Forest Avenue, Suite 2
Chico, CA 95928

Touch Stone
1390 East Lasser Avenue
Chico, CA 95926

CHINO

Jericho Outreach
Men's Home
5151 F Street
Chino, CA 91710

Women's Home
12591 Benson Avenue
Chino, CA 91710

San Bernardino County Chino Multiple Diagnosis Clinic
6180 Riverside Drive
Suite H
Chino, CA 91710

CHULA VISTA

Acupuncture Institute for Addiction Free Life
236 F Street
Chula Vista, CA 91910

MAAC Project Health Services Division
1180 3rd Avenue
Chula Vista, CA 91910

Nosotros
73 North 2nd Avenue
Building B
Chula Vista, CA 91910

McAlister Institute for Treatment and Education (MITE) Options for Recovery/ South Bay
251 Palomar Street
Suite A
Chula Vista, CA 91911

Mental Health Systems Kinesis South
835 3rd Avenue, Suite E
Chula Vista, CA 91911

San Diego Treatment Services Third Avenue Clinic
1161 3 Avenue
Chula Vista, CA 91911

Bayview Hospital Medical Health System
330 Moss Street
Chula Vista, CA 91911

CITRUS HEIGHTS

Oak House Corporation
Oak House I and II
7919 Oak Avenue
Citrus Heights, CA 95610

CLAREMONT

Crossroads
1269 North Harvard Avenue
Claremont, CA 91711

CLAYTON

Bi Bett Corporation Diablo Valley Ranch Male Recovery Community
11540 Marsh Creek Road
Clayton, CA 94517

CLEARLAKE

Alcohol and Other Drug Services Southshore
7000 B South Center Drive
Clearlake, CA 95422

Drug Abuse Alternatives Center
14709 Lakeshore Drive
Clearlake, CA 95422

CLOVIS

Central Valley Indian Health Program Substance Abuse Services
20 North Dewitt Street
Clovis, CA 93612

COLOMA

Progress House/Men's Facility
838 Beach Court Road
Coloma, CA 95613

COLTON

Western Clinical Health Services (WCHS) Inland Health Services
2275 East Cooley Drive
Colton, CA 92324-6324

COLUSA

Colusa County Behavioral Health Services
85 East Webster Street
Colusa, CA 95932

COMPTON

Compton Special Services Center
404 North Alameda Street
Compton, CA 90221

Get Off Drugs Women's Home
1416 South Tamarind Street
Compton, CA 90220

Kazi House Residential Drug Program
930 West Compton Boulevard
Compton, CA 90220

King/Drew Substance Abuse Treatment Program
3221 North Alameda Street
Building 4, Suite J
Compton, CA 90222

Mini Twelve Step House The Solution Drop-In Center
200 North Long Beach Boulevard
Compton, CA 90221

Shields for Families Exodus
1500 East Kay Street
Compton, CA 90221

CONCORD

Affordable Detox
2481 Pacheco Street
Concord, CA 94520

Bi Bett Corporation
Frederic Ozanam Center
2931 Prospect Street
Concord, CA 94518

Shennum Center
2090 Commerce Avenue
Concord, CA 94520

Mount Diablo Medical Pavilion Center for Recovery
2740 Grant Street
Concord, CA 94520

New Connections The Keller House
1760 Clayton Road
Concord, CA 94520

New Leaf Treatment Center
2151 Salvio Street, Suite T
Concord, CA 94520-2458

Recovery Management Services
Crossroads Recovery Center II
2480 Pacheco Street
Concord, CA 94520

Crossroads Recovery Center III
2118 East Street
Concord, CA 94520

Crossroads Recovery Center IV
2080 East Street
Concord, CA 94520

Crossroads Treatment Center I
2449 Pacheco Street 2nd Floor
Concord, CA 94520

Sunrise House
135 Mason Circle
Unit M
Concord, CA 94520

CORNING

Tehama Alcohol Recovery Center
Right Road
275 Solano Street
Corning, CA 96021

CORONA

Charter Behavioral Health System of Southern California/Corona
2055 Kellogg Avenue
Corona, CA 91719

Riverside County Substance Abuse Program/Corona
623 North Main Street
Suite D-11
Corona, CA 91719

CORONADO

Coronado Recovery Center
830 Orange Avenue
Coronado, CA 92118

COSTA MESA

Addiction Institute
3151 Airway Building
Suite C-1
Costa Mesa, CA 92626

Breakaway Health Corporation Breakthrough
3151 Airway Avenue, Suite D-1
Costa Mesa, CA 92626

Cope Center
440 Fair Drive
Suite K
Costa Mesa, CA 92626

First Step House of Orange County
2015 Charle Street
Costa Mesa, CA 92627

Gold Coast Counseling Center, Inc.
2950 Airway Avenue, Suite B-3
Costa Mesa, CA 92626

Hope Institute Center for Recovery and Family Education Inc
2900 Bristol Street, Suite C-206
Costa Mesa, CA 92626

Matrix Center at Costa Mesa
275 Victoria Street Suite 2-F
Costa Mesa, CA 92627

New Directions for Women
2601 Willo Lane
Costa Mesa, CA 92627

Newport Mesa Halfway House
1865 Anaheim Street
Costa Mesa, CA 92627

Orange County Health Care Agency Newport Mesa Drug Abuse Service
3115 Redhill Avenue
Costa Mesa, CA 92626

Rap Center
666 West Baker Street
Suite 421
Costa Mesa, CA 92626

South Coast Counseling Center
693 Plumer Street
Costa Mesa, CA 92627

Southern California Alcohol and Drug Programs Heritage House
2212 Placentia Avenue
Costa Mesa, CA 92627

COTATI

A Step Up
420 East Cotati Avenue
Cotati, CA 94931

COVELO

Yuki Trails Substance Abuse Program
Covelo, CA 95428

COVINA

National Council on Alcohol/Drug Dependency of East San Gabriel and Pomona Valleys
754 East Arrow Highway
Suite F
Covina, CA 91722

Santa Anita Family Services Pathways
716 North Citrus Avenue
Covina, CA 91723

Stepping Stones Home I and II
17727 East Cypress Street
Covina, CA 91722

CRESCENT CITY

Del Norte County Drug and Alcohol Services
384 Elk Valley Road
Crescent City, CA 95531

Humboldt Addictions Services Programs (HASP) Del Norte County
200 Marine Way
Crescent City, CA 95531

CULVER CITY

Driver Safety Schools AM/PM Culver City Budget School
4244 Overland Avenue
Culver City, CA 90230

DALY CITY

Asian American Recovery Services Project ODASA
244 92nd Street
Daly City, CA 94015

DANA POINT

Witts Inn
24901 Dana Point Harbor Drive
Suite 220 and 230
Dana Point, CA 92629

Christina House
33025 Christina Street
Dana Point, CA 92629

DANVILLE

San Ramon Valley Discovery Center
530 La Gonda Way
Suite A
Danville, CA 94526

DEER PARK

Crutchers Serenity House
50 Hillcrest Street
Deer Park, CA 94576

Saint Helena Hospital Alcohol and Chemical Recovery
650 Sanitarium Road
Deer Park, CA 94576

DELANO

Aegis Medical Systems, Inc.
1019 Jefferson Street
Delano, CA 93215

Traffic and Alcohol Awareness School of Kern (TAASK)
623 Main Street
Delano, CA 93215

DESCANSO

Phoenix House San Diego Residential Drug Free Program
23981 Sherilton Valley Road
Descanso, CA 91916

DESERT HOT SPRINGS

Desert Rehabilitation Services
Hacienda Valdez
12890 Quinta Way
Desert Hot Springs, CA 92240

The Ranch
7885 Annandale Avenue
Desert Hot Springs, CA 92240

Soroptimist House of Hope
13525 Cielo Azul Way
Desert Hot Springs, CA 92240

DIXON

Dixon Family Services
155 North 2 Street
Dixon, CA 95620

DOWNEY

Kaiser Permanente/Bellflower Med Ctr Imperial Outpatient Clinic
9449 East Imperial Highway
Downey, CA 90242

Southern California Alcohol and Drug Programs
Awakenings Program
11500 Paramount Boulevard
Downey, CA 90241

La Casita De Las Mamas of Downey
10615 Downey Avenue
Downey, CA 90241

DUBLIN

Occupational Health Services Drinking Driver Program
6670 Amador Plaza Road
Suite 203
Dublin, CA 94568

DULZURA

Rancho L Abri
18091 Bee Canyon Road
Dulzura, CA 91917

EAST PALO ALTO

Free at Last/Intensive Outpatient Unit
1946 University Avenue
East Palo Alto, CA 94303

EL CAJON

El Cajon Drug Court
1357 Broadway, Suite 100
El Cajon, CA 92021

McAlister Institute for Treatment and Education (MITE)
East County Center
1365 North Johnson Avenue
Suite 108
El Cajon, CA 92020

Pregnant Inmates Program
1365 North Johnson Avenue
Suite 111
El Cajon, CA 92020

San Diego Health Alliance East Office
234 North Magnolia Avenue
El Cajon, CA 92020

EL CENTRO

Imperial County MH Alcohol and Drug Programs
Outpatient Clinic
1030 Broadway
Suite 104
El Centro, CA 92243

Healthy New Life/Perinatal Treatment Program
1331 Clark Road
Building 3
El Centro, CA 92243

Sober Roads
395 Broadway
Suite 111
El Centro, CA 92243

Sure Helpline Center
120 North 6th Street
El Centro, CA 92243

Volunteers of America Alcohol and Drug Program
1331-B Clark Road
El Centro, CA 92243

EL MONTE

California Hispanic Commission on Alcohol/Drug Abuse Casa Blanca Service Center
12042 Ramona Boulevard
El Monte, CA 91732

Community Health Projects Medical Group
11041 Valley Boulevard
El Monte, CA 91731

Mid Valley Alcohol Recovery Service
3430 Cogswell Road
El Monte, CA 91732

Twin Palms Recovery Center
3574 Lexington Avenue
El Monte, CA 91731

ENCINITAS

Phoenix House Impact Program
345 Saxony Road, Suite 104
Encinitas, CA 92024

Phoenix House
335 Saxony Road
Encinitas, CA 92024

San Luis Rey Hospital
335 Saxony Road
Encinitas, CA 92024

ESCONDIDO

Fellowship Center Alcohol and Other Drug Services
736 East Grand Avenue
Escondido, CA 92025

Mental Health Systems Kinesis North
474 West Vermont Avenue
Escondido, CA 92027

Mental Health Systems North Inland Regional Recovery Center
620 North Ash Street
Escondido, CA 92025

North County Serenity House
123 South Elm Street
Escondido, CA 92025

Serenity Too
117 West Elm Street
Escondido, CA 92027

Vietnam Veterans of San Diego New Resolve Program
1207 South Escondido Boulevard
Escondido, CA 92025

EUREKA

Alcohol/Drug Care Services
1335 C Street
Eureka, CA 95501

Healthy Moms Program
2944 D Street
Eureka, CA 95501

Humboldt County Alcohol and Drug Programs
2922 I Street
Eureka, CA 95501

North Coast Substance Abuse Council Crossroads
1205 Myrtle Avenue
Eureka, CA 95501

Saint Joseph Hospital Family Recovery Services
2700 Dolbeer Street
Eureka, CA 95501

United Indian Health Child and Family Services
2120 Campton Road
Eureka, CA 95501

United Indian Lodge
116 9th Street
Eureka, CA 95501

EXETER

Courage to Change
1230 North Anderson Street
Exeter, CA 93321

FAIRFIELD

Solano County Health and Social Services Freedom Outreach
1735 Enterprise Drive
Building 1, Suite 104
Fairfield, CA 94533

Youth and Family Services Womens Substance Abuse Programs
934 Missouri Street, Suite A
Fairfield, CA 94533

FAIR OAKS

Messenger Clinic
4009-A Bridge Street
Fair Oaks, CA 95628-7503

Social Health and Addiction Recov Prog Fair Oaks Recovery Center
8312 Madison Avenue
Fair Oaks, CA 95628

FONTANA

Kaiser Permanente Hospital Chemical Dependency Recovery Program/Fontana
17046 Marygold Avenue
Marygold Annex
Fontana, CA 92335

Merrill Community Services, Inc.
16846 Merrill Avenue
Suite 202
Fontana, CA 92335

San Bernardino Mental Health Fontana Perinatal Treatment
8621 Juniper Avenue, Suite 101
Fontana, CA 92335

FOREST KNOLLS

Serenity Knolls Chemical Dependency Recovery Program
145 Tamal Road
Forest Knolls, CA 94933

FORT BRAGG

Growth Advocates Healing Center Whole Person/Growth for Adv Healing
200 South Franklin Street
Fort Bragg, CA 95437

Mendocino County Alcohol and Other Drug Programs/Fort Bragg
120 West Fir Street
Fort Bragg, CA 95437

FORTUNA

Center for Individual Recovery Services
173 South Fortuna Boulevard
Fortuna, CA 95540

Fortuna Community Services Humboldt Alcohol Recovery Treatment
2331 Rohnerville Road
Fortuna, CA 95540

FOSTER CITY

Avalon Family Counseling
225 Bonita Lane
Foster City, CA 94404

FOUNTAIN VALLEY

Pathways to Discovery
18350 Mount Langley Street
Suite 205
Fountain Valley, CA 92708

FREMONT

BHC Fremont Hospital
39001 Sundale Drive
Fremont, CA 94538

Carnales Unidos Reformando Adictos (CURA) Therapeutic Community
37437 Glenmore Drive
Fremont, CA 94536

Second Chance Phoinix Women's Program
37957 Fremont Boulevard
Fremont, CA 94536

Solidarity Fellowship
34413 Blackstone Way
Fremont, CA 94555

FRENCH CAMP

San Joaquin County
Methadone Maintenance Clinic
Office of Substance Abuse Recovery House
Outpatient Methadone Detox Clinic
Residential Treatment Center
500 West Hospital Road
French Camp, CA 95231

FRESNO

Addiction Research/Treatment, CAL Detox
East Cartwright Clinic
3103 East Cartwright Street
Fresno, CA 93725

South Orange Clinic
1235 E Street
Fresno, CA 93706

Van Ness Clinic
539 North Van Ness Street
Fresno, CA 93728

Aegis Medical Systems
34 East Minarets Avenue
Fresno, CA 93650

Alcoholism and Drug Abuse Council
4411 North Cedar Avenue
Suite 108
Fresno, CA 93721

California Substance Abuse Institute
2913 Tulare Street
Fresno, CA 93721

Cedar Vista Hospital
7171 North Cedar Avenue
Fresno, CA 93720

Comprehensive Alcohol Program (CAP) Residential
2445 West Whitesbridge Road
Fresno, CA 93706

Eleventh Hour Residential and Outpatient Programs
5639 East Park Circle Drive
Fresno, CA 93727

Family Communication Center Fresno Youth Advocates
1039 U Street
Fresno, CA 93721

Focus to Life Extended Service Program
440 North Blackstone Street
Fresno, CA 93706

Fresno County Hispanic Commission on Alcohol and Drug Abuse Services
1444 Fulton Street
Fresno, CA 93721

Genesis Group Home Spirit of Woman
728 North Echo Avenue
Fresno, CA 93728

Kaiser Permanente Chemical Dependency Services
4785 North 1st Street 2nd Floor
Fresno, CA 93726

King of Kings
Men's Recovery Home
2267 South Geneva Street
Fresno, CA 93706

Pregnant Post-Partum Women's Program
1350 East Annadale Avenue
Fresno, CA 93706

Residential Pregnancy and Post-Partum Visions Program
1530 West Whitesbridge Road
Fresno, CA 93706

West Fresno Outpatient Services
2385 South Fairview Avenue
Suite 17
Fresno, CA 93706

Nuestra Casa Recovery Home
1414 West Kearny Boulevard
Fresno, CA 93706

Tower Recovery Center
1028 North Fulton Street
Suite 101
Fresno, CA 93728

Third Floor Community Involvement Center
4969 east Clinton Street
Fresno, CA 93727

West Whitesbridge Klise Center
2855 West Whitesbridge Road
Fresno, CA 93706

Turning Point
Substance Abuse Treatment Unit
2904 East Belgravia Street
Fresno, CA 93701

After Care
1638 L Street
Fresno, CA 93721

VA Central CA Health Care System Chemical Dependence Treatment Program
2615 East Clinton Avenue
Room 116-D
Fresno, CA 93703

FRONTERA

California Institution for Women
16756 Chino-Corona Road
Frontera, CA 91720

FULLERTON

Addiction Treatment Center
Commonwealth Street
Fullerton, CA 92831

KC Services, Inc.
801 South Euclid Street
Suite 201
Fullerton, CA 92832

Orange County Health Care Agency North Orange County Alcohol and Drug Abuse Services
211 West Commonwealth Avenue
Suite 204
Fullerton, CA 92832

Saint Jude Medical Center Outpatient Family Recovery Services
251 East Imperial Highway
Suite 440
Fullerton, CA 92835

Western Pacific Fullerton Program Outpatient Detox and Methadone Maintenance
218 East Commonwealth Avenue
Fullerton, CA 92832

Woodglen Recovery Junction
771 West Orangethorpe Avenue
Fullerton, CA 92832

GARBERVILLE

Singing Trees Recovery Center
2061 Highway 101 South
Garberville, CA 95442

GARDENA

Behavioral Health Services Omni
15519 Crenshaw Boulevard
Gardena, CA 90249

GARDEN GROVE

California Hispanic Commission on Alcohol and Drug Abuse Unidos Recovery Home
9842 West 13 Street, Suite B
Garden Grove, CA 92844

Roque Center Residential
9842 West 13 Street, Suite A
Garden Grove, CA 92844

GARDEN VALLEY

Progress House II Women's Facility
5607 Mount Murphy Road
Garden Valley, CA 95633

Special Services for Groups Pacific Asian Alcohol and Drug Program
14112 South Kingsley Drive
Gardena, CA 90247

GEORGETOWN

El Dorado Council on Alcoholism Lifeskills/Divide Wellness Center
6065 Highway 193
Georgetown, CA 95634

GILROY

Community Solutions
8475 Forest Street
Suite A2
Gilroy, CA 95020

GLENDALE

Glendale Memorial Hospital and Health Center Alpha Addiction Center
1330 South Glendale Avenue
Glendale, CA 91205

New Insights
431 North Brand Boulevard
Suite 304
Glendale, CA 91203

Right on Programs
522 East Broadway, Suite 101
Glendale, CA 91205

Verdugo Mental Health Center
Substance Abuse Program
1540 east Colorado
Glendale, CA 91205

Positive Directions
225-D North Maryland Avenue
Glendale, CA 91206

GLENDORA

Project Info Community Prevention and Recovery Programs
1505 South Sunflower Avenue
Glendora, CA 91740

GOLETA

Aegis Medical Systems
5710 Hollister Avenue
Goleta, CA 93117

Santa Barbara Neighborhood Clinics Isla Vista Medical Clinic
970 Embarcadero Del Mar
Goleta, CA 93117

GRAND TERRACE

Drug Alternative Program Recovery House
11810 Kingston Street
Grand Terrace, CA 92313

GRASS VALLEY

Nevada County Council on Alcoholism Substance Abuse Treatment and Recovery
440 Henderson Street, Suite C
Grass Valley, CA 95945

Team III Family Council Center
256 Buena Vista Drive, Suite 210
Grass Valley, CA 95945

GREENBRAE

Ross Hospital Chemical Dependency Services
1111 Sir Francis Drake Boulevard
Greenbrae, CA 94904

GRIDLEY

Butte County Behavioral Health Gridley Family Counseling Center
995 Spruce Street
Gridley, CA 95948

GROVER BEACH

Casa Solana
383 South 13 Street
Grover Beach, CA 93433

HANFORD

Alcohol/Drug Education and Counseling Center
289 East 8th Street
Hanford, CA 93230

Cornerstone Community Alcohol/Drug Recovery Systems
Men's Recovery
801-805 West 7 Street
Hanford, CA 93230

Women's Program
817 West 7th Street
Hanford, CA 93230

HAPPY CAMP

River of Wellness and Recovery of The Karuk Tribal Health Program
64236 2nd Avenue
Happy Camp, CA 96039

HARBOR CITY

Western Health Harbor City Clinic
1647 West Anaheim Street
Harbor City, CA 90710

HAWTHORNE

Behavioral Health Services
Pacifica House
2501 West El Segundo Boulevard
Hawthorne, CA 90250

Patterns
12917 Cerise Avenue
Hawthorne, CA 90250

HAYWARD

Horizon Services Cronin House
2595 Depot Road
Hayward, CA 94545

Second Chance/Hayward Recovery Center
22297 Mission Boulevard
Hayward, CA 94541

Successful Alternatives for Addiction and Counseling Services
409 Jackson Street, Suite 201
Hayward, CA 94544

Terra Firma Diversion/ Education Services
26785 Mission Boulevard
Hayward, CA 94544

HEMET

Double Check Retreat
47552 East Florida Avenue
Hemet, CA 92544

I Am New Life Ministries
38400 San Ignacio Road
Hemet, CA 92543

Koalacare of California, Inc. Ace Program
413 East Latham Road
Suite 108
Hemet, CA 92543

Riverside County Substance Abuse Program/Hemet
1005 North State Street
Hemet, CA 92543

Riverside Recovery Resources
First Step House
40329 Stetson Avenue
Hemet, CA 92544

Our House
41040 Acacia Avenue
Hemet, CA 92544

Sun Ray Addictions
980 North State Street, Suite 2
Hemet, CA 92543

HERALD

River City Recovery
12490 Alta Mesa Road
Herald, CA 95638

HESPERIA

San Bernardino County Perinatal Treatment Program
11951 Hesperia Road
Hesperia, CA 92345

San Bernardino Dept of Behavioral Health Victor Valley Multi-Diagnosis Clinic
11951 Hesperia Road
Hesperia, CA 92345

HOLLISTER

San Benito County Substance Abuse Program
1111 San Felipe Road
Suite 108
Hollister, CA 95023

HOOPA

Hoopa Valley Tribal Council Division of Human Services
Orchard Avenue
Hoopa, CA 95546

HUNTINGTON PARK

Diversion Safety Program, Inc.
Escuela Latina De Alcohol
6606 Pacific Boulevard
Huntington Park, CA 90255

INDIO

ABC Recovery Center, Inc.
44-374 Palm Street
Indio, CA 92201

Awareness Program Drinking Driver
45-561 Oasis Street
Indio, CA 92201

Riverside Colatino Alcohol/Drug Abuse Casa Las Palmas
83844 Hopi Avenue
Indio, CA 92201

Riverside County Substance Abuse Program
83-912 Avenue 45
Suite 9
Indio, CA 92201

INGLEWOOD

Aegis Medical Systems
614 West Manchester Boulevard
Suite 104
Inglewood, CA 90301

Behavioral Health Services Inc Inglewood Prevention and Recovery Center
279 West Beach Avenue
Inglewood, CA 90302

Community Information and Resource Center
1630 Centinela Avenue Suite 1
Inglewood, CA 90302

El Dorado Community Service Center Inglewood Medical and Mental Healtt Services
4450 West Century Boulevard
Inglewood, CA 90304

Industry Community Interface Enterprises
101 North Labrea Avenue
Suite 402
Inglewood, CA 90301

Inglewood Substance Abuse Traffic Violators Agency
400 South La Brea Avenue
Suite 202
Inglewood, CA 90301

Los Angeles Vets Welfare to Work Program
733 South Hindry Avenue
Inglewood, CA 90301

Pride Health Services
8619 Crenshaw Boulevard
Inglewood, CA 90305

Working Alternatives Century Community Correctional Center
4026 West Century Boulevard
Inglewood, CA 90304

IONE

Department of Youth Authority Manzanita Substance Abuse Programs
201 Waterman Road
Ione, CA 95640

JACKSON

Amador County Alcohol and Drug Services
1001 Broadway
Suite 106
Jackson, CA 95642

JOSHUA TREE

Morongo Basin Mental Health Panorama Ranch
65675 Sullivan Road
Joshua Tree, CA 92252

LAGUNA BEACH

Brandys Friends Family Counseling Center
362 Third Street, Suite 200
Laguna Beach, CA 92651

LAGUNA HILLS

Carequest Program
25431 Cabot Road, Suite 111
Laguna Hills, CA 92653

LAGUNA NIGUEL

Gold Coast Counseling Center
28052 Camino Capistrano
Suite 214
Laguna Niguel, CA 92677

LA JOLLA

Practical Recovery Services
8950 Villa La Jolla Drive
Suite 1130
La Jolla, CA 92037

Scripps Memorial Hospital McDonald Center
9904 Genessee Avenue
La Jolla, CA 92037

LAKE ELSINORE

Riverside Recovery Resources Community Recovery Center
323 North Main Street
Lake Elsinore, CA 92530

LAKE FOREST

Chapman Counseling Adolescent Program
23361 El Toro Road
Suite 207
Lake Forest, CA 92630

LAKEPORT

Alcohol and Other Drug Services/Northlake
858 Lakeport Boulevard
Lakeport, CA 95453

Lake County Tribal Health Consortium
925 Berins Court
Lakeport, CA 95453

LAKEWOOD

Lakewood Regional Medical Center New Beginnings
3700 East South Street
Lakewood, CA 90712

LA MESA

Charter Behavioral Health Systems Alvarado Parkway Institute
7050 Parkway Boulevard
La Mesa, CA 91942

Federal Probation Program
7808 El Cajon Boulevard
Building 1 Suite H
La Mesa, CA 91941

Mental Health Systems Pegasus East
7841 El Cajon Boulevard, Suite C
La Mesa, CA 91941

Recovery Learning Centers
4670 Nebo Drive Suite 200
La Mesa, CA 91941

Vista Hill Foundation Parent Care Family Recovery Center
5360 Jackson Drive, Suite 120
La Mesa, CA 91942

LAMONT

Community Service Organization (CSO) De Colores
8000 Segrue Street
Lamont, CA 93241

LANCASTER

Alcohol Drug Abuse Center
43423 Division Street, Suite 108
Lancaster, CA 93535

Antelope Valley Council on Alcoholism and Drug Dependency
44815 Fig Avenue
Suite 206
Lancaster, CA 93534

High Road Program
44823 Date Avenue
Lancaster, CA 93534

Miracle Star Women's Recovering Community
44664 North Cedar Avenue
Lancaster, CA 93534

Tarzana Treatment Center
44447 North 10th Street
Lancaster, CA 93534

Western Pacific Medical Corporation Antelope Valley Medical Clinic
45335 Sierra Highway
Lancaster, CA 93534

LA PUENTE

Bay Area Addiction Research/ Treatment, CAL Detox
15229 East Amar Road
La Puente, CA 91744

LARKSPUR

Bay Area Community Resources
375 Doherty Drive
Larkspur, CA 94939

Marin Services for Women Outpatient Unit
444 Magnolia Avenue, Suite 101
Larkspur, CA 94939

Residential Unit
127 King Street
Larkspur, CA 94939

LAWNDALE

Lawndale Medical and Mental Health Services
4429 West 147th Street
Lawndale, CA 90260

LEMON GROVE

McAlister Institute for Treatment and Education (MITE)
Options Recovery East
2049 Skyline Drive
Lemon Grove, CA 91945

LEMOORE

Naval Air Station/Lemoore Counseling and Assistance Center
Barracks 1
Lemoore, CA 93246

LODI

Valley Community Counseling Services
301 West Locust Street
Lodi, CA 95240

LOMA LINDA

Jerry L. Pettis Memorial VA Medical Center Alcohol and Drug Treatment Program
11201 Benton Street
Room 116A1
Loma Linda, CA 92357

LOMPOC

Aegis Medical Systems Medical Group
200 East College Street
Lompoc, CA 93436

Family Life Counseling Service Inc
410 East Ocean Avenue
Lompoc, CA 93436

Vandenberg AFB Mental Health 30th Medical Group
338 South Dakota Avenue
Building 13-850
Lompoc, CA 93437

LONG BEACH

Behavioral Health Services Redgate Memorial Hospital
1775 Chestnut Avenue
Long Beach, CA 90813

Cambodian Association of America Community Prevention and Recovery Program
2501 Atlantic Avenue
Long Beach, CA 90806

Church at Long Beach House of Levi Christian Men's Home
725 Rose Avenue
Long Beach, CA 90813

Family Services of Long Beach
1043 Pine Avenue
Long Beach, CA 90813

Flossie Lewis Alcoholism Recovery Center
Alcoholism Recovery Center
351 East 6th Street
Long Beach, CA 90802

New Life Center
615 Elm Street
Long Beach, CA 90802

Transitional Sober Living Center
351 East 6th Street
Long Beach, CA 90802

Harbor Area High Gain Program, Inc.
330 East 3rd Street
Long Beach, CA 90802

Industry Community Interface Enterprises
555 East Artesia Street
Suite B
Long Beach, CA 90806

Long Beach Alcohol and Drug Rehab Program
Central Clinic
1133 East Rhea Street
Long Beach, CA 90806

Grand Avenue Clinic
2525 Grand Avenue
Long Beach, CA 90815

North Clinic
6335 Myrtle Avenue
Long Beach, CA 90805

National Council on Alcoholism and Other Drug Dependencies/Woman to Woman
3750 Long Beach Boulevard
Long Beach, CA 90807

New Found Life
2211 and 2137 East Ocean Boulevard
Long Beach, CA 90803

Palm House Alcoholism Recovery Home
2515 East Jefferson Street
Long Beach, CA 90810

Salsido Recovery Center Freedom House
250 East Louise Street
Long Beach, CA 90805

Southern California Alcohol/ Drug Problems Baby Step Inn
1755 Freeman Avenue
Long Beach, CA 90804

Substance Abuse Foundation of Long Beach
3125 East 7th Street
Long Beach, CA 90804

Tarzana Treatment Center/Long Beach
2101 Magnolia Avenue
Long Beach, CA 90806

Veterans' Affairs Medical Center Substance Abuse Treatment Program
5901 East 7 Street
Ward 116-A
Long Beach, CA 90822

West County Medical Clinic Substance Abuse Program
100 East Market Street
Long Beach, CA 90805

Western Health Long Beach Clinic
2933 East Anaheim Street
Long Beach, CA 90804

LOS ALAMITOS

Twin Town Treatment Center
10741 Los Alamitos Boulevard
Los Alamitos, CA 90720

LOS ANGELES

Addiction Alternatives
1125 South Beverly Drive
Suite 401
Los Angeles, CA 90035

Addiction Research/Treatment, CAL Detox
Hollywood Clinic
6411 Hollywood Boulevard
2nd Floor
Los Angeles, CA 90028

West Olympic
1926 West Beverly Boulevard
Los Angeles, CA 90057

Alcoholism Center for Women, Inc.
1147 South Alvarado Street
Los Angeles, CA 90006

A Los Angeles Driver Education Center
Eagle Rock
2607 Colorado Boulevard
Suite 104
Los Angeles, CA 90041

Alta Med Health Services Buena Care
1701 Zonal Avenue
Los Angeles, CA 90033

Alternative Action Programs
2511 South Barrington Avenue
Los Angeles, CA 90064

Alternatives Unit
2530 Hyperion Avenue
Los Angeles, CA 90027

American Health Services Hollywood Medical and Mental Health Services
8348 Beverly Boulevard
Los Angeles, CA 90048

Asian American Drug Abuse Program, Inc.
Therapeutic Community Residential
5318 South Crenshaw Boulevard
Los Angeles, CA 90043

Special Deliveries/Perinatal Services
3850 Martin Luther King Boulevard
Suite 201
Los Angeles, CA 90008

Avalon/Carver Community Center Drug Abuse Program
4920 South Avalon Boulevard
Los Angeles, CA 90011

Behavioral Health Services Hollywood Family Recovery Center
6838 Sunset Boulevard
Los Angeles, CA 90028

Boyle Heights
3421 East Olympic Boulevard
Los Angeles, CA 90023

Unit 2
4099 North Mission Road
Building A
Los Angeles, CA 90032

Beverlywood Mental Health Center
8926 Sawyer Street
Los Angeles, CA 90035

California Hispanic Comm. on Alcohol and Drug Abuse
Aguila Recovery Home
6157 North Figueroa Street
Los Angeles, CA 90042

Latino Alcohol and Drug Abuse Service Center
5801 East Beverly Boulevard
Los Angeles, CA 90022

Latinas Recovery Home/Saint Louis
327 North Saint Louis Street
Los Angeles, CA 90033

Hispanic Alcohol Recovery Home
4754 East Brooklyn Avenue
Los Angeles, CA 90022

Latinos Recovery Home/Wabash
2436 Wabash Avenue
Los Angeles, CA 90033

Mujeres Recovery Home
530 North Avenue, Suite 54
Los Angeles, CA 90042

Paloma Recovery Home
328 North Avenue, Suite 59
Los Angeles, CA 90042

Vista CPRP/Sol Youth Resources Project
109 and 111 North Avenue 56
Los Angeles, CA 90042

Canon Human Services Center
9705 South Holmes Avenue
Los Angeles, CA 90002

Casa de Hermandad
West Area Opportunity Center
11821 West Pico Boulevard
Los Angeles, CA 90064

Chabad Residential Treatment Center for Men
5675 West Olympic Boulevard
Los Angeles, CA 90029

Children's Hospital of Los Angeles Division of Adolescent Medicine/ Substance Abuse Services
5000 Sunset Boulevard, 4th floor
Los Angeles, CA 90027

Childrens Institute International
711 South New Hampshire Avenue
Los Angeles, CA 90005

Community Health Foundation Perinatal CPRP
1904 Bailey Street
Los Angeles, CA 90033

Covenant House
1325 North Western Avenue
Los Angeles, CA 90027

CRI Help Socorro RDF
5110 South Huntington Drive
Los Angeles, CA 90032

Dare U to Care Outreach Ministry
316 West 120th Street
Los Angeles, CA 90061

Didi Hirsch CMHC Dignity Center
672 South Lafayette Park Place
Suite 6
Los Angeles, CA 90057

Do It Now Foundation of Southern California, Inc.
6565 Sunset Boulevard
Suite 417
Los Angeles, CA 90028

East Los Angeles Health Task Force Comprehensive Substance Abuse Program
630 South Saint Louis Street
Los Angeles, CA 90023

East Los Angeles Womens Health Center MELA Counseling Service Center
5240 East Beverly Boulevard
2nd Floor
Los Angeles, CA 90022

El Centro De Ayuda Corporation El Centro Substance Abuse Treatment Center
1972 East Cesar Chavez Avenue
Los Angeles, CA 90033

El Centro Del Pueblo/Alvarado
2501 West 7th Street
Los Angeles, CA 90057

Felicity House
3701 Cardiff Avenue
Los Angeles, CA 90034

Higher Goals
10510 South Vermont Avenue
Los Angeles, CA 90044

His Sheltering Arms
Family Services Center
112 West 111 Street
Los Angeles, CA 90061

Recovery Home
11101 South Main Street
Los Angeles, CA 90061

Homeless Health Care Los Angeles
1010 South Flower Street
Suite 500
Los Angeles, CA 90015

Industry Community Interface Projects
2126 South La Brea Street
Suite 203
Los Angeles, CA 90016

Jeff Grand Medical Group Outpatient Methadone Maintenance and Detox
3130 South Hill Street
Los Angeles, CA 90007

Jewish Family Services of Los Angeles Alcohol and Drug Action Program
6380 Wilshire Boulevard
Los Angeles, CA 90048

Kaiser Permanente Chemical Dependency Recovery Program
Culver Marina
12001 West Washington Boulevard
Los Angeles, CA 90066

Korean Community Services
4416 West Beverly Boulevard
Los Angeles, CA 90004

Living in Recovery
951 North Mariposa Avenue
Los Angeles, CA 90029

Los Angeles Gay and Lesbian Center Mental Health Services
1625 North Schrader Boulevard
Los Angeles, CA 90028

Los Angeles Treatment Services
11427 South Avalon Boulevard
Los Angeles, CA 90061

Los Angeles Centers for Alcahol/Drug Abuse LACADA/ Homeless Outreach Project
333 South Central Avenue
Los Angeles, CA 90013

Lynwood Women and Children Center Watts Women and Children
8005 South Figueora Street
Los Angeles, CA 90003

Mary Lind Foundation
Bimini Recovery Home
155 South Bimini Place
Los Angeles, CA 90004

Rena B Recovery Home
4445 Burns Avenue
Los Angeles, CA 90029

Royal Palms Recovery Home
360 South Westlake Avenue
Los Angeles, CA 90057

Matrix Center/West Los Angeles
12304 Santa Monica Boulevard, Suite 200
Los Angeles, CA 90025

Matrix Institute
5220 West Washington Boulevard
Suite 101
Los Angeles, CA 90016

Mid Valley Recovery Services Mariposa Recovery Home
453 South Indiana Street
Los Angeles, CA 90063

Mini Twelve-Step House
303 East 52 Street
Los Angeles, CA 90011

MJB Transitional Recovery
11152 South Main Street
Los Angeles, CA 90061

Narcotic Educational Foundation of America
5055 Sunset Boulevard
Los Angeles, CA 90027

National Council on Alcoholism and Drug Dependency Main Directions
985 East 108th Street
Los Angeles, CA 90059

Natural High
3801 South Western Avenue
Los Angeles, CA 90062

Ness Counseling Center
8512 Whitworth Drive, Suite 102
Los Angeles, CA 90035

People in Progress Nonresidential Recovery Services
2500 Wilshire Boulevard
Suite 1155
2nd Floor
Los Angeles, CA 90057

Pizarro Treatment Center Outpatient Methadone Maintenance
1525 Pizarro Street
Los Angeles, CA 90026

Plaza Community Center The Esperanza Project
648 South Indiana Street
Los Angeles, CA 90023

Principles
510 New High Street
Los Angeles, CA 90012

Salvation Army
Harbor Light Center
809 East 5 Street
Los Angeles, CA 90013

Harmony Hall
3107 South Grand Avenue
Los Angeles, CA 90007

Safe Harbor
721 East 5th Street
Los Angeles, CA 90013

Shields for Families Project
Eden
1721 East 120th Street
Trailer 6
Los Angeles, CA 90059

Genesis Family Day Treatment Program
12021 South Wilmington Street, Lot C
Los Angeles, CA 90059

Soledad Enrichment Action Program
161 South Fetterly Avenue
Los Angeles, CA 90022

Special Service for Groups Pacific Asian Alcohol Program
5325 South Vermont Avenue
Los Angeles, CA 90020

Sunrise Community Counseling Center In/Outpatient
537 South Alvarado Street
Los Angeles, CA 90057

Union Rescue Christian Life Discipleship Program
226 South Main Street
Los Angeles, CA 90012

United American Indian Involvement
1125 West 6th Street, Suite 400
Los Angeles, CA 90017

United Women in Transition
5001 Budlong Avenue
Los Angeles, CA 90037

Van Ness Recovery House
1919 North Beachwood Drive
Los Angeles, CA 90068

Veterans Affairs Outpatient Clinic Drug Dependence Treatment Program
351 East Temple Street, 11C
Los Angeles, CA 90012-3328

Volunteers of America Screening and Evaluation Services
541 South Crocker Street
Los Angeles, CA 90013

Volunteers of America of Los Angeles
Alcohol Services
515 East 6th Street
Los Angeles, CA 90021

Central City Recovery Program
515 East 6th Street, 9th Floor
Los Angeles, CA 90021

Washington Medical Center and Recovery Center
12101 West Washington Boulevard
Los Angeles, CA 90066

Watts Health Foundation
House of Uhuru
8005 South Figueroa Street
Los Angeles, CA 90003

Weingart Center Association Stairs II Program
566 South San Pedro Street
Los Angeles, CA 90013

West Los Angeles Treatment Program Clinic I and Clinic II
2321 Pontius Avenue
Los Angeles, CA 90064

Wilshire Treatment Center
11704 Wilshire Boulevard
Suite D-228
Los Angeles, CA 90025

LOS GATOS

South Bay Teen Challenge
16735 Lark Avenue
Los Gatos, CA 95032

West Valley Treatment and Recovery Center
375 Knowles Drive
Los Gatos, CA 95030

LOYALTON

Sierra County Human Services Alcohol and Drug Department
704 Mill Street
Loyalton, CA 96118

LUCERNE VALLEY

True Vines Training Center for Men
10180 Banta Road
Lucerne Valley, CA 92356

LYNWOOD

Los Angeles Health Services Lynwood Clinic
11315 South Atlantic Avenue
Lynwood, CA 90262

One Nation Under God Christian Church Living High in the Lord Ministries
12526 Waldorf Drive
Lynwood, CA 90262

Principles California Regional Detention Facility
11705 Alameda Street
Lynwood, CA 90262

Shields for Families Ark Comprehensive Child Development Pg
11705 Deputy Yamamoto Place, Suite A
Lynwood, CA 90262

MADERA

Madera Counseling Center Substance Abuse Services
14277 Road 28
Madera, CA 93639

Yosemite Women's Center[
126 North B Street Street
Madera, CA 93637

MALIBU

Westside Sober Living Centers Promises Residential Treatment Center
20725 Rockcroft Drive
Malibu, CA 90265

MAMMOTH LAKES

Mono County Alcohol and Drug Program
Sierra Centre Mall
3rd Floor
Mammoth Lakes, CA 93546

MANHATTAN BEACH

San Pedro Penisula Hospital Chemical Dependency Treatment Center
1022 Sepulveda Boulevard
Manhattan Beach, CA 90266

MANTECA

San Joaquin Council for the American Indian/Three Rivers Lodge
13505 Union Road
Manteca, CA 95336

Valley Community Counseling Services
110 Sherman Avenue
Manteca, CA 95336

MARINA DEL RAY

Daniel Freeman Marina Hospital Exodus Recovery Center
4650 Lincoln Boulevard
Marina Del Ray, CA 90292

MARIPOSA

Kings View Community Services Mariposa Counseling Center
5085 Bullion Street
Mariposa, CA 95338

MARKLEEVILLE

Alpine County Mental Health Department Alcohol and Drug Program
260 Laramie Street
Markleeville, CA 96120

MARTINEZ

Born Free
111 Allen Street
Martinez, CA 94533

Discovery Program Discovery House
4639 Pacheco Boulevard
Martinez, CA 94553

Ujima Family Recovery
904 Mellus Street
Martinez, CA 94553

MARYSVILLE

Aegis Medical Systems
320 H Street, Suite 2
Marysville, CA 95901

Center for Behavioral Health of Marysville
1496 North Beale Road
Marysville, CA 95901

MCCHORD AFB

McChord Air Force Base Substance Abuse Program
62 MDOS/SGOMH 160 G Street
McChord AFB, CA 98438-1130

MCCLELLAN AFB

McClellan AFB Substance Abuse Program Social Actions Office
77 Medical Group/SGOHA 5727 Perrin Street
Suite 1 Bldg 1042
McClellan AFB, CA 95652-1231

MENLO PARK

Veterans' Affairs Health Care System Addiction Treatment Service Psychiatric Services
795 Willow Road
137 ATS
Menlo Park, CA 94025

MERCED

Central Valley Addiction Center
17 East Main Street
Merced, CA 95340-5044

Community/Social Model Advocates, Inc.
Hobie House
1301 Yosemite Parkway
Merced, CA 95340

Lifestyle Management Drinking Driver Program
1521 West Main Street
Merced, CA 95340

Merced Alcohol and Drug Services Recovery Assistance for Teens
1836 K Street
Merced, CA 95340

Merced County Alcohol and Drug Abuse Services Perinatal Center
658 West Main Street
Merced, CA 95340

MISSION VIEJO

Charter Behavioral Health System of Southern California/Mission Viejo
23228 Madero Street
Mission Viejo, CA 92691

MODESTO

Aegis Medical Systems, Inc.
801 17th Street, Suite E
Modesto, CA 95350

Family Service Agency of Stanislaus County
First-Step Program
707 14th Street
Modesto, CA 95351

New Hope Recovery House
1406 Fordham Avenue
Modesto, CA 95350

Recovery Crossroads
1024 J Street Suite 427
Modesto, CA 95354-0844

Recovery Systems Associates
330 McHenry Avenue Suite C
Modesto, CA 95354

Stanislaus Behavioral Health Center
1501 Claus Road
Modesto, CA 95355

Stanislaus County Dept. of Mental Health
Alcohol and Drug Treatment Program
800 Scenic Drive
Building D North
Modesto, CA 95350

Genesis Narcotic Replacement Therapy
800 Scenic Drive SW
Building D South
Modesto, CA 95350

Juvenile Drug Court
2215 Blue Gum Street
Main Building
Modesto, CA 95350

Men in Recovery
8224 West Grayson Road
Modesto, CA 95358

Substance Abuse Services Outpatient Drug Free
1501 F Street
Modesto, CA 95354

MONROVIA

Santa Anita Family Services Pathways
605 South Myrtle Avenue
Monrovia, CA 91016

Spencer Recovery Hospital
345 West Foothill Boulevard
Monrovia, CA 91016

MONTAGUE

Next Step Perinatal Services
211 South 13th Street
Montague, CA 96064

MONTCLAIR

Inland Health Services (IHS) Montclair
4761 Arrow Highway
Montclair, CA 91763

MONTEREY

Community Hospital Recovery Center
576 Hartnell Street
Monterey, CA 93940

MONTEREY PARK

Alhambra Safety Services
926 East Garvey Avenue
Suite A
Monterey Park, CA 91754

MONTGOMERY CREEK

Wilderness Recovery Center
19650 Cove Road
Montgomery Creek, CA 96065

MOORPARK

Turning Point Counseling
3939 Hitch Boulevard
Moorpark, CA 93021-8706

MOUNTAIN VIEW

Community Health Awareness Program
711 Church Street
Mountain View, CA 94041

El Camino Hospital Chemical Dependency Services
2500 Grant Road
ECH133
Mountain View, CA 94040

Siskiyou County Mental Health Alcohol and Drug Abuse Services
909 Ream Avenue
Mount Shasta, CA 96067

MURRIETA

Anderson and Associates Counseling Services
26811 Hobie Circle
Suite 02
Murrieta, CA 92362

Dr Raese and Associates
25095 Jefferson Avenue, Suite 202
Murrieta, CA 92562

NAPA

Alternatives For Better Living
1100 Lincoln Avenue, Suite 204
Napa, CA 94558

Napa County Drinking Driver Program
900 Coombs Street
Suite M16
Napa, CA 94559

Napa County Human Services Alcohol and Drug Program
2344 Old Sonoma Road
Napa, CA 94559

Our Family Corporation
Jefferson House
3552 Jefferson Street
Napa, CA 94558

Napa State Hospital
Evergreen Drive/D Ward
Napa, CA 94559

Redwood House
2033 Redwood Road
Napa, CA 94558

NATIONAL CITY

Healthy Beginnings/Nueva Esperanza Paradise Valley Hospital
2345 East 8th Street, Suite 110
National City, CA 91950

NEVADA CITY

Nevada County Dept. of Mental Health
Lovett Recovery Center
10075 Bost Avenue
Nevada City, CA 95959

Mental Health Services
10433 Willow Valley Road
Nevada City, CA 95959

NEWARK

Second Chance Tri-Cities Program
6330 Thornton Avenue
Newark, CA 94560

NEWBURY PARK

Ventura County DDP Conejo Valley DDP
2824 Camino Dos Rios, Suite 101
Newbury Park, CA 91320

NEWHALL

ACT Behavioral Center
24876 Apple Street, Suite C
Newhall, CA 91321

El Dorado Community Service Center Santa Clarita Medical and Mental Health Services
24625 Arch Street
Newhall, CA 91321

National Council on Alcoholism and Drug Dependence of San Fernando Valley
24779 Valley Street
Newhall, CA 91321

NEWPORT BEACH

Alternative Sentencing Relapse Prevention
Newport Boulevard, Suite 101
Newport Beach, CA 92663

Hoag Memorial Hospital Chemical Dependency Center
1 Hoag Drive
Newport Beach, CA 92658

Sober Living by the Sea
2811 Vista Way
Newport Beach, CA 92663

NORCO

Walden House Therapeutic Community at The California Rehabilitation Center
5th and Western Street
Norco, CA 91760

NORTH FORK

Sierra Tribal Consortium
57128 Road 225
North Fork, CA 93643

NORTH HIGHLANDS

Mexican American Alcoholism Program MAAP Sacramento DDP and Drug Diversion
3437 Myrtle Avenue, Suite 420
North Highlands, CA 95660

NORTH HILLS

Veterans Affairs Medical Center Chemical Dependency Treatment Program
16111 Plummer Street
Ward 41-C 116-A
North Hills, CA 91343

NORTH HOLLYWOOD

Chandler Lodge Foundation
11455 Chandler Boulevard
North Hollywood, CA 91601

Cri-Help, Inc. George T Pfleger Center
11027 Burbank Boulevard
North Hollywood, CA 91601

Western Pacific North Hollywood
11321 Camarillo Street
North Hollywood, CA 91602

NORWALK

Los Angeles Centers for Alcohol/Drug Abuse Recovery House
11400 Norwalk Boulevard
Suite 305
Norwalk, CA 90650

Southern California Alcohol/ Drug Problems Cider House
11400 Norwalk Boulevard
Building 209
Norwalk, CA 90650

Western Pacific Norwalk Medical Clinic
11902 Rosecrans Boulevard
Norwalk, CA 90650

NOVATO

Henry Ohlhoff House/North
5394 Nave Drive
Novato, CA 94949

Sunny Hill Children's Services Threshold for Change
619 Canyon Road
Novato, CA 94947

OAKDALE

Stanislaus County Dept of Mental Health East Side Counseling Center
631 West F Street
Oakdale, CA 95361

OAKHURST

Kings View Community Services Oakhurst Counseling Center
49774 Roak 426, Suite D
Oakhurst, CA 93644

OAKLAND

Thunder Road Chemical Dependency Recovery Hospital
390 40th Street
Oakland, CA 94609

Alameda County Healthy Infant Program Summit Medical Center
3012 Summer Street
Suite G-625
Oakland, CA 94609

Allen Temple Haight Ashbury Recovery Center
9925 14th Street
Oakland, CA 94621

Allied Fellowship Services
1524 29th Avenue
Oakland, CA 94606

American Indian Family Healing Center New Dawn Lodge/White Cloud Lodges
1815 39th Avenue
Suites D and C
Oakland, CA 94601

Asian Community Mental Health Services
310 8th Street
Suite 201
Oakland, CA 94607

Bi Bett Corporation
East Oakland Recovery Center
8900 International Bboulevard
Oakland, CA 94621

Orchid Women's Recovery Center
1342 East 27th Street
Oakland, CA 94606

East Bay Community Recovery Project
Project Pride
2441 San Pablo Avenue, 2nd floor
Oakland, CA 94606

First Step Alcohol and Drug Crisis Center
1531 Jefferson Street
Oakland, CA 94612

Healthy Babies Project
Harriet Tubman Recovery Center
1004 36th Street
Oakland, CA 94609

Harriet Tubman Recovery Center II
3328 Elm Street
Oakland, CA 94609

Maudell Shirek
3229 Elm Street
Oakland, CA 94609

Maudell Shirek Recovery Village
471 34th Street
Oakland, CA 94609

Highland Hospital Substance Abuse Program Healthy Start
14ll East 31st Street
Oakland, CA 94602

Kaiser Permanente Chemical Dependency Services
969 Broadway Street
Oakland, CA 94607

Mandana House Community Recovery Center
3989 Howe Street
Oakland, CA 94611

Merritt Peralta Institute Treatment Services
3012 Summitt Street
Oakland, CA 94609

Missionary Recovery Center
1739 8th Street
Oakland, CA 94602

Narcotic Education League
El Chante Alcoholism Recovery Home
425 Vernon Street
Oakland, CA 94610

Si Se Puede
3315 International Boulevard
Oakland, CA 94601

Native American Health Center
3124 East 14th Street
Oakland, CA 94601

Occupational Health Service DWI Education Program
340 Pendleton Way
Suite B 129
Oakland, CA 94621

Praise Fellowship Ministries Men's Recovery Facility
7400 MacArthur Boulevard
Oakland, CA 94605

Saint Marys Center Recovery 55
635 22nd Street
Oakland, CA 94612

Solid Foundation
Keller House
353 Athol Avenue
Oakland, CA 94606

Mandela I
6939 McArthur Boulevard
Oakland, CA 94601

Mandela II
3408 Andover Street
Oakland, CA 94610

Women's Center
4778 East 14th Street
Oakland, CA 94621

West Oakland Health Center
Cocaine Recovery Center East
9006 MacArthur Boulevard
Oakland, CA 94605

Wistar Redemption and Recovery
220 Wistar Road
Oakland, CA 94603

ZDK 14th Street Clinic and Medical Group
1124 International Boulevard
Oakland, CA 94703

OCEANSIDE

McAlister Institute for Treatment and Education (MITE)
North Coastal
514 North Hill Street
Oceanside, CA 92054

North Coastal Detox
4010 Via Serra Street
Oceanside, CA 92056

Mental Health Systems Pegasus West
560 Greenbrier Drive
Oceanside, CA 92054

Rebuild
2103 El Camino Real, Suite 203
Oceanside, CA 92008

Tri City Medical Center
4002 Vista Way
Oceanside, CA 92056

Turning Point Crisis Center
1738 South Tremont Street
Oceanside, CA 92054

ONTARIO

Bilingual Family Counseling Service Center for Recovery
317 West F Street
Ontario, CA 91762

Community Health Projects/ Ontario
324 North Laurel Avenue
Ontario, CA 91762

Inland Aids Project
1135 North Mountain Street
Ontario, CA 91762

Inland Valley Drug and Alcohol Recovery Services Caroline House
1646 East Caroline Street
Ontario, CA 91764

Marin Recovery Home
1636 North Marin Avenue
Ontario, CA 91764

Orange Recovery Center
1003 North Orange Avenue
Ontario, CA 91764

Valley Improvement Programs
210 West B Street
Ontario, CA 91762

ORANGE

Chapman House
3806 East Roberta Avenue
Orange, CA 92869

City of Orange Police Department Crisis Intervention Unit
1107 North Batavia Street
Orange, CA 92667

Mariposa Women's Center
812 Town and Country Road
Orange, CA 92668

Touchstones
525 North Parker Street
Orange, CA 92668

Turning Point Recovery Center
805 West LaVeta Street, Suite 103
Orange, CA 92868

ORANGEVALE

New Dawn
6043 Roloff Way
Orangevale, CA 95662

ORLAND

Glenn County Health Services/ Orland Substance Abuse Department
1187 East South Street
Orland, CA 95963

ORLEANS

River of Wellness and Recovery of The Karuk Tribal Health Program
Highway 96
Orleans, CA 95556

OROVILLE

Behavioral Health Services Feather River Indian Health
2167 Montgomery Street
Oroville, CA 95965

Butte County Behavioral Health Services
Adult Services/Oroville
18-C County Center Drive
Oroville, CA 95965

Oroville Community Counseling Center
2856 Olive Highway, Suite A
Oroville, CA 95965

OXNARD

Alternative Action Programs
2630 Saddle Avenue
Oxnard, CA 93033

Aegis Medical Systems
620 South D Street
Oxnard, CA 93030

2055 Saviers Road
Suite 10
Oxnard, CA 93033

Rainbow Recovery Centers I
1826 East Channel Island Boulevard
Oxnard, CA 93033

Shamrock House
1334 East Channel Islands Boulevard
Oxnard, CA 93033

Oxnard Center
2651 South C Street
Suite 1
Oxnard, CA 93033

New Start for Moms
315 North A Street
Oxnard, CA 93030

Ventura County Hispanic Commission on Alcohol and Drug Services/Casa Latina
1430 Junewood Way
Oxnard, CA 93030

Victory Outreach Oxnard
200 Pleasant Valley Road
Oxnard, CA 93030

PACIFICA

Pyramid Alternatives
480 Manor Plaza
Pacifica, CA 94044

PACIFIC GROVE

Beacon House
468 Pine Avenue
Pacific Grove, CA 93950

PACOIMA

Didi Hirsch CMHC Via Avanta Program
11643 Glenoaks Boulevard
Pacoima, CA 91331

El Proyecto Del Barrio Arleta
8902 Woodman Avenue
Pacoima, CA 91331

PALMDALE

American Health Services Palmdale Medical
2720 East Palmdale Boulevard, Suite 129
Palmdale, CA 93550

Antelope Valley Council on Alcoholism and Drug Dependency
38345 30th Street, Suite E
Palmdale, CA 93550

Midway Ranch Sober Living Center
40836 20th Street West
Palmdale, CA 93551

PALM DESERT

Crossroads Counseling
Highway 111, Suite 202
Palm Desert, CA 92260-3909

PALM SPRINGS

Lifes Journey Center
291 East Camino Monte Vista Street
Palm Springs, CA 92262

Michael's House Treatment Center for Men
430 South Cahuilla Street
Palm Springs, CA 92262

PALO ALTO

Daytop Village Adult Services
2560 Pulgas Avenue
Palo Alto, CA 94303

Free at Last
Malaika House
2043 Euclid Avenue
Palo Alto, CA 94303

Walker House
1095 Weeks Street
Palo Alto, CA 94303

North County Alcohol Services Center
231 Grant Avenue
Palo Alto, CA 94306

Stanford Alcohol and Drug Treatment Center Dept of Psychiatry/Behavioral Sciences
401 Quarry Road Room 1353
Palo Alto, CA 94305-5541

PANORAMA CITY

El Proyecto Del Barrio
9140 Van Nuys Boulevard
Panorama City, CA 91402

Western Pacific Med Corp Western Pacific Panorama Med Clinic
9462 Van Nuys Boulevard
Panorama City, CA 91402

PARADISE

Butte County Behavioral Health Paradise Community Counseling Center
5910 Clarke Road, Suite W
Paradise, CA 95969

Skyway House
6373 Oak Way
Paradise, CA 95967

PARAMOUNT

Creative Alternatives
8528 1/2 Rosecrans Avenue
Paramount, CA 90723

PASADENA

Aegis Medical Systems
1724 East Washington Boulevard
Pasadena, CA 91101

Bishop Gooden Home
191 North El Molino Avenue
Pasadena, CA 91101

California Drug Consultants
659 East Walnut Street
Pasadena, CA 91101

Casa de las Amigas
160 North El Molino Avenue
Pasadena, CA 91101

City of Pasadena/Dept of Public Health Pasadena Recovery Program
1845 North Fair Oaks Avenue
Pasadena, CA 91103

Grandview Foundation
1230 North Marengo Avenue
Pasadena, CA 91103

225 Grandview Street
Pasadena, CA 91104

Las Encinas Hospital Chemical Dependency Program
2900 East Del Mar Boulevard
Pasadena, CA 91107

Pasadena Council on Alcoholism and Drug Depedency/Referral Agency
181 North Hudson Avenue
Pasadena, CA 91101

Principles Impact Drug/Alcohol Treatment Center
2659 & 2661 Nina Street
Pasadena, CA 91103

1680 North Fair Oaks Avenue
Pasadena, CA 91103

145 North Vista Avenue
Suite 101
Pasadena, CA 91107

Saint Luke Medical Center Share Unit
2632 East Washington Boulevard
Pasadena, CA 91107-7021

Urban Revitalization Development Corporation/ Choices
1460 North Lake Avenue
Suite 107
Pasadena, CA 91104

Walter Moving Home
218 South Madison Avenue
Pasadena, CA 91101

PATTON

Patton State Hospital
3102 East Highland Avenue
Patton, CA 92369

PERRIS

Perris Valley Recovery Programs
236 East Third Street, Suite B
Perris, CA 92570

PETALUMA

Comprehensive Counseling Center
35 Maria Drive, Suite 861
Petaluma, CA 94952

Henry Ohlhoff Outpatient Programs Petaluma Office
35 Maria Drive, Suite 852
Petaluma, CA 94954

Saint Anthony Foundation Saint Anthony Farm
11207 Valley Ford Road
Petaluma, CA 94952

PHELAN

Aegis Medical Systems
Phelan Clinic
203777 Phelan Road
Phelan, CA 92371

PICO RIVERA

Cornerstone Health Services Outpatient Methadone Clinic
8207 Whittier Boulevard
Pico Rivera, CA 90660

Eastside Health Services
5200 San Gabriel Place
Suite B and C
Pico Rivera, CA 90660

PINOLE

Doctors Hospital of Pinole New Beginnings Program
2151 Appian Way
Pinole, CA 94564

Tri-Cities Discovery Center
2586 Appian Way
Pinole, CA 94564

PITTSBURG

Addiction Research/Treatment, CAL Detox
45 Civic Avenue
Room 128
Pittsburg, CA 94565

Bi Bett Corporation
East County Detox Center/DUI
500 School Street
Pittsburg, CA 94565

East County Wollam House/ Perinatal
510 Wollam Avenue
Pittsburg, CA 94565

Born Free East
550 School Street
Pittsburg, CA 94565

East County Wollam House for Women
498 Wollam Avenue
Pittsburg, CA 94565

New Connections/Pittsburg Bay Point
440 Railroad Avenue
Pittsburg, CA 94565

UJIMA Family Recovery Services
East Intensive Day Treatment
369 East Leland Road
Pittsburg, CA 94565

PLACERVILLE

El Dorado Council on Alcoholism (EDCA) Lifeskills
2810 Coloma Road
Placerville, CA 95667

New Morning Youth and Family Services
6765 Green Valley Road
Placerville, CA 95667

Progress House Outpatient Program
2914 A Cold Springs Road
Placerville, CA 95667

PLEASANT HILL

Bi Bett Corporation Gregory Recovery Center
270 Campbell Lane
Pleasant Hill, CA 94523

Drake House
808 Grayson Road
Pleasant Hill, CA 94523

PLEASANTON

Valley Community Health Center, Inc.
3922 Valley Avenue, Suite A
Pleasanton, CA 94566

POINT REYES STATION

West Marin County Community Outreach Project
3922 Valley Aavenue, Suite A
Point Reyes Station, CA 94956

POMONA

Aegis Medical Systems
Garey Clinic
1050 North Garey Avenue
Pomona, CA 91767

Pomona Unit
152 West Artesia Street
Pomona, CA 91768

Behavioral Health Services American Recovery Center
2180 West Valley Boulevard
Pomona, CA 91768

Inland Valley Drug and Alcohol Recovery Services
375 South Main Street, Suite 111
Pomona, CA 91766

National Council on Alcoholism/Drug Dependence of East San Gabriel
375 South Main Street, Suite 102
Pomona, CA 91766

Pomona Community Crisis Center
221 North Palomares Street
Pomona, CA 91766

Prototypes Women's Center
845 East Arrow Highway
Pomona, CA 91767

PORT HUENEME

Anacapa Hospital Substance Abuse Services
307 East Clara Street
Port Hueneme, CA 93041

PORTERVILLE

Alcohol and Drug Services of Tulare Alternative Services
215 North D Street
Porterville, CA 93257

Indian Health Services Tule River Alcoholism Program
Route 7
Porterville, CA 93257

Paar Center Porterville Halfway House
218-232 West Belleview Avenue
Porterville, CA 93257

237 West Belleview Avenue
Porterville, CA 93257

SRS
38 West Morton Avenue
Porterville, CA 93257

Turning Point Youth Services
288 North 2nd Street
Porterville, CA 93257

QUINCY

Plumas County Alcohol and Drug Dept.
Courthouse Annex and Highway 70
Quincy, CA 95971

RAMONA

Broad Horizons
1236 H Street
Ramona, CA 92065

Group Conscience/Pemarro
1482 Kings Villa Road
Ramona, CA 92065

Mental Health Systems North Rural Recovery Center
323 Hunter Street
Ramona, CA 92065

RANCHO CORDOVA

D and A Detox Center
2721 Barbera Way
Rancho Cordova, CA 95670

RANCHO CUCAMONGA

Matrix Institute on Addictions
9375 Archibald Avenue, Suite 204
Rancho Cucamonga, CA 91730

RANCHO MIRAGE

Betty Ford Center at Eisenhower
39000 Bob Hope Drive
Rancho Mirage, CA 92270

Pine Ridge Treatment Center
71650 Sahara Road Sahara Plaza
Suite 1
Rancho Mirage, CA 92270

RED BLUFF

Tehama County Health Agency Alcohol and Drug Division
447 Walnut Street
Red Bluff, CA 96080

REDDING

Cornerstone Recovery Systems
13144 Bear Mountain Road
Redding, CA 96003

Empire Recovery Center
1237 California Street
Redding, CA 96001

Guardian Rehabilitation Northstate Recovery System
2801 Eureka Way
Redding, CA 96001

Remi Vista Inc
3191 Churn Creek Road
Redding, CA 96002

Shasta County Alcohol and Drug Program
2770 Pioneer Drive, Suite 200
Redding, CA 96001

Perinatal Program
2770 Pioneer Drive, Suite 240
Redding, CA 96001

Shasta Options
2530 Larkspur Lane
Redding, CA 96002

Shasta Sierra Work Furlough Program
1727 South Street
Redding, CA 96001

Noble House Substance Abuse and Growth Recovery
15799 Nauvoo Trail
Redding, CA 96001

REDLANDS

Jackson/Bibby Awareness Group, Inc.
1200 Arizona Street
Suite B-10
Redlands, CA 92374

Loma Linda University Behavioral Medicine Center
1710 Barton Road
Redlands, CA 92373

Redlands Drug Court
802 West Colton Avenue, Suite C
Redlands, CA 92374

Redlands/Yucaipa Guidance Clinic Association Inc
1323 West Colton Avenue
Suite 200
Redlands, CA 92374

REDWOOD CITY

Avalon Family Counseling Services
915 Middle Field Road Suite 4
Redwood City, CA 94063

Daytop Village Adolescent
631 Woodside Road
Redwood City, CA 94061

El Centro de Libertad/The Freedom Center
650 Main Street, Suite 600
Redwood City, CA 94063

Professional Treatment Redwood City Treatment Clinic
500 Arguello Street
Redwood City, CA 94063

Service League of San Mateo County Hope House
3789 Hoover Street
Redwood City, CA 94063

REDWOOD VALLEY

Consolidated Tribal Health Project, Inc.
6991 North State Street
Redwood Valley, CA 95470

RESEDA

Fully Alive Center
18645 Sherman Way
Reseda, CA 91335

Kaiser Permanente Chemical Dependency Recovery Program
18040 Sherman Way
Reseda, CA 91335

Safety Education Center
18700 Sherman Way, Suite 118
Reseda, CA 91335

Western Pacific Reseda Program Outpatient Detox and Methadone Maintenance
18437 Saticoy Street
Reseda, CA 91335

RIALTO

San Bernardino County Office of Alcohol and Drug Programs and Treatment Services
850 East Foothill Boulevard
Suite E
Rialto, CA 92376

RICHMOND

Addiction Research/Treatment, CAL Detox
1313 Cutting Boulevard
Richmond, CA 94804

Born Free/Richmond
100 38 Street
Room 1608
Richmond, CA 94804

Contra Costa County Supervised Treatment and Recovery Program (STAR)
205 41st Street
Richmond, CA 94805

Criminal Justice Service West Location
205 41st Street
Richmond, CA 94805

Neighborhood House of North Richmond Hollman Detoxification and Fauerso Center
208 23rd Street
Richmond, CA 94804

Rectory Women's Recovery Center
CJ Hawkins House
1515 24th Street
Richmond, CA 94804

Sojourne Community Counseling Center
3029 Mac Donald Avenue
Richmond, CA 94804

Ujima Family Recovery Services Ujima West Intensive Day Treatment
3939 Bissell Street
Richmond, CA 94805

West GAADDS
205 41st Street
Richmond, CA 94805

RIDGECREST

College Health IPA Ridgecrest Unit
1400 North Norma Street
Suite 13
Ridgecrest, CA 93555

Traffic and Alcohol Awareness School of Kern (TAASK)
443 West Church Street
Ridgecrest, CA 93555

RIO VISTA

Rio Vista Care
125 Sacramento Street
Rio Vista, CA 94571

RIVERSIDE

Born Free
8310 Baxter Way
Riverside, CA 92504

Knollwood Psychiatric and Chemical Dependency Center
5900 Brockton Avenue
Riverside, CA 92506

Pine Ridge Treatment Center
5995 Brockton Avenue
Suite B
Riverside, CA 92506

March AFB Social Actions Office Drug and Alcohol Abuse Control Program
22 CSG/SLD
Building 466
Riverside, CA 92518

My Family Recovery Center
17270 Roosevelt Avenue
Riverside, CA 92508

A Woman's Place
4295 Brockton Avenue
Riverside, CA 92501

Recovery for Women
7211 Magnolia Avenue
Riverside, CA 92504

Riverside County Substance Abuse Program/Alcohol
1777 Atlantic Avenue
Riverside, CA 92507

Riverside Recovery Resources
3757 Elizabeth Street
Riverside, CA 92506

Sammon House
1420 Orange Street
Riverside, CA 92501

10 Acre Ranch, Inc.
6067 Beach Street
Riverside, CA 92509

Western Clinical Health Service (WCHS)
Inland Health Services
1021 West La Cadena Drive
Riverside, CA 92501

Whiteside Manor Alcoholic Recovery Home
2743 Orange Street
Riverside, CA 92501

9935 Challen Street
Riverside, CA 92501

Youth Service Center of Riverside
3847 Terracina Drive
Riverside, CA 92506

ROCKLIN

Rocklin Community Counseling Center
5175 Pacific Street, Suite D
Rocklin, CA 95677

ROSEMEAD

Family Care Center Substance Abuse Program
4022 North Rosemead Boulevard
Rosemead, CA 91770

ROSEVILLE

Adolescent Intercept Professional Recovery Services
220 Douglas Boulevard
Roseville, CA 95678

Aegis Medical Systems
360 Sunrise Boulevard
Roseville, CA 95678

Amicus Counseling Services
3017 Douglas Boulevard
Roseville, CA 95661

Charter Behavioral Health System of Northern California
101 Cirby Hills Drive
Roseville, CA 95678

Sierra Council on Alcohol and Drug Dependency Roseville Service Center
1A Sierragate Plaza
Suite 110
Roseville, CA 95678

Sierra Family Services/Roseville
424 Vernon Street
Roseville, CA 95678

RUNNING SPRINGS

Pine Ridge Treatment Center
2727 Highland Drive
Running Springs, CA 92382

SACRAMENTO

American Indian Substance Abuse Program, Inc. Turquoise Indian Lodge
2727 P Street
Sacramento, CA 95816

Another Choice Another Chance
5524 Assembly Court Suite 27
Sacramento, CA 95823

BHC Sierra Vista Hospital
8001 Bruceville Road
Sacramento, CA 95823

Bi-Valley Medical Clinic
2100 Capitol Avenue
Sacramento, CA 95816

Norwood
310 Harris Avenue
Suite A
Sacramento, CA 95838

California Hispanic Commission Alcohol and Drug Abuse Amigas Recovery Home
101 Southlite Circle
Sacramento, CA 95831

Center for AIDS Research Education and Services (CARES)
1500 21st Street
Sacramento, CA 95814

Center for Behavioral Health Inc
7225 East Southgate Drive
Suite D
Sacramento, CA 95823

Change
2701 Cottage Way, Suite 34
Sacramento, CA 95825

Chemical Dependency Center for Women
1507 21st Street, Suite 100
Sacramento, CA 95814

Options for Recovery/Passages
7000 Franklin Boulevard
Suite 110
Sacramento, CA 95823

Del Paso Heights Neighborhood Sacramento County Alcohol and Drug Bureau
3970 Research Drive
Sacramento, CA 95838

Facts
2726 Rio Linda Boulevard
Sacramento, CA 95815

Gateway Foundation, Inc. Gateway Recovery House
4049 Miller Way
Sacramento, CA 95817

Getting Sober Staying Sober/A Hand Up
2942 La Solidad Way
Sacramento, CA 95817

Kaiser Permanente Medical Center Chemical Dependency Program
6600 Bruceville Road
Sacramento, CA 95823

Mexican American Alcoholism Program
Mi Casa Recovery Home
2515 48 Avenue
Sacramento, CA 95822

National Council on Alcohol and Drug Dependency/ Sacramento County Affiliate
1300 Ethan Way, Suite 250
Sacramento, CA 95825

Oak Park Multi Service Center Alcohol and Drug Bureau
3415 Martin Luther King Jr. Boulevard
Sacramento, CA 95817

River City Recovery Center
E Street Unit
2218 E Street
Sacramento, CA 95816

G Street Unit
2217 G Street
Sacramento, CA 95816

Sacramento County Probation Drug Court
2140 Stockton Boulevard
Sacramento, CA 95817

Sacramento Black Alcoholism Center (SBAC)
2425 Alhambra Boulevard
Suite F
Sacramento, CA 95817

Sacramento Urban Indian Health Leo Camp Alcohol Program
801 Broadway
Sacramento, CA 95818

Salvation Army
Adult Rehabilitation Center
1615 D Street
Sacramento, CA 95814

The Effort
Alternative House
1550 Juliesse Avenue
Sacramento, CA 95815

Counseling Center
1820 J Street
Sacramento, CA 95816

Detoxification Program
7586 Stockton Boulevard
Sacramento, CA 95816

Volunteers of America
Options for Recovery
1001 Grand Avenue
Sacramento, CA 95838

Yale Mother/Infant Program
1009 Yale Street
Sacramento, CA 95818

SALINAS

Community Human Services
Methadone Clinic
1101 F North Main Street
Salinas, CA 93906

Proyecto Unidad
209 Pajaro Street Suite B
Salinas, CA 93901

Door to Hope
Women's Recovery Center
165 Clay Street
Salinas, CA 93901

Gente Del Sol Community Recovery Center
5 Williams Road
Salinas, CA 93905

Monterey County Health Department Perinatal Recovery Services
209 Pajaro Street, Suite A
Salinas, CA 93906

Sun Street Centers
Residential Recovery Program
8 Sun Street
Salinas, CA 93901

Women and Children Recovery Services
209 Pajaro Street, Suite A
Salinas, CA 93906

Valley Health Associates Medetrac
622 East Alisal Street, Suite 6
Salinas, CA 93905

SAN ANDREAS

Calaveras County Alcohol/Drug Abuse Program
891 Mountain Ranch Road
Government Center Department 64-66
San Andreas, CA 95249

SAN ANSELMO

Sunny Hills Childrens Services
300 Sunny Hills Drive Building E
San Anselmo, CA 94960

SAN BERNARDINO

Casa de Ayuda
7255 Garden Drive
San Bernardino, CA 92404

7274 Garden Drive
San Bernardino, CA 92404

Casa de San Bernardino
735 North D Street
San Bernardino, CA 92401

Center for Community Counseling and Education Agape House
1643 East Highland Avenue
Suite C
San Bernardino, CA 92404

Drug Court of San Bernardino
595 North Arrowhead Avenue
Suite A
San Bernardino, CA 92401

Hase and Associates Systems
353 West 6 Street
San Bernardino, CA 92401

Industry Community Interface Projects (ICI Projects)
265 East Mill Street, Suite 1
San Bernardino, CA 92408

Inland AIDS Project
186 East Highland Avenue
San Bernardino, CA 92405

Inland Behavioral Services
1963 North E Street
San Bernardino, CA 92405

Mental Health Systems
Pegasus DUI
2301 North Sierra Way
San Bernardino, CA 92405

Probationers Recovery Through Intervention and Drug Education (PRIDE)
595 North Arrowhead Avenue
San Bernardino, CA 92401

New House, Inc.
Men's Program
840 North Arrowhead Avenue
San Bernardino, CA 92401

Women with Children Under Five Years and Pregnant Women
856 North Arrowhead Avenue
San Bernardino, CA 92401

Pine Ridge Outpatient Center
1881 Commerce Center East, Suite 108
San Bernardino, CA 92408

San Bernardino County Dept of Public Health
799 East Rialto Avenue
San Bernardino, CA 92415

Veterans Alcoholic And Rehabilitation Program (VARP)
Gibson House for Men
1100 North D Street
San Bernardino, CA 92410

Gibson Recovery Home for Women
1135 North D Street
San Bernardino, CA 92410

Harris House
907 West Rialto Avenue
San Bernardino, CA 92410

SAN BRUNO

Casa Aztlan
3080 Longview Drive
San Bruno, CA 94066

SAN CARLOS

First Chance
335 Quarry Road
San Carlos, CA 94070

SAN CLEMENTE

Mainstream Support Group Recovery Home
607 Avenida Las Flores
San Clemente, CA 92672

Pacific Hills Treatment Center
217 and 219 Avenida Monterey
San Clemente, CA 92672

SAN DIEGO

Advanced Health Care
3703 Camino del Rio South, Suite 200
San Diego, CA 92108

Behavioral Health Group/ Frontier ADTC
10435 Chubb Lane
San Diego, CA 92101

Charter Behavioral Health System of San Diego/North LLC
11878 Avenue of Industry
San Diego, CA 92128

Cobar House
4318 Meade Avenue
San Diego, CA 92116

Community Connection Resource Center Solutions Outpatient Program
4080 Centre Street, Suite 207
San Diego, CA 92103

Comprehensive Health Center Project Hope
1760 Euclid Avenue
San Diego, CA 92105

Crash
Golden Hill House
2410 E Street
San Diego, CA 92102

Options for Recovery Central
5605 El Cajon Boulevard
San Diego, CA 92115

Short Term/RDF
4161 Marlborough Avenue
San Diego, CA 92105

Short Term II
4890 67 Street
San Diego, CA 92105

South City Regional Center
220 North Euclid Avenue
Suite 120
San Diego, CA 92114

Crossroads Foundation
3594 4 Avenue
San Diego, CA 92103

Episcopal Community Service (ECS) Mid-City Regional Recovery Center
2855 El Cajon Boulevard
San Diego, CA 92104

Ethridge Center Inc
2230 Logan Avenue
San Diego, CA 92113

Family Center for Substance
1959 Grand Avenue
San Diego, CA 92109-4511

Freedom House Community Connection Resource Center
4318 Louisiana Street
San Diego, CA 92104

Griffin and Wong Institute for Education and Training
2870 4th Avenue, Suite 100
San Diego, CA 92103

HHS South Bay Drug Court Treatment and Testing Program
1515 Palm Avenue, Suite A
San Diego, CA 92101

House of Metamorphosis
Parolee Partnership Program
412 30th Street
San Diego, CA 92102

Residential Center
2970 Market Street
San Diego, CA 92102

Isis Center
892 27th Street
San Diego, CA 92154

Kaiser Permanente Medical Group Chemical Dependency Recovery Program
3420 KenyonStreet
San Diego, CA 92103

Kearny Mesa Regional Recovery Center
7601 Convoy Court
San Diego, CA 92111

MAAC Project Recovery Home Casa de Milagros
1127 South 38th Street
San Diego, CA 92113

Mental Health Systems
Harmony Women's Recovery Center
6150 Mission Gorge Road
Suite 116
San Diego, CA 92120

Mid-Coast Counseling and Recovery Center
4926 Savannah Street, Suite 175
San Diego, CA 92110

Probationers in Recovery/Metro
6153 Fairmont Avenue, Suite 102
San Diego, CA 92120

Mesa Vista Hospital Chemical Dependency Program
7850 Vista Hill Avenue
San Diego, CA 92123

Naval Station Substance Abuse Rehab Department
3075 Corbina Alley
Building 268
San Diego, CA 92136-5127

New Hope Center
4324 34th Street
San Diego, CA 92104

Partners in Prevention Education and Recovery
3274 Rosecrans Street
San Diego, CA 92110

Pathfinders of San Diego
Recovery Home
2980 Cedar Street
San Diego, CA 92102

San Diego Center for Psychotherapy
600 B Street, Suite 1420
San Diego, CA 92101

San Diego Community Treatment Center
502 10th Avenue
San Diego, CA 92101

San Diego Health Alliance
West Office
7020 Friars Road
San Diego, CA 92108

San Diego Treatment Services Home Avenue Clinic
3940 Home Avenue
San Diego, CA 92105

San Diego Youth and Community Services
Teen Options
3660 Fairmount Avenue
San Diego, CA 92105

Scripps Clinic Chemical Dependency Treatment Program
4320 La Jolla Village Drive, Suite 140
San Diego, CA 92122

Stepping Stone, Inc.
Long Term Rehab
3767 Central Avenue
San Diego, CA 92105

Nonresidential Program
3425 5th Avenue
San Diego, CA 92103

Substance Abuse Counseling Center
Community Service MCAS Miramar
San Diego, CA 92145-2008

The Way Back
2516 A Street
San Diego, CA 92102

Turning Point Home of San Diego
1315 25th Street
San Diego, CA 92102

Twelve-Step House Heartland House
5855 Streamview Drive
San Diego, CA 92105

U.S. Marine Corps Substance Abuse Control Center
Marine Corps Recruit Depot
Building 6-E
San Diego, CA 92141

Union of Pan Asian Communities Pan Asian Alcohol/Drug Treatment Program
3288 El Cajon Boulevard, Suite 13
San Diego, CA 92104

Venture Day MHS, Inc.
6460 Boulder Lake Avenue
San Diego, CA 92119-3154

Veterans' Affairs Medical Center Alcohol and Drug Treatment Program
3350 La Jolla Village Drive
Suite 116A
San Diego, CA 02161

Vista Pacifica
7989 Linda Vista Road
San Diego, CA 92111

Volunteers of America Alcohol Services Center
741 11th Avenue
San Diego, CA 92101

1111 Island Avenue
San Diego, CA 92101

SAN FERNANDO

Northeast Valley Health Corporation community Prevention and Decovery Program
1053 North Maclay Street
San Fernando, CA 91340

SAN FRANCISCO

Addiction Research/Treatment CAL Detox Market Clinic
1111 Market Street 1st Floor
San Francisco, CA 94103

Alcoholics Rehabilitation Association First Step Home
1035 Haight Street
San Francisco, CA 94117

Asian American Recovery Services, Inc. Residential Program
2024 Hayes Street
San Francisco, CA 94117

Bakers Place
Acceptance Place
673 San Jose Avenue
San Francisco, CA 94110

Ferguson Place
1249 Scott Street
San Francisco, CA 94115

Bay Area Service Network Residential of Haight-Ashbury Clinics
111 Taylor Street, Suite 301
San Francisco, CA 94102

Bayview Hunters Point Foundation
Alice Griffith Clinic
43 Nichols Street
San Francisco, CA 94124

Substance Abuse Program
1625 Carrol Street
San Francisco, CA 94124

Youth Services
5015 3rd Street
San Francisco, CA 94124

Center on Juvenile and Criminal Justice Supportive Living Program
1671 25th Avenue
San Francisco, CA 94122

Epiphany Center Outpatient Treatment
100 Masonic Avenue
San Francisco, CA 94118

Fort Help Methadone Program
495 3 Street
San Francisco, CA 94107

Freedom from Alcohol and Drugs
1353 48th Avenue
San Francisco, CA 94122

Unit II
1362–1366 48th Avenue
San Francisco, CA 94122

Unit III
1569–1569A and 1569B 48th Avenue
San Francisco, CA 94122

Friendship House Association of American Indians
80 Julian Avenue
San Francisco, CA 94103

Golden Gate for Seniors
637 South Van Ness Avenue
San Francisco, CA 94110

Haight-Ashbury Free Clinics
Alcohol Treatment Services
425 Divisadero Street, Suite 201
San Francisco, CA 94117

Bill Pone Memorial Unit
1696 Haight Street
San Francisco, CA 94117

Black Extended Family Program
330 Ellis Street
San Francisco, CA 94102

Drug Detoxification Project
529 Clayton Street
San Francisco, CA 94117

Smith House
766 Stanyan Street
San Francisco, CA 94117

Henry Ohlhoff House
601 Steiner Street
San Francisco, CA 94117

Outpatient Programs
2423 Clement Street
San Francisco, CA 94121

2418 Clement Street
San Francisco, CA 94121

Iris Center Women's Counseling and Recovery Services
333 Valencia Street, Suite 222
San Francisco, CA 94103

Jelani House
1601 Quesada Avenue
San Francisco, CA 94124

Outpatient Services
1588 Quesada Avenue
San Francisco, CA 94124

Kaiser Permanente Hospital Chemical Dependency Recovery Program
1201 Fillmore Street
San Francisco, CA 94115

Laguna Honda Hospital Rehabilitation Center
375 Laguna Honda Boulevard
San Francisco, CA 94116-1499

Liberation House Programs
1724 Steiner Street
San Francisco, CA 94115

Milestones
291 10th Street
San Francisco, CA 94103

Mission Council on Alcohol Abuse for the Spanish Speaking
820 Valencia Street
San Francisco, CA 94110

Morrisania West
205 13th Street, Suite 3300
San Francisco, CA 94103

New Leaf Substance Abuse Services
1853 Market Street
San Francisco, CA 94103

North of Market
Senior Alcohol Program
333 Turk Street
San Francisco, CA 94102

Portero Hill Neighborhood House ZAP Project
953 Haro Street
San Francisco, CA 94107

Project Adapt
2020 Hayes Street
San Francisco, CA 94117

Saint Anthony's Foundation Covenant House
818 Steiner Street
San Francisco, CA 94117

Ozanam Reception Center
1175 Howard Street
San Francisco, CA 94103

Salvation Army Harborlight Center
Detox Primary Program
1275 Harrison Street
San Francisco, CA 94103

San Francisco General Hospital
Stimulant Treatment Outpatient Program
3180 18th Street
Suite 205
San Francisco, CA 94110

Opiate Treatment Outpatient Program/Methadone Detox
1001 Potrero Avenue
Building 90 Ward 93
San Francisco, CA 94110

Substance Abuse Services/ Methadone Maintenance
1001 Potrero Avenue
Building 90 Ward 93
San Francisco, CA 94110

Swords to Plowshares Veterans Rights Organization
1063 Market Street
San Francisco, CA 94103

Twelve-Step Programs
4049 Judah Street, Suite B
San Francisco, CA 94122

Veterans Affairs Medical Center Substance Abuse Program
4150 Clement Street, Suite 116-E
San Francisco, CA 94121

Walden House
890 Hayes Street
San Francisco, CA 94117

Adult Residential Program
815 Buena Vista West
San Francisco, CA 94117

Walden Multi-Service Center
1885 Mission Street
San Francisco, CA 94110

Adolescent Program
214 Haight Street
San Francisco, CA 94102

Western Addition Recovery House
940 Haight Street
San Francisco, CA 94117

Westside Community Mental Health Center
Inner City Program
973 Market Street
San Francisco, CA 94103

Westside Methadone Treatment Program
1153 Oak Street
San Francisco, CA 94115

Westside Youth Awareness Program
1140 Oak Street
San Francisco, CA 94117

Women's Alcoholism Center
Aviva House Recovery Home
1724 Bryant Street
San Francisco, CA 94110

Florette Pomeroy House
2263 Bryant Street
San Francisco, CA 94110

Lee Woodward Counseling Center
2201 Sutter Street
San Francisco, CA 95115

Mia House
300 Holyoke Street
San Francisco, CA 94132

SAN GABRIEL

Family Counseling Services
314 East Mission Drive
San Gabriel, CA 91776

SANGER

Fresno County Hispanic Commission on Alcohol and Drug Abuse Services
2640 Jensen Avenue
Sanger, CA 93657

SAN JACINTO

Anderson Associates Counseling Services
166 East Main Street
Suite 2
San Jacinto, CA 92586

La Vista Women's Alcoholic Recovery Center
2220 Girard Street
San Jacinto, CA 92581

SAN JOSE

Adult and Child Guidance Center Comadres Program
380 North 1st Street, Suite 200
San Jose, CA 95112

950 West Julian Street
San Jose, CA 95126

Alert Driving, Inc. (ADI) Advanced Drug Diversion Institute
3150 Almaden Expressway
Suite 145
San Jose, CA 95118

Alexian Associates Family Psychology and Counseling
3110 Provo Court, Suite A
San Jose, CA 95127-1034

ARH Recovery Homes
House on The Hill
9505 Malech Road
San Jose, CA 95151

Mariposa Lodge
9500 Malech Road
San Jose, CA 95151

Treatment Options
2345 and 2355 Mather Drive
San Jose, CA 95116

Asian Americans for Community Involvement
2400 Moorpark Avenue, Suite 300
San Jose, CA 95112

Benny McKeown Center
1281 Fleming Avenue
San Jose, CA 95127

Blossoms Perinatal Center
Gardner Family
3030 Alum Rock Avenue
San Jose, CA 95127

Central Treatment and Recovery Center
976 Lenzen Avenue, 1st Floor
San Jose, CA 95126

Central Valley Methadone Clinic
2425 Enborg Lane
San Jose, CA 95128

Charter Behavioral Health System of San Jose
455 Silicon Valley Boulevard
San Jose, CA 95138

Columbia Good Samaritan Hospital Recovery Center
2425 Samaritan Drive
San Jose, CA 95124

Combined Addicts and Professional Services (CAPS)
Outpatient Program
693 South 2nd Street
San Jose, CA 95112

Residential Unit
398 South 12th Street
San Jose, CA 95112

Drug Abuse Treatment
2220 Moorpark Avenue
San Jose, CA 95128

East Valley Treatment and Recovery
1675 Burdette Drive, Suite B
San Jose, CA 95121

Economic Social Opportunities, Inc. Rehab Health Services
1445–1447 Oakland Road
San Jose, CA 95112

Horizon Services Horizon South
650 South Bascom Avenue
San Jose, CA 95128

Indian Health Center of Santa Clara Valley Inc
1333 Meridan Avenue
San Jose, CA 95125

National Traffic Safety Institute
275 North 4th Street, 2nd Floor
San Jose, CA 95112

Office of Children Adolescent and Family Services (OCAFS)/ Foothill
230 Pala Avenue
San Jose, CA 95127

Pate House Recovery Home
35 South 12th Street
San Jose, CA 95112

Pathway House
102 South 11th Street
San Jose, CA 95112

Proyecto Primavera Garner Family Care Corp.
614 Tully Road
San Jose, CA 95111

Sullivan Recovery Home
2345 Mather Drive
San Jose, CA 95116

Support Systems Homes III
1032 Thornton Way
San Jose, CA 95128

Willow Home
808 Palm Street
San Jose, CA 95110

SAN LEANDRO

HAART
15400 Foothill Boulevard
San Leandro, CA 94578

Horizon Community Center
1403 164 Avenue
San Leandro, CA 94578

Telecare Vida Nuvea
15750 Foothill Boulevard
San Leandro, CA 94578-1012

SAN LUIS OBISPO

Cottage Care Outpatient Center of San Luis Obispo
555 Chorro Street, Suite D-2
San Luis Obispo, CA 93405

Life Steps Drug Alcohol Free Living Center
1217 Mill Street
San Luis Obispo, CA 93401

San Luis Obispo County
Drug and Alcohol Services
1102 Laurel Lane
San Luis Obispo, CA 93401

SAN MARCOS

Mental Health Systems Teen Recovery Center/North
150 Valpreda Road, Suite 104
San Marcos, CA 92069

Occupational Health Services
1637 Capalina Road
San Marcos, CA 92069

San Diego Health Alliance North Office
1560 Capalina Street
Suite A
San Marcos, CA 92069

SAN MARTIN

Santa Clara Bureau of Alcohol and Drug Programs/South County Methadone Clinic
80 West Highland Avenue
San Martin, CA 95046

SAN MATEO

Palm Avenue Detoxification
2251 Palm Avenue
San Mateo, CA 94403

Project Ninety
15 9 Avenue
San Mateo, CA 94401

Solidarity Family Center
1668 South Norfolk Avenue
San Mateo, CA 94403

Solidarity Family Center
1668 South Norfolk Street
San Mateo, CA 94403

Womens Recovery Association Hillside House
217 North Delaware Street
Suite B
San Mateo, CA 94401

SAN PABLO

Ujima Family Recovery Services
1901 Church Lane
San Pablo, CA 94806

SAN PEDRO

Beacon House Association of San Pedro
1003 South Beacon Street
San Pedro, CA 90731

Channel View House
124 West 11th Street
San Pedro, CA 90731

Lighthouse
130 West 10th Street
San Pedro, CA 90731

Palos Verdes House
1012 South Palos Verdes Street
San Pedro, CA 90731

Fred Brown's Recovery Services
13th Street House
1235 West 13th Street
San Pedro, CA 90731

Mesa House
14th Street Services
349 West 14th Street
San Pedro, CA 90731

19th Street Services
856 West 19th Street
San Pedro, CA 90731

Women's House
270 West 14th Street
San Pedro, CA 90731

House of Hope Foundation
235 West 9th Street
San Pedro, CA 90731

Joint Efforts, Inc. Outpatient Services
505 South Pacific Avenue
Suite 205
San Pedro, CA 90731

San Pedro Peninsula Hospital Chemical Dependency Treatment Center
1386 West 7th Street
San Pedro, CA 90732

SAN RAFAEL

Bay Area Institute for Family Therapy
2400 Las Gallinas Street
Suite 260
San Rafael, CA 94903

Center Point
Lifelink Perinatal Services
1477 Lincoln Avenue
San Rafael, CA 94901

Nonresidential Services
1601 2nd Street, Suite 104
San Rafael, CA 94901

The Manor
603 D Street
San Rafael, CA 94901

Henry Ohlhoff Outpatient Programs
526 3rd Street
San Rafael, CA 94901

Kaiser Permanente Medical Group Chemical Dependency Services
820 Las Gallinas Avenue
San Rafael, CA 94903

Marin Services for Men
424 Mission Avenue
San Rafael, CA 94901

Marin Treatment Center Outpatient Services
1466 Lincoln Avenue
San Rafael, CA 94901

SAN RAMON

New Bridge Foundation of San Ramon
125 Ryan Industrial Court
Suite 202
San Ramon, CA 94583

San Ramon Regional Medical Center New Beginnings
6001 Norris Canyon Road
San Ramon, CA 94583

SANTA ANA

Addiction Alternatives
1851 East 1st Street, Suite 840
Santa Ana, CA 92705

California Treatment Services Third Street Clinic
717 East 3rd Street
Santa Ana, CA 92701

Cornerstone Adult Outpatient
2130 East 4th Street, Suite 160
Santa Ana, CA 92705-3827

Orange County Health Care Agency BHC Narcotic Treatment Program
1725 West 17th Street
Room 146-B
Santa Ana, CA 92701

Orange County Health Services Agency/Orange County Drug Court
1200 North Main Street
Suite 630
Santa Ana, CA 92701

Phoenix House Adult and Adolescent Programs
1207 East Fruit Street
Santa Ana, CA 92701

Santa Ana Alcohol and Drug Abuse Services
1200 North Main Street
Suite 100-B
Santa Ana, CA 92701

Straight Talk Gerry House
1225 West 6th Street
Santa Ana, CA 92703

Gerry House West
217 North Cooper Street
Santa Ana, CA 92703

Villa Center
910 North French Street
Santa Ana, CA 92701

SANTA BARBARA

Aegis Medical Systems
217 Camino Del Remedio Street
Santa Barbara, CA 93110

American Indian Health Services
4141 State Street Suite B-6
Santa Barbara, CA 93110

Community Counseling Center
923 Olive Street, Suite 1
Santa Barbara, CA 93101

Council on Alcoholism and Drug Abuse
232 East Canon Perdido Street
Santa Barbara, CA 93102

Drug Abuse Preventive Center
24 West Arrellaga Street
Santa Barbara, CA 93101

Gay and Lesbian Resource Center Counseling and Recovery Services
126 East Haley Street
Suite A-17
Santa Barbara, CA 93101

Sansum/Santa Barbara Medical Foundation Clinic/Foundation For Recovery
215 Pesetas Lane
Santa Barbara, CA 93110

Santa Barbara Cottage Hospital
419 Pueblo Street
Santa Barbara, CA 93102

Santa Barbara Council on Alcohol and Drug Abuse/ Project Recovery
133 East Haley Street
Santa Barbara, CA 93102

Santa Barbara Rescue Mission and Bethel House
535 Eats Yanonali Street
Santa Barbara, CA 93102

Zona Seca
Alcohol/Drug Abuse Counseling Agency
26 West Figueroa Street
Santa Barbara, CA 93103

SANTA CLARA

Pathway Society
1659 Scott Boulevard, Suite 30
Santa Clara, CA 95050

SANTA CRUZ

Community Support Services
290 Pioneer Street
Santa Cruz, CA 95060

Janus of Santa Cruz
200 7 Avenue
Suite 150
Santa Cruz, CA 95062

Narconon International/ Narconon of Northern California
8699 Empire Grade Road
Santa Cruz, CA 95060

New Life Community Services Inc
707 Fair Avenue
Santa Cruz, CA 95060

Santa Cruz Community Counseling Center
Alto Counseling Center/North
271 Water Street
Santa Cruz, CA 95060

Sobriety Works
1051 41st Avenue
Santa Cruz, CA 95062-4400

Sunflower House
125 Rigg Street
Santa Cruz, CA 95060

Youth Services North County
709 Mission Street
Santa Cruz, CA 95060

Triad Santa Cruz Clinic Outpatient Methadone Maintenance
1000-A Emeline Avenue
Santa Cruz, CA 95060

Women's Crisis Support Shelter Services
1658 Soquel Drive, Suite A
Santa Cruz, CA 95060

SANTA MARIA

Aegis Medical Systems
115 East Fesler Street
Santa Maria, CA 93454

Central Coast Headway Drugs and Alcohol Awareness Program
318 West Carmen Lane
Santa Maria, CA 93454

Charles Golodner Counseling Group
301 South Miller Street, Suite 105
Santa Maria, CA 93454

Cottage Care Outpatient Center of Santa Maria
201 South Miller Street, Suite 105
Santa Maria, CA 93454

Family Life Counseling Services
301 South Miller Street, Suite 103
Santa Maria, CA 93454

Good Samaritan Shelter
Recovery Point
406 South Pine Street
Santa Maria, CA 93454

SANTA MONICA

Alcoholism Council West Area High Gain Project
1424 4 Street
Suite 205
Santa Monica, CA 90401

Clare Foundation
Adult Recovery Home
1871 9 Street
Santa Monica, CA 90405

Drug Court Program
1002 Pico Boulevard
Santa Monica, CA 90404

Santa Monica Recovery/Detox Center
907 Pico Boulevard
Santa Monica, CA 90405

Signs of Recovery Program
1023 Pico Boulevard
Santa Monica, CA 90405

Saint John's Hospital and Health Center Chemical Dependency Center
1328 22 Street
Santa Monica, CA 90404

Santa Monica Bay Area Drug Abuse Council/New Start
2714 Pico Boulevard
Suite 210
Santa Monica, CA 90401

SANTA PAULA

Community Health Projects
625 East Main Street
Santa Paula, CA 93060

Rainbow Recovery Youth Center
15005 Faulkner Road
Santa Paula, CA 93060

Santa Clara Valley Alcoholism Services United
Outpatient Program
951 East Main Street
Santa Paula, CA 93060

Recovery Home/Casa Un Paso Adelante
222 8th Street
Santa Paula, CA 93060

SANTA ROSA

California Human Development Corp Athena House
1539 Humboldt Street
Santa Rosa, CA 95404

Campobello Chemical Dependency Recovery Center
3400 Guerneville Road
Santa Rosa, CA 95401

Casa Calmecac
857 Dutton Avenue
Santa Rosa, CA 95407

Drink/Link Moderation Programs Products and Services
Santa Rosa, CA 95402

Drug Abuse Alternatives Center
Outpatient Treatment
Perinatal Day Treatment
Redwood Empire Addictions Program (REAP)
Turning Point Program
2403 Professional Drive
Santa Rosa, CA 95403

Lower Lake Transitional Living Center
2403 Professional Drive, Suite 101
Santa Rosa, CA 95403

R House
Oak Park Facility
5136 Oak Park Way
Santa Rosa, CA 95409

Santa Rosa Treatment Program
1901 Cleveland Avenue
Unit B
Santa Rosa, CA 95403

Sonoma County Alcohol, Drug, and Tobacco Services
2759 Bennett Valley Road
Santa Rosa, CA 95404

Drinking Driver Program
1300 Coddington Center
Santa Rosa, CA 95401

Ruth Place
1018 Ruth Place
Santa Rosa, CA 95401

Unity House
920 West 8th Street
Santa Rosa, CA 95401

Sonoma County Indian Health Project Behavioral Health Department
791 Lombardi Court
Suite 101
Santa Rosa, CA 95407

Villa Lodge
3640 Stony Point Road
Santa Rosa, CA 95407

Womens Recovery Services/A Unique Place
98-140 Hendley Street
Santa Rosa, CA 95401

SANTA YNEZ

Santa Ynez Tribal Health Clinic Substance Abuse Services
3410 East Highway 246
Santa Ynez, CA 93460

SAUGUS

Live Again Recovery Home
38215 North San Francisquito Canyon Road
Saugus, CA 91350

SAUSALITO

Bay Area Community Resources Marin City Project
740 Drake Avenue
Sausalito, CA 94965

SCOTTS VALLEY

Camp Recovery Center
3192 Glen Canyon Road
Scotts Valley, CA 05066

Triad Community Services Outpatient Drug and Alcohol Treatment Program
5271 Scotts Valley Drive
Suite 200
Scotts Valley, CA 95066

SEASIDE

Community Human Services
Genesis Residential Center
1152 Sonoma Avenue
Seaside, CA 93955

SHAFTER

Traffic and Alcohol Awareness School of Kern (TAASK)
511 Central Valley Highway
Shafter, CA 93263

SIERRA MADRE

Lifechanges Counseling Center
37 Auburn Street, Suite 5
Sierra Madre, CA 91024

SIMI VALLEY

Aegis Medical Systems
2943 Sycamore Drive
Suite 1
Simi Valley, CA 93065

Rainbow Recovery Centers II
3165 Tapo Canyon Road
Simi Valley, CA 93063

Ventura County Department of Public Health Simi Valley Center
4322 Eileen Street
Simi Valley, CA 93063

SKYFOREST

Rim Family Services
28545 Highway 18
Skyforest, CA 92385

SONORA

Tuolumne County Alcohol/Drug Services
12801 Cabezut Road
Sonora, CA 95370

SOUTH GATE

Southern California Alcohol and Drug Program
8627 California Avenue
South Gate, CA 90280

SOUTH LAGUNA BEACH

South Coast Medical Center Genesis Chemical Dependency Services
31872 Coast Highway
South Laguna Beach, CA 92677

SOUTH LAKE TAHOE

Sierra Recovery Center
931 Macinaw Street
South Lake Tahoe, CA 96150

2677 Reaves Street
South Lake Tahoe, CA 96150

972 Tallac Avenue, Suite B
South Lake Tahoe, CA 96150

Tahoe Turning Point Juvenile
Heavenly Treatment Center
TEHMENA
South Lake Tahoe, CA 96151

Residential Treatment Centers
562 Tehema Street
South Lake Tahoe, CA 96151

Tahoe Youth and Family Services, Inc.
1021 Fremont Avenue
South Lake Tahoe, CA 95705

SOUTH SAN FRANCISCO

First Chance Program
383 East Grand Avenue
South San Francisco, CA 94080-0007

Kaiser Permanente Medical Center Chemical Dependency Service
1200 El Camino Real
South San Francisco, CA 94080

Sitike Counseling Center
306 Spruce Avenue
South San Francisco, CA 94080

STANTON

Stanton Detox
Roque Center
10936 Dale Street
Stanton, CA 90680

Western Pacific Stanton Medical Clinic
10751 Dale Street
Stanton, CA 90680

STOCKTON

Aegis Medical Systems
8626 Lower Sacramento Road, Suite 41
Stockton, CA 95210

Focus
322 North California Street
Stockton, CA 95202

Jesus Saves Ministries
438 South Sutter Street
Stockton, CA 95203

Maynards Chemical Dependency Recovery Center
4550 North Pershing Street
Suite 3
Stockton, CA 95207

Narrow Gate Counseling Consortium
930 North Hunter Street
Stockton, CA 95269

Saint Joseph's Behavioral Health Center
2510 North California Street
Stockton, CA 95204

Salvation Army Adult Rehabilitation Center
1247 South Wilson Way
Stockton, CA 95205

San Joaquin County Chemical Dependency Counseling Center
620 North Aurora Street
Stockton, CA 95205

Starting Point
701 East Park Street
Stockton, CA 95202

Xenia Ark Residential Treatment Center
1609 North Wilson Way
Stockton, CA 95205

STUDIO CITY

Quest Counseling
3959 Laurel Canyon Boulevard, Suite C
Studio City, CA 91604

SUN VALLEY

People in Progress Sun Valley Community Rehab Center
8140 Sunland Boulevard
Sun Valley, CA 91352

SUSANVILLE

Lassen County Alcohol and Drug Program
476 Alexander Avenue
Susanville, CA 96130

Promises Perinatal Program
701-373 Johnstonville Road
Susanville, CA 96130

Lassen Indian Health Center Substance Abuse Services
795 Joaquin Street
Susanville, CA 96130

SYLMAR

Industry Community Interface (ICI) Enterprises
13741 Foothill Boulevard
Suite 110
Sylmar, CA 91342

MaClay House Inc
13370 Sayre Street
Sylmar, CA 91342

Oasis Women's Recovering Community
13832 Polk Street
Sylmar, CA 91342

Phoenix Houses of Los Angeles
11600 Eldridge Avenue
Sylmar, CA 91342

Shepherds Recovery
13466 Hubbard Street
Sylmar, CA 91342

TAFT

Memorial Center Taft Outpatient Clinic
401 Finley Drive
Taft, CA 93268

Traffic and Alcohol Awareness School of Kern (TAASK)
Taft College WESTEC 210 East Center Street
Taft, CA 93268

TAHOE CITY

Sierra Family Services/Tahoe
2690 Lake Forest Road
Suite 202
Tahoe City, CA 96145

TARZANA

Center for Counseling and Education
6025 Etiwanda Street
Tarzana, CA 91356

Looking Glass Counseling Center
19318 Ventura Boulevard
Suite 206
Tarzana, CA 91356

Ronald Nicholas, Ph.D. Marriage and Family Professional Corp.
6025 Etiwanda Avenue
Tarzana, CA 91356

Tarzana Treatment Center
18646 Oxnard Street
Tarzana, CA 91356

Valley Women's Center and Family Recovery Center
5530 Corbin Avenue
Suite 325
Tarzana, CA 91356

TEMECULA

Riverside County Substance Abuse Program
41002 Country Center Drive
Temecula, CA 92590

Hill Alcohol and Drug Treatment Center
29377 Rancho California Road
Temecula, CA 92591

THERMAL

Riverside Cnty Latino Commission on Alcohol and Drug Services/Casa Cecilia Recovery Home
83-385 Rosa Avenue
Thermal, CA 92274

THOUSAND OAKS

Ventura Recovery Cente
166 Siesta Avenue
Thousand Oaks, CA 91360

Be Free Treatment Center at Conejo Counseling Center
3625 Thousand Oaks Boulevard
Suite 6
Thousand Oaks, CA 91362

TORRANCE

Childrens Institute International South County Facility
21810 Normandie Avenue
Torrance, CA 90509

Life Change Residential Treatment Center
2815 Artesia Boulevard
Torrance, CA 90504

National Council on Alcohol and Drug Dependency
1334 Post Avenue
Torrance, CA 90501

Options for Recovery The Stork Club
1124 West Carson Street
Building N33
Torrance, CA 90502

South Bay Drug Abuse Coalition
2370 West Carson Street
Suite 136
Torrance, CA 90501

Southwest Driver Benefits Program
2370 West Carson Street
Suite 150
Torrance, CA 90501

Torrance Memorial Medical Center Outpatient Chemical Dependency Program
3330 Lomita Boulevard, 7th Floor
Torrance, CA 90505

Twin Town Treatment Center
2171 Torrance Boulevard
Torrance, CA 90501

TRACY

Valley Community Counseling Center
19 East 6th Street
Tracy, CA 95376

TRAVIS AFB

Alcohol and Drug Treatment Inpatient Program
530 Hickam Avenue
Travis AFB, CA 94535

TRUCKEE

Nevada County Substance Abuse Treatment and Recovery (NCSA)
10015 Palisades Drive, Suite 1
Truckee, CA 96162

TGIF Counseling Center Substance Abuse Services
10075 Levon Avenue, Suite 102
Truckee, CA 96161

TULARE

Alcohol Center for Teenagers (ACT)
23393 Road 68
Tulare, CA 93274

Kings View Substance Abuse Program Tulare County
559 East Bardsley Avenue
Tulare, CA 93275

TUOLUMNE

MACT Health Board Tuolumne Rural Indian Health Program
19590 Mi Wu Street
Tuolumne, CA 95379

Maynards Chemical Dependency Recovery Center
19325 Cherokee Road
Tuolumne, CA 95379

TUSTIN

Recovery Homes of America, Inc.
Cornerstone
13682 Yorba Street
Tustin, CA 92680

TWENTYNINE PALMS

Combined Drug and Alcohol Counseling Center (CDACC) Manpower Program
Marine Corps Air Ground Combat Center
Twentynine Palms, CA 92278

UKIAH

Ford Street Project
139 Ford Street
Ukiah, CA 95482

Guidville Rancheria
419h Talmage Road
Ukiah, CA 95482

Mendocino County Public Health Dept. Division of Alcohol and Other Drug Programs
302 West Henry Street
Ukiah, CA 95482

Mendocino County Youth Project
776 South State Street
Ukiah, CA 95482

UNION CITY

Kaiser Permanente Chemical Dependency Services
3552 Whipple Road
Union City, CA 94587

UPLAND

Arrow House
1439 West Arrow Highway
Upland, CA 91786

Inland Valley Drug and Alcohol Recovery Services Recovery Center
934 North Mountain Avenue
Suite A
Upland, CA 91786

San Antonio Community Hospital
999 San Bernardino Road
Upland, CA 91786

VACAVILLE

Latino Substance Abuse Program
190 South Orchard Street
Suite B-101
Vacaville, CA 95688

VALLEJO

Bi Bett Corporation
Recovery Connection
604 Broadway
Vallejo, CA 94590

Shamia Recovery Center
126 Ohio Street
Vallejo, CA 94590

Southern Solano Alcohol Council
419 Pennsylvania Street
Vallejo, CA 94590

Genesis House
1149 Warren Avenue
Vallejo, CA 94591

House of Acts Substance Abuse Program
627 Grant Street
Vallejo, CA 94590

Kaiser Permanente Chemical Dependency Services
800 Sereno Drive
Vallejo, CA 94589

Youth and Family Services Adolescent Substance Abuse Programs
408 Tennessee Street
Vallejo, CA 94590

VAN NUYS

American Health Services/Van Nuys
6265 Sepulveda Boulevard, Suite 9
Van Nuys, CA 91411

High Road Program
14430 Sherman Way
Van Nuys, CA 91405-2340

I/ADARP Van Nuys Clinic
7400 Van Nuys Boulevard
Suite 207
Van Nuys, CA 91411

National Council on Alcoholism and Drug Dependency of San Fernando Valley
14557 Friar Street
Van Nuys, CA 91411

New Directions for Youth
7400 Van Nuys Boulevard
Suite 203
Van Nuys, CA 91405

Northeast Valley Health Corporation Mi Descanso
6819 Sepulveda Boulevard
Suite 102
Van Nuys, CA 91405

Western Pacific Medical Corporation/Van Nuys
14332 Victory Boulevard
Van Nuys, CA 91401

VENICE

Didi Hirsch CMHC Outpatient Drug Abuse Services
1600 Main Street, Suite B
Venice, CA 90291

Inglewood Medical and Mental Health Services/Venice
2014 Lincoln Boulevard
Venice, CA 90291

Phoenix Houses
503 Ocean Front Walk
Venice, CA 90291

Promises Residential Treatment
3743 South Barrington Avenue
Venice, CA 90291

VENTURA

Center for Creative Change
8 North Fir Street
Ventura, CA 93001

Khepra House
105 West Harrison Avenue
Ventura, CA 93001

Medical Support Services to Substance Abusers
3291 Loma Vista Road
Ventura, CA 93003

Miracle House
92 & 94 South Anacapa Street
Ventura, CA 93001

Paul Booth Addictions Education
290 Maple Court, Suite 254
Ventura, CA 93003

Prototypes Women's Center
152 North Dos Caminos Avenue
Ventura, CA 93003

4973 Terry Drive
Ventura, CA 93003

Ventura County Drinking Driver Program
702 County Square Drive
Ventura, CA 93003

Vista Del Mar Hospital Addiction Medicine Services
801 Seneca Street
Ventura, CA 93001

VICTORVILLE

High Desert Child/Adolescent and Family Service Center
16248 Victor Street
Victorville, CA 92392

Jackson/Bibby Awareness Group DUI Services
14420 Civic Drive, Suite 3
Victorville, CA 92392

Pine Ridge Treatment Center
15367 Bonanza RoadSuite A
Victorville, CA 92392

Saint John of God Health Care Services
Alpha House
Casa San Raphael
How House Men's Program
Alpha Tot House
13333 Palmdale Road
Victorville, CA 92392

VISALIA

Alcohol and Drug Services of Tulare County Alternative Services
2223 North Shirk Road
Visalia, CA 93291

Kings View Substance Abuse Program Tulare County/New Generation
1011 West Center Street
Visalia, CA 93277

Tulare County Alcoholism Council
Mothering Heights
504 South Locust Street
Visalia, CA 93277

New Visions for Women
1425 East Walnut Street
Visalia, CA 93292

Pine Recovery Center
120 West School Street
Visalia, CA 93291

Tulare County Hispanic Commission El Primer Paso
1350 South Crowe Street
Visalia, CA 93277

Turning Point of Central California Turning Point Youth Services
119 South Locust Street
Visalia, CA 93291

VISTA

Amity at Vista
2260 Watson Way
Vista, CA 92083

Choices in Recovery
733 South Santa Fe Avenue
Vista, CA 92083

Mental Health Systems
North County Drug Court
1855 East Vista Way Suite 9
Vista, CA 92084

Options for Recovery
1381 East Vista Way
Vista, CA 92083

Probationers in Recovery
1855 East Vista Way
Vista, CA 92083

WALNUT CREEK

Criminal Justice Treatment Program Post Conviction DDP
2020 North Broadway, Suite 101
Walnut Creek, CA 94596

Kaiser Permanente Chemical Dependency Program
1425 South Main Street
Walnut Creek, CA 94596

Walnut Creek Hospital Dual Diagnosis and Addiction Services
175 La Casa Via
Walnut Creek, CA 94598

WATSONVILLE

Fenix Services
Family Outpatient Services
406 Main Street
Suite 403
Watsonville, CA 95076

Hermanas Recovery Home
640 Rodriguez Street
Watsonville, CA 95076

Pajaro Valley Prevention and Student Assistance Program
335 East Lake Avenue
Watsonville, CA 95076

Paloma House
321 East Beach Street
Watsonville, CA 95076

Santa Cruz Community Counseling Center
Alto Counseling Center/South
11-D Alexander Street
Watsonville, CA 95076

Si Se Puede
161 Miles Lane
Watsonville, CA 95076

Youth Services/South
241 East Lake Street
Watsonville, CA 95076

Watsonville Community Hospital Alcohol and Drug Treatment Center
75 Nielson Street
Watsonville, CA 95076

WEAVERVILLE

Trinity County Counseling Center Alcohol and Drug Program
801 Main Street, Suite A
Weaverville, CA 96093

Rainbow to Recovery
801 Main Street, Suite P
Weaverville, CA 96093

WEST COVINA

Community Health Projects/Los Angeles
West Covina Unit
1825 East Thelborn Street
West Covina, CA 91790

Rickman Recovery Center/West Covina
1107 South Glendora Avenue
West Covina, CA 91790

Safety Education Center
1400 West Covina Parkway
3rd Floor
West Covina, CA 91790

San Gabriel Valley Driver Improvement Program
1502 West Covina Parkway
Suite 207
West Covina, CA 91790

WEST HILLS

Pine Grove Hospital Matrix Center
7011 Shoup Avenue
West Hills, CA 91307-2337

WEST HOLLYWOOD

A Los Angeles Driver Education Center
8350 Santa Monica Boulevard
Suite 107
West Hollywood, CA 90069

WESTMINSTER

Orange County Health Care Agency Alcohol and Drug Abuse Services
14180 Beach Boulevard Suite 203
Westminster, CA 92683

WEST SACRAMENTO

John H Jones Community Clinic Drug Treatment Program
950 Sacramento Avenue
West Sacramento, CA 95691

Sacramento Recovery House, Inc.
1520 Madrone Avenue
Apartment 2
West Sacramento, CA 95691-2632

WHITTIER

Aegis Medical Systems/Whittier Methadone Treatment Program
11738 Valley View Avenue
Suite B
Whittier, CA 90604

Awakenings
12322 Clearglen Avenue
Whittier, CA 90604

Center for Recovery from Compulsivities HOW House
7237 Milton Avenue
Whittier, CA 90602

Fred C. Nelles Youth Correction Facility
11850 East Whittier Boulevard
Whittier, CA 90601

Presbyterian Intercommunity Hospital
12401 East Washington Boulevard
Whittier, CA 90602

Southern California Alcohol and Drug Programs Foley House
10501–10519 Mills Avenue
Whittier, CA 90606

WILLITS

Lucky Deuce DUI/DDP
145 South Main Street
Willits, CA 95490

Mendocino County Alcohol and Other Drug Programs
72 West Commercial Street
Willits, CA 95490

WILMINGTON

Aegis Medical Systems
936 North Wilmington Boulevard
Wilmington, CA 90744

Behavioral Health Services Wilmington Comm. Prevention/Recovery Center
1318 Avalon Boulevard
Wilmington, CA 90744

La Clinica Del Pueblo
1547 North Avalon Boulevard
Wilmington, CA 90744

Transcultural Health Development
117 East Harry Bridges Boulevard
Wilmington, CA 90744

WINNETKA

Women's Odyssey Organization, Inc.
20830 Parthenia Street
Winnetka, CA 91306

WINTERHAVEN

Fort Yuma Alcohol and Drug Abuse Prevention Program
1888 San Pasqual Road
Winterhaven, CA 92283

WOODLAND

Beamer Street Detoxification and Residential Treatment Center
178 West Beamer Street
Woodland, CA 95695

Yolo Alcoholic Recovery Center Cache Creek Lodge
435 Aspen Street
Woodland, CA 95695

YOUNTVILLE

Veterans' Home of California Alcohol and Drug Treatment Program
200 California Avenue
Yountville, CA 94599

YREKA

Siskiyou Alcohol and Drug Abuse Services
804 South Main Street
Yreka, CA 96097

The Kuruk Tribal Health Program of River of Health and Wellness
1519 South Oregon Street
Yreka, CA 96097

YUBA CITY

First Steps
539 Garden Highway, Suite C
Yuba City, CA 95991

Philbricks Place
2250 Sanborn Road, Building 3
Yuba City, CA 95993

YUCCA VALLEY

Morongo Basin Mental Health Choices DDP
55475 Santa Fe Trail
Yucca Valley, CA 92284-0000

COLORADO

AKRON

Centennial Mental Health Center Substance Abuse Services
871 East First Street
Akron, CO 80720

ALAMOSA

Crossroads Managed Care Systems Inc
2265 Lava Lane
Alamosa, CO 81101

San Luis Valley Mental Health Addiction Services
522 Alamosa Avenue
Alamosa, CO 81101

ARVADA

Alcohol Behavior Information, Inc.
7550 Grant Place
Arvada, CO 80002

Arvada Counseling Center
7850 Vance Drive, Suite 280
Arvada, CO 80003-2128

Empowerment Counseling Services Northwest
5460 Ward Road, Suite 215
Arvada, CO 80002

ASPEN

Colorado West Aspen Counseling Center
405 Castle Creek Road, Suite 9
Aspen, CO 81611

Springs Counseling Center Youth Facility
455 Rio Grande Place
Aspen, CO 81611

AURORA

Abusive Behavior Center
509-A Sable Boulevard
Aurora, CO 80011

Anchor Counseling Inc
15290 East 6th Avenue, Suite 200
Aurora, CO 80011-8833

Aurora Center for Treatment, Ltd.
1591 Chambers Road, Suite E
Aurora, CO 80011

Countermeasures
1450 South Havana Street, Suite 712
Aurora, CO 80012

Dynamic Directions Counseling Services
2323 South Troy Street, Suite 1-226
Aurora, CO 80014-1980

Insights Counseling Service, Inc.
15200 East Girard Avenue
Aurora, CO 80014-5039

Rangeview Counseling Center
1591 Fulton Street, Suite 101
Aurora, CO 80012

AVON

Spring Counseling Center PC
0150 East Beaver Creek Boulevard Suite 207
Avon, CO 81620

BOULDER

Boulder Alcohol Education Center
1525 Spruce Street
Suite 100
Boulder, CO 80302

Boulder Clinic Inc
1317 Spruce Street
Boulder, CO 80302

Boulder Community Hospital
311 Mapleton Avenue
Boulder, CO 80302

Boulder County Health Department
Recovery Program
3450 Broadway
Boulder, CO 80304

Substance Abuse Program
1333 Iris Avenue
Boulder, CO 80302

Personal Growth Services
2305 Canyon Boulevard, Room 205
Boulder, CO 80302

Rangeview Counseling Center
1800 30th Street Suite 220
Boulder, CO 80301

Serenity Center for Personal Growth, Inc.
1800 30th Street
Sussex One Suite 220-I
Boulder, CO 80301

BRECKENRIDGE

Colorado West Detox Center
501 North Park Avenue
Breckenridge, CO 80424-0754

BRIGHTON

Educational Center for Addictions
710 South Main Street
Brighton, CO 80601

Patterns for Positive Living
14 Main Street, Suite F
Brighton, CO 80601

BUCKLEY AFB

Buckley Air Force Base Substance Abuse Program
82 MDOS/SGOMH 275 South Aspen Street
Buckley AFB, CO 80011-9547

BUENA VISTA

Rocky Mountain Behavioral Health
715 East Main Street
Buena Vista, CO 81211-9799

BURLINGTON

Centennial Mental Health Center Alcohol and Drug Outpatient Program
1291 Circle Drive
Burlington, CO 80807

CANON CITY

Covenant Counseling
503 Main Street
Canon City, CO 81212

Rocky Mountain Behavioral Health, Inc.
618 Main Street
Canon City, CO 81215

CARBONDALE

Springs Counseling Center
1101 Village Road, Suite LL-2A
Carbondale, CO 81623-1571

CASTLE ROCK

Dynamic Directions
314 Wilcox Street
Castle Rock, CO 80104

CHEYENNE WELLS

Centennial Mental Health Center, Inc.
Keefe Memorial Hospital
Cheyenne Wells, CO 80810

COLORADO SPRINGS

Adult/Youth Counseling Service
223 North Wahsatch Avenue, Suite 101
Colorado Springs, CO 80903

Bridge to Awareness Counseling Center, Inc.
606 South Tejon Street
Colorado Springs, CO 80903

Cate Alcohol Education Program
4740 Flintridge Drive
Suite 201-B
Colorado Springs, CO 80918

Cedar Springs BHC Services, Inc.
2135 Southgate Road
Colorado Springs, CO 80906

Chemical Dependency Center
2741 East Las Vegas Street
Colorado Springs, CO 80906

Genesis
715 South Circle Drive
Suite 105
Colorado Springs, CO 80910

Gordon S. Riegel Chemical Dependency Center
825 East Pikes Peak Avenue
Colorado Springs, CO 80903

Health Challenge Counseling Center, Inc.
3750 Astrozon Boulevard
Suite A
Colorado Springs, CO 80910

McMaster Center/El Paso County Dept. of Health and Environment
301 South Union Boulevard
Colorado Springs, CO 80910

Pathways Confidential Counseling, Inc.
1767 South 8th Street
Suite 100
Colorado Springs, CO 80905

Pike's Peak Mental Health Center
Outpatient Division
179 Parkside Drive
Colorado Springs, CO 80910

Positive Change
1120 North Circle Street
Suite 11
Colorado Springs, CO 80909

Tombs Counseling Services
2860 South Circle Drive
Suite 2129
Colorado Springs, CO 80906

COMMERCE CITY

Adams Community Mental Health Center
7191 Holly Street
Commerce City, CO 80022

Insights Counseling Services, Inc.
6025 Parkway Drive, Suite 110
Commerce City, CO 80022-5412

CORTEZ

Cortez Addiction Recovery Services, Inc.
35 North Ash Street
Cortez, CO 81321

CRAIG

Yampa Valley Psychotherapists
2045 West Victory Way
Craig, CO 81625

DELTA

Creative Counseling Place
550 Palmer Street, Suite 103
Delta, CO 81416

Midwestern Colorado Mental Health Center
Center for Mental Health
195 Stafford Lane
Delta, CO 81416

Options Counseling Center
261 Hartig Drive
Suite A North
Delta, CO 81416

DENVER

Addiction Residence Treatment Services (ARTS) Peer I Residential Treatment Facility
3712 West Princeton Circle
Denver, CO 80236

Adolescent Counseling Exchange
948 Santa Fe Drive
Denver, CO 80204

Alcohol Counseling Services of Colorado, Inc.
1300 South Lafayette Street
Denver, CO 80210

Alpar Human Development Services Alcohol Outpatient Treatment
1330 Leyden Street, Suite 103
Denver, CO 80222

Anchor Counseling, Inc.
1009 Grant Street, Suite 50
Denver, CO 80203

A Treatment Agency Inc
1745 South Federal Boulevard
Denver, CO 80219-4861

Bi-Community Correctional Services Denver Day Reporting Center
1555 Clarkson Street
Denver, CO 80203

Bridge Counseling Center
2422 South Federal Boulevard
Denver, CO 80219

Broadway Counseling Services
725 South Broadway Street
Suite 16
Denver, CO 80209

Center for Behavioral Health
2465 South Downing Street
Suite 110
Denver, CO 80210

Center for Human Development Colorado MOVES
2345 South Federal Boulevard
Suite 103
Denver, CO 80219

Choosing Life Center
1626 High Street
Denver, CO 80218

Colorado Inhalant Abuse Program
1115 Broadway Street, Suite 102
Denver, CO 80203

Columbia Health One Presbyterian/Saint Luke's Medical Center
1719 East 19th Avenue
Denver, CO 80218

Community Alcohol/Drug Rehab and Education Center (CADREC)
3315 Gilpin Street
Denver, CO 80205

Comprehensive Addiction Treatment Services
2222 East 18 Avenue
Denver, CO 80206

Denver Area Youth Services/ Days Adolescent Substance Abuse Treatment
1240 West Bayaud Avenue
Denver, CO 80223

Denver Health Community Detox Behavioral Health Services
1155 Cherokee Street
Denver, CO 80204

Denver Indian Health and Family Services
3749 South King Street
Denver, CO 80236

Dry Creek Treatment Center
222 Milwaukee Street, Suite 408-B
Denver, CO 80206-5008

Essex Growth Center
2789 West Alameda Street
Denver, CO 80219

Fresh Start, Inc.
2250 East 16th Avenue
Denver, CO 80206

Gateway Treatment Center
1250 South Parker Road
Suite 103
Denver, CO 80231

IDEA
2828 North Speer Boulevard
Suite 116
Denver, CO 80211

Inner Connections, Inc.
1556 Williams Street
Denver, CO 80218

Insights Counseling Services, Inc.
2200 East 104th Avenue
Suite 213
Denver, CO 80233

Maria Droste Services of Colorado
1355 South Colorado Boulevard
Suite C-100
Denver, CO 80222

Metropolitan Counseling Services
1601 South Federal Boulevard
Heritage Plaza Suite 115
Denver, CO 80219

Mile High Club Alcohol Abuse Halfway House
1444 Wazee Street, Suite 125
Denver, CO 80205

Multi Addictions Processing Agency (MAPA)
1650 Franklin Street, Lower Level
Denver, CO 80218

Outpatient Behavioral Health Services (OBHS)
320 West 8th Avenue, Unit 2
Denver, CO 80204

Rebound Foundation Adventures in Change
3445 West Mansfield Street
Denver, CO 80236

Servicios de la Raza Alcohol Abuse Program
4055 Tejon Street
Denver, CO 80211

Sobriety House, Inc. Stepping Stone
107 Acoma Street
Denver, CO 80223

Southwest Family Services
1800 South Sheridan Boulevard
Suite 303
Denver, CO 80232

Special Services Clinic, Inc.
301 Knox Court
Denver, CO 80219

Stout Street Foundation
1609 Gaylord Street
Denver, CO 80206-1206

1647 Gaylord Street
Denver, CO 80206

UCHSC Addiction Research Treatment Services (ARTS) Outpatient Clinic
1827 Gaylord
Denver, CO 80206

University of Colorado Health Science Center Addiction Research Treatment Service
3738 West Princeton Circle
Denver, CO 80236

Veterans' Affairs Medical Center
Substance Abuse Program
1055 Clermont Street
Denver, CO 80220

Wellspring Alcohol and Drug Abuse Services
1660 South Albion Street
Suite 600
Denver, CO 80222

Western Clinical Health Services
1038 Bannock Street
Denver, CO 80204

Youthtrack Alliance
920 Clarkson Street
Denver, CO 80128

DURANGO

La Plata Counseling South West Community Corrections
1111 Camino Del Rio Street
Durango, CO 81301

Southwest Colorado Mental Health Center Detoxification
281 Sawyer Drive
Durango, CO 81301

ELIZABETH

Centennial Mental Health Center Alcohol and Drug Outpatient Program
349 East Washington Street
Elizabeth, CO 80107

ENGLEWOOD

Mile HI Counseling, Inc.
300 East Hampden Avenue
Suite 230
Englewood, CO 80110

Valley Hope Alcohol/Drug Center
8000 East Prentice Avenue
Suite D-12
Englewood, CO 80111-2727

Alternative Pathways, Inc.
4195 South Broadway Street
Englewood, CO 80110-4632

ESTES PARK

Harmony Foundation, Inc. Alcohol/Drug Abuse Program
1600 Fish Hatchery Road
Estes Park, CO 80517

EVERGREEN

Mountain Treatment Services
6949 Highway 73
Evergreen, CO 80439

FAIRPLAY

Rocky Mountain Behavioral Health, Inc.
1271 Castello Avenue, Suite B
Fairplay, CO 80440

FLORENCE

Clear View Center
521 West 5th Street
Florence, CO 81226

FORT CARSON

U.S. Army MEDDAC Community Counseling Center
MCXE/AS/MH/AD
Specker and Ellis Street
Fort Carson, CO 80913

FORT COLLINS

Center for Life Skills Education
400 West Magnolia Street
Fort Collins, CO 80521

Hope Counseling Center
301 East Olive Street
Fort Collins, CO 80524

Larimer County Institute for Alcohol Awareness
253 Linden Street
Suite 206
Fort Collins, CO 80524

Managed Adolescent Care Center
400 Remington Street, Suite 202
Fort Collins, CO 80525

Mountain Crest Behavioral Healthcare System Addictive Disease Unit (ADU)
4601 Corbett Drive
Fort Collins, CO 80528

Seven Lakes Recovery Program
2362 East Prospect Avenue
Fort Collins, CO 80525

FORT MORGAN

Centennial Mental Health Center
910 East Railroad Street
Fort Morgan, CO 80701

FRANKTOWN

Running Creek Counseling Service
7601 Burning Tree Drive
Franktown, CO 80116

FRASER

Rangeview Counseling Center
193 County Road, Suite 804
Fraser, CO 80442

FRISCO

Bair Counseling Center
619 Main Street
Frisco, CO 80443

Columbine Recovery Center
1000 North Summit Boulevard
Suite 200
Frisco, CO 80443

GLENWOOD SPRINGS

Valley View Youth Recovery Center
1906 Blake Avenue
Glenwood Springs, CO 81601

GOLDEN

Counseling Evaluation and Treatment Program Inc
607 10th Street, Suite 103
Golden, CO 80401

Division of Youth Services Lookout Mountain School
2901 Ford Street
Golden, CO 80401

GRAND JUNCTION

Adult Adolescent Alcohol Treatment (AAAT)
726 Colorado Avenue
Grand Junction, CO 81506

Colorado West Mental Health Center
450 Ouray Street
Grand Junction, CO 81501

Counseling Works
1904 North 7th Street
Grand Junction, CO 81501

Division of Youth Services
360 28th Road
Grand Junction, CO 81501

Dos Rios Counseling Service
1008 North 5th Street
Grand Junction, CO 81501

Saint Mary's Recovery Services
436 South 7th Street
Grand Junction, CO 81501

Outpatient Site
744 Horizon Drive, Suite 210
Grand Junction, CO 81501

In Roads Counseling
1141 North 25 Street
Suite F
Grand Junction, CO 81501

Veterans' Affairs Medical Center Substance Abuse Treatment Program (SATP)
2121 North Avenue
Building 6
Grand Junction, CO 81501

GREELEY

ARC Counseling Center
1122 9th Street, Suite 102
Greeley, CO 80631-3277

Island Grove Regional Treatment Center, Inc.
1140 M Street
Greeley, CO 80631

Psychcare Family Recovery Center
928 12th Street
Greeley, CO 80631

Residential Treatment Center
1776 6 Avenue
Greeley, CO 80631

HIGHLANDS RANCH

Addiction Treatment Outpatient Services
7120 East County Line Road, Suite 101
Highlands Ranch, CO 80126

HOLYOKE

Centennial Mental Health Center Substance Abuse Services
109 North Campbell Street
Holyoke, CO 80734

IDAHO SPRINGS

Chicago Creek Roads, Inc. Hopeful Futures
984 Highway 103
Idaho Springs, CO 80452

Clear Creek Counseling
1504 Main Street
Idaho Springs, CO 80452

IGNACIO

Southern Ute Alcohol Recovery Center Peaceful Spirit
296 Mouache Road
Ignacio, CO 81137

JULESBURG

Centennial Mental Health Center Substance Abuse Services
115 Elm Street
Julesburg, CO 80737

LAFAYETTE

Lafayette Alcohol Education and Therapy
201 East Simpson Street
Suite 201-B
Lafayette, CO 80026

LA JARA

SLV Family Resources
304 Walnut Street
La Jara, CO 81140

LA JUNTA

Pathfinders
207 1/2 Colorado Avenue
La Junta, CO 81050

LAKEWOOD

Alternative Behaviors Counseling
1949 Wadsworth Street
Suite 206
Lakewood, CO 80215

Alternative Homes for Youth/ Cedars
5400 West Cedar Avenue
Lakewood, CO 80226

Attitude Development Services
12211 West Alameda Parkway, Suite 220
Lakewood, CO 80228

Bi Day Reporting
2099 Wadsworth Boulevard
Lakewood, CO 80215-3383

Cenikor Foundation, Inc. Alcohol and Drug Abuse Program
1533 Glen Ayr Drive
Lakewood, CO 80215

Community Resource for Alcohol and Family Treatment
200 South Sheridan Boulevard, Suite 240
Lakewood, CO 80226

Crossroads Counseling, Ltd.
8000 West 14 Avenue
Suite 1
Lakewood, CO 80215

Empowerment Counseling Services, Inc.
1675 Carr Street
Suite 110-B
Lakewood, CO 80215

Jefferson County Health Department Substance Abuse Counseling Program
260 South Kipling Street
Lakewood, CO 80226

DBA Family Counseling Center
7430 West 16th Avenue
Lakewood, CO 80215

Milestone Counseling Services Inc
8533 West Colfax Avenue
Lakewood, CO 80215

Serenity Education and Therapy
2255 South Wadsworth Boulevard
Suite G-3
Lakewood, CO 80226

Touchstone Counseling Center
777 South Wadsworth Boulevard
Irongate 2 Suite 205
Lakewood, CO 80226

LAMAR

New Lifestyles
105 South 5th Street
Lamar, CO 81052

Southeast Colorado for Drug Free Communities
1006 South Main Street
Lamar, CO 81052

LAS ANIMAS

Resada Alcohol and Drug Abuse Program
11000 Road, Garage 5
Las Animas, CO 81054

LEADVILLE

Mount Hope Recovery, Inc.
130 West 5th Street
Leadville, CO 80461

LIMON

Centennial Mental Health Center Substance Abuse Services
606 Main Street
Limon, CO 80828

LITTLETON

Alternative Counseling
1500 West Littleton Boulevard, Suite 201
Littleton, CO 80120

Arapahoe Mental Health Center Aquarius Center/Sami
5500 South Sycamore
Littleton, CO 80120

LONGMONT

Longmont United Hospital Addiction
1331 Linden Street
Longmont, CO 80501

LOVELAND

Hope Counseling Center
446 North Garfield Avenue
Loveland, CO 80537

Larimer County Institute for Alcohol Awareness
314 East 4th Street
Loveland, CO 80537-5604

MONTROSE

Midwestern Colorado Mental Health Center The Center for Mental Health/Substance Abuse Services
605 East Miami Road
Montrose, CO 81401

Montrose Memorial Hospital Care Center
800 South 3rd Street
Montrose, CO 81401

Touchstone Counseling
118 North Cascade Street
Montrose, CO 81401

MORRISON

Lost and Found, Inc. Substance Abuse Program
9189 South Turkey Creek Road
Morrison, CO 80465

NORWOOD

Midwestern Colorado Mental Health Center The Center for Mental Health/Norwood
1510 West Grand Avenue
Norwood, CO 81423

PAGOSA SPRINGS

Rio Blanco Counseling Center
244 Pagosa Street
Pagosa Springs, CO 81147

PARKER

First Step Counseling
10290 South Progress Way
Suite 105
Parker, CO 80134

Parker Valley Hope
22422 East Main Street
Parker, CO 80138

PETERSON AFB

Peterson Air Force Base Substance Abuse Program
21 MDOS/SGOMH 559 Vincent Street
Peterson AFB, CO 80914-7804

PUEBLO

Associates for Psychotherapy and Education, Inc.
229 West 12th Street
Pueblo, CO 81003-2810

Awareness Institute, Inc.
1245 Palmer Avenue, Suite 210
Pueblo, CO 81004

Colorado Mental Health Institute at Pueblo Circle Program
1600 West 24 Street
Building 116
Pueblo, CO 81003

Crossroads Managed Care Systems, Inc.
509 East 13th Street
Pueblo, CO 81001

1700 East Evans Street
Pueblo, CO 81004

Parkview Episcopal Medical Center Chemical Dependency Program
58 Club Manor Drive
Pueblo, CO 81008

Pueblo Youth Services Bureau Substance Abuse Services
112 West D Street
Pueblo, CO 81003

STERLING

Centennial Mental Health Sterling Office Substance Abuse Services
211 West Main Street
Sterling, CO 80751

THORNTON

Arapahoe House, Inc.
8801 Lipan Street
Thornton, CO 80221

Empowerment Counseling Services
9101 North Pearl Street, Suite 231
Thornton, CO 80229

TRINIDAD

Crossroads Managed Care Systems, Inc.
Outpatient Care
1004 Carbon Place
Trinidad, CO 81082

WALSENBURG

Crossroads Managed Care Systems, Inc.
622 South Albert Street
Walsenburg, CO 81089

WHEAT RIDGE

Adolescent and Family Institute of Colorado, Inc.
10001 West 32nd Avenue
Wheat Ridge, CO 80033

Choices in Living Counseling Center, Inc.
7100 West 44 Avenue
Suite 102
Wheat Ridge, CO 80033

Family Violence Program
4243 Harlan Street
Wheat Ridge, CO 80033-5119

West Pines Hospital at Lutheran Medical Center
3400 Lutheran Parkway
Wheat Ridge, CO 80033

WOODLAND PARK

Journeys Counseling and Education Center
320 Burdette Street
Woodland Park, CO 80863

WRAY

Prairie Land Recovery Center
340 South Birch Street
Wray, CO 80758

YUMA

Centennial Mental Health Center Alcohol and Drug Outpatient Program
215 South Ash Street
Yuma, CO 80759

CONNECTICUT

ANSONIA

Lower Naugatuck Valley Council on Alcohol/Drug Abuse Alcoholism and Drug Abuse Program
75 Liberty Street
Ansonia, CT 06401

AVON

Reid Treatment Center
Intensive Treatment
121 West Avon Road
Avon, CT 06001

BLOOMFIELD

Blue Ridge Center Administrative Unit
1095 Blue Hills Avenue
Bloomfield, CT 06002

BRANFORD

Branford Counseling Center Outpatient
342 Harbor Street
Branford, CT 06405

BRIDGEPORT

Chemical Abuse Services Agency, Inc. (CASA) Administrative Unit
690 Artic Street
Bridgeport, CT 06604

Greater Bridgeport Community Mental Health Center Acute Substance Abuse Treatment Unit
1635 Central Avenue
Bridgeport, CT 06610

Helping Hand Center
488 Stratford Avenue
Bridgeport, CT 06608

1124 Iranistan Avenue
Bridgeport, CT 06608

Horizons
Intensive Residential Drug Free Program
1635 Fairfield Avenue
Bridgeport, CT 06605

Regional Network of Programs, Inc.
Administrative Unit
Golden Hill Treatment Center/ Detox
Methadone Maintenance
Regional Adolescent Program (RAP)
171 Golden Hill Street
Bridgeport, CT 06604

Center for Human Services
1549 Fairfield Avenue
Bridgeport, CT 06605

Regional Counseling Services
480 Bond Street
Bridgeport, CT 06610

BRIDGEWATER

Midwestern Connecticut Council on Alcoholism
McDonough House/Intensive and Intermediate Residential Programs
132 Hut Hill Road
Bridgewater, CT 06752

BRISTOL

Behavioral Health Services
25 Newell Road Suite D-20
Bristol, CT 06010

Bristol Hospital Behavioral Health Services
440 C North Main Street
Bristol, CT 06010

Counseling Center of Bristol Hospital Evening Chemical Dependency Program
440 C North Main Street
Bristol, CT 06010

CANAAN

Mountainside Lodge, Inc.
187 South Canaan Road
Route 7
Canaan, CT 06018

DANBURY

Danbury Youth Services, Inc.
32 Stevens Street
Danbury, CT 06810

Midwestern Connecticut Council on Alcoholism
Outpatient Unit
Women's Program
238 White Street
Danbury, CT 06810

DANIELSON

Community Prevention/ Addiction Services Outpatient Program
37 Commerce Avenue
Danielson, CT 06239

Perception Programs, Inc.
New Perceptions 232 Broad Street
Danielson, CT 06239

DARIEN

Youth Options Darien Unit
120 Brookside Road
Darien, CT 06820

DAYVILLE

United Services, Inc. Alcohol and Drug Abuse Services
1007 North Main Street
Dayville, CT 06241

DERBY

Griffin Hospital
241 Seymour Avenue
Derby, CT 06418

EAST HARTFORD

Paces Counseling Associates Inc
991 Main Street, Suite 3-A
East Hartford, CT 06108

ENFIELD

New Directions, Inc. of North Central Connecticut
55 Main Street
Enfield, CT 06082

Evening Treatment
Intensive Outpatient
5102 Bigelow Commons
Enfield, CT 06082

FAIRFIELD

Fairfield Community Services
370 Beach Road
Fairfield, CT 06430

FARMINGTON

John Dempsey Hospital Alcohol and Drug Abuse Treatment Center
263 Farmington Avenue
Farmington, CT 06030

GLASTONBURY

Clayton House
203-205 Williams Street
Glastonbury, CT 06033

Rushford Center at Glastonbury
124 Hebron Avenue
Glastonbury, CT 06033

GREENWICH

Greenwich Hospital Recovery Program
Perryridge Road
Greenwich, CT 06830

GROTON

Connection, Inc. Women's Services of Groton
542 Long Hill Road
Groton, CT 06340

Counseling and Assistance Center
Box 27
Groton, CT 06349

HAMDEN

Wakeman Hall at the Children's Center
1400 Whitney Avenue
Hamden, CT 06517

HARTFORD

Alcohol and Drug Recovery Centers, Inc.
Coventry House/Pregnant Women's Program
46 Coventry Street
Hartford, CT 06112

Detoxification Center
500 Vine Street
Hartford, CT 06112

Ceder Crest Regional Hospital Blue Hills Substance Services
51 Coventry Street
Hartford, CT 06112

Community Health Services, Inc. Chemical Dependency Program
520 Albany Avenue
Hartford, CT 06120

Community Substance Abuse Centers
55 Fishfry Street
Hartford, CT 06120

Ambulatory Detox
Methadone Maintenance
55 Fishfry Street
Hartford, CT 06120

Greater Hartford Multiservice Center
136 Collins Street
Hartford, CT 06105

Hartford Dispensary Clinic
345 Main Street
Hartford, CT 06106

Henderson/Johnson Clinic
Methadone Maintenance Program
12 Weston Street
Hartford, CT 06120

Hispanic Alcohol and Substance Abuse Program
80 Jefferson Street
Hartford, CT 06106

Hogar Crea International of Connecticut, Inc.
33 Center Street
Hartford, CT 06120

Methadone to Abstinence Program Outpatient Methadone Maintenance
14 Weston Street
Hartford, CT 06120

Outpatient Counseling Center of ADRC Inc
16 Conventry Street
Hartford, CT 06112

Salvation Army Adult Rehab Center Alcohol Abuse Program
333 Homestead Avenue
Hartford, CT 06112

Wheeler Clinic, Inc. Hartford Outpatient
645 Farmington Avenue
Hartford, CT 06115

Youth Challenge of Greater Hartford
15–17 May Street
Hartford, CT 06105

Youth Challenge Mission for Women
32 Atwood Street
Hartford, CT 06105

LAKEVILLE

McCall Foundation, Inc.
c/o Northwest Center for Mental Health
315 Main Street
Lakeville, CT 06039

LEBANON

Southeast Council on Alcohol and Drug Dependency, Inc. (SCADD) Lebanon Pines Long-term Treatment
37 Camp Moween Road
Lebanon, CT 06249

LITCHFIELD

McAuliffe Manor
7 North Street
Litchfield, CT 06759

MANCHESTER

Community Prevention/ Addiction Services
87-B Oak Street
Manchester, CT 06040

New Hope Manor, Inc. Residential
48 Hartford Road
Manchester, CT 06040

MANSFIELD CENTER

Natchaug Hospital, Inc.
Adult Inpatient Detox
Quinnebaug Day Treatment Center
Sachem Adult Partial
189 Storrs Road
Mansfield Center, CT 06250

MERIDEN

Midstate Medical Center
435 Lewis Avenue
Meriden, CT 06451

MIDDLEBURY

Cornerstone Continuous Care
900 Straits Turnpike
Middlebury, CT 06762

MIDDLETOWN

Connecticut Valley Hospital Addiction Services Division
Silver Street
Middletown, CT 06457

Connection, Inc.
99 Eastern Drive
Middletown, CT 06457

Connection House
167 Liberty Street
Middletown, CT 06457

Greater Middletown Counseling Center
196 Court Street
Middletown, CT 06457

Rushford Center, Inc.
Administrative and Prevention Unit
Outpatient Uit
Intensive Residential Unit
Intermediate Residential Unit
MISA/Healthy Living Program
Non-Hospital Medical Unit
Partial Hospitalization Program
1250 Silver Street
Middletown, CT 06457

MILFORD

Milford Mental Health Clinic, Inc.
Administrative Unit
Outpatient
Peer Counseling Program
949 Bridgeport Avenue
Milford, CT 06460

MONROE

Regional Network of Programs, Inc. Monroe Builds Communication
1014 Monroe Turnpike
Masuk High School
Monroe, CT 06468

MOOSUP

Youth Challenge Bible Training Center
Long-term Training/Rehab
111 North Sterling Road
Moosup, CT 06354

NEW BRITAIN

Farrell Treatment Center
Outpatient Unit
Intensive Residential Unit
586 Main Street
New Britain, CT 06051

Hartford Dispensary/New Britain Clinic
19 Rockwell Avenue
New Britain, CT 06051

New Britain General Hospital Dept. of Behavioral Health
100 Grand Street
New Britain, CT 06050

Wheeler Clinic, Inc. Lifeline/ Pregnant Women's Program
35 Russell Street
New Britain, CT 06052

NEW HAVEN

Affiliates for Consultation and Therapy
389 Orange Street
New Haven, CT 06511

Alcohol Services Organization of South Central Connecticut, Inc.
871 State Street
New Haven, CT 06511

Apt Foundation, Inc.
Central Treatment Unit/Women with Children
1 Long Wharf Drive, Suite 10
New Haven, CT 06519

Legion Avenue Clinic/Methadone
60–62 Legion Avenue
New Haven, CT 06519

Orchard Clinic
Park Hill clinic
Women in Treatment
540 Ella T Grasso Boulevard
New Haven, CT 06519

Connecticut Mental Health Center
Substance Abuse Treatment Outpatient Program
1 Long Wharf Drive
New Haven, CT 06511

Dept. of Psychiatry
34 Park Street
New Haven, CT 06508

Crossroads, Inc.
54 East Ramsdell Street
New Haven, CT 06515

Amethyst House
48 Howe Street
New Haven, CT 06511

Hill Health Corporation
232 Cedar Street
New Haven, CT 06519

Hill Health Center/Northside Community Outpatient Services
226 Dixwell Avenue
New Haven, CT 06511

Multicultural Ambulatory Addiction Services
426 East Street
New Haven, CT 06511

NEWINGTON

Veterans Affairs Medical Center Substance Abuse Services
555 Willard Avenue, Suite 116-A
Newington, CT 06111

NEW LONDON

Care Center
516 Vauxhall Street
Suite 102
New London, CT 06320

Care Plus
190 Governor Winthrop Boulevard, Suite 101
New London, CT 06320

Hartford Dispensary New London Clinic
931 Bank Street
New London, CT 06320

Southeast Council on Alcohol and Drug Dependency, Inc. (SCADD)
Altruism House/Women
1000 Bank Street
New London, CT 06320

189 Howard Street
New London, CT 06320

Outpatient Program Detox
47 Colt Street
New London, CT 06320

NEW MILFORD

New Milford Youth Agency
50 East Street
New Milford, CT 06776

NEWTOWN

Apt Foundation, Inc.
Alpha House
Daytop Intermediate and Long Term Program
Mile Hill Road
Newtown, CT 06470

NORTH STONINGTON

Stonington Institute
Partial Hospitalization Program
75 Swantown Hill Road
North Stonington, CT 06359

NORWALK

Connecticut Counseling Centers, Inc.
Norwalk Methadone Program
Norwalk Outpatient Treatment Program
20 North Main Street
Norwalk, CT 06854

Connecticut Renaissance, Inc.
Administrative Unit
Norwalk Outpatient Unit
83 Wall Street
Norwalk, CT 06850

Dept of Psychiatry and Addictions Norwalk Hospital
24 Stevens Street
Norwalk, CT 06856

Family and Childrens Agency, Inc. Project Reward
165 Flax Hill Road
Norwalk, CT 06854

Liberation and Meridian Partners in Recovery
4 Elmcrest Terrace
Norwalk, CT 06850

Pivot Ministries, Inc.
17 Quintard Avenue
Norwalk, CT 06854

Vitam Center, Inc.
Administrative Unit
Residential Drug Free Unit
57 West Rocks Road
Norwalk, CT 06852

NORWICH

Hartford Dispensary Norwich Clinic
Norwich Hospital
Lippett Building
Norwich, CT 06360

SE Council on Alcohol and Drug Dependency, Inc. (SCADD) Altruism House/ Male
313 Main Street
Norwich, CT 06360

OLD SAYBROOK

The Connection, Inc. Valley Shore Counseling Center
263 Main Street, Suite 108
Old Saybrook, CT 06475

ORANGE

Family Service of Greater Waterbury
35 Porter Avenue
Orange, CT 06477

PLAINVILLE

Wheeler Clinic, Inc.
Adolescent Screening and Treatment Program
Intensive Outpatient/Day Treatment Program
Night Treatment Program
91 Northwest Drive
Plainville, CT 06062

PORTLAND

Elmcrest Behavioral Health Network
25 Marlborough Street
Portland, CT 06480

Stonehaven
325 Main Street
Portland, CT 06480

PUTNAM

Community Prevention and Addictoion Services
391 Pomfret Street
Putnam, CT 06260

ROCKY HILL

Department of Veterans Affairs Veterans Recovery Center
287 West Street
Rocky Hill, CT 06067

SHARON

Midwestern Connecticut Council on Alcoholism Trinity Glen
149 West Cornwall Road
Sharon, CT 06069

SOUTH WINDSOR

Connecticut North Treatment Center
15 Morgan Farms Drive
South Windsor, CT 06074

STAFFORD SPRINGS

Johnson Memorial Hospital
201 Chestnut Hill Road
Stafford Springs, CT 06076

Stafford Family Services
21 Hyde Park Road
Stafford Springs, CT 06076

STAMFORD

LMG, Inc.
115 Main Street
Stamford, CT 06901

Liberation Clinic
125 Main Street
Stamford, CT 06901

Viewpoint Recovery Program Intermediate Long Term
104–106 Richmond Hill Avenue
Stamford, CT 06902

STRATFORD

Family Resource Associates
3300 Main Street
Stratford, CT 06614

TORRINGTON

McCall Foundation, Inc.
Intensive Residential Program
Evening Program
Outpatient Program
58 High Street
Torrington, CT 06790

McCall House/Intermediate Residential
127 Migeon Avenue
Torrington, CT 06790

VERNON ROCKVILLE

Natchaug Hospital, Inc. River East Day Hosp Land Treatment Center
428 Hartford Turnpike Road
Vernon Rockville, CT 06066

WATERBURY

Center for Psychiatry and Clinical Neuroscience
1389 West Main Street, Suite 106
Waterbury, CT 06708

Central Naugatuck Valley Help, Inc.
Administrative Unit
Nonresidential Program
Residential Unit
900 Watertown Avenue
Waterbury, CT 06708

Connecticut Counseling Centers, Inc.
Waterbury Methadone Program
Waterbury Outpatient Program
4 Midland Road
Waterbury, CT 06708

Connecticut Renaissance, Inc. Residential Treatment Facility
31 Wolcott Street
Waterbury, CT 06702

Family Intervention Center
1875 Thomaston Avenue
Waterbury, CT 06704

Family Service of Greater Waterbury
34 Murray Street
Waterbury, CT 06710

Morris Foundation, Inc.
Administrative Unit
Center for Alcohol and Drug-Free Living
26 North Elm Street
Waterbury, CT 06702

Driving While Intoxicated
Therapeutic Shelter
142 Griggs Street
Waterbury, CT 06702

Morris/Kendall House
26 North Elm Street
Waterbury, CT 06702

Woman and Children Program
79 Beacon Street
Waterbury, CT 06702

Saint Mary's Hospital Behavioral Healthcare Services
56 Franklin Street
Waterbury, CT 06706

WEST HAVEN

Veterans Affairs Medical Center Substance Abuse Treatment Program
950 Campbell Avenue
Suite 116-A3
West Haven, CT 06516

WESTPORT

Alcohol and Drug Dependency Council, Inc.
420 Post Road West
Westport, CT 06880

Hall Brooke Hospital Substance Abuse Unit
47 Long Lots Road
Westport, CT 06881

WILLIMANTIC

Community Prevention/ Addiction Services
Thomas Murphy Center
1493 West Main Street
Willimantic, CT 06226

Hartford Dispensary Willimantic Clinic
54-56 Boston Post Road
Willimantic, CT 06226

Perception Program, Inc.
Perception House
134 Church Street
Willimantic, CT 06226

New Perspective Counseling Service
Right Turn Adolescent Program
90 South Park Street
Willimantic, CT 06226

United Services, Inc. Addiction Recovery Services
132 Mansfield Avenue
Willimantic, CT 06226

WINSTED

McCall Foundation, Inc.
231 North Main Street
Winsted, CT 06098

DELAWARE

CLAYMONT

Open Door, Inc.
3301 Green Street
Claymont, DE 19703

DELAWARE CITY

Cornerstone Alcohol and Drug Residential Program
New Castle Avenue, Building 8
Delaware City, DE 19706

Northeast Treatment Centers
Governor Bacon Health Center
Cottage 5
Delaware City, DE 19706

Reflection House
Delaware City, DE 19706

DOVER

ABR Counseling Associates
1550 South Governor Avenue
Dover, DE 19904

Kent County Counseling Services
1525 Lebanon Road
Dover, DE 19901

Pace Alcohol and Drug Counseling
707 Walker Road
Dover, DE 19904

Phoenix Mental Health of Dover
567 South Governors Avenue
Dover, DE 19904

Serenity Place
327 Martin Street
Dover, DE 19901

St. Jones Center for Behavioral Health
725 Horsepond Road
Dover, DE 19901

DOVER AIR FORCE BASE

Dover Air Force Base Substance Abuse Office
263 Chad Street
Dover Air Force Base, DE 19902

ELLENDALE

Kent/Sussex Detoxification Center
Main Street
Ellendale School House
Ellendale, DE 19941

GEORGETOWN

Children and Family First
410 South Bedford Street
Georgetown, DE 19947-1850

Corinthian House
219–221 South Race Street
Georgetown, DE 19947

Houston Hall
431 East Market Street
Georgetown, DE 19947

Psychotherapeutic Community Services Associates
16 North Railroad Avenue
Georgetown, DE 19947-1242

Tau House
11 West Pine Street
Georgetown, DE 19947

Thresholds, Inc. Sussex County Unit
526-D North Dupont Highway
113 Professional Building
Georgetown, DE 19947

MILFORD

People's Place Counseling Center
219 South Walnut Street
Milford, DE 19963

NEWARK

Newark Family Counseling Center
501 Ogletown Road
Hudson State Service Center
Newark, DE 19711

North East Treatment Center
7-D Peddlers Row Peddlers Village
Newark, DE 19702

NEW CASTLE

Women's Correctional Institution (WCI) Village
660 Baylor Boulevard
New Castle, DE 19720

SEAFORD

Behavioral Health Services
801 Middleford Road Nanticoke Memorial Hospital
Seaford, DE 19973

SELBYVILLE

ABR Counseling Associates/ Sussex County
33 Keenwik Road
Selbyville, DE 19975-0000

SMYRNA

Greentree Drug and Alcohol Program Delaware Correction Center
Route 1
Smyrna, DE 19977

WILMINGTON

Aquila
2110 Duncan Road
Wilmington, DE 19808

Brandywine Counseling, Inc.
2713 Lancaster Avenue
Wilmington, DE 19805

Riverfront Site
350 South Madison Street
Wilmington, DE 19801

Children and Family First
2005 Baynard Boulevard
Wilmington, DE 19802

Key Program
1301 East 12 Street
Wilmington, DE 19809

Limen House
624 North Broom Street
Wilmington, DE 19805

Limen House for Men
903 Madison Street
Wilmington, DE 19801

Net Counseling Center
813 West Street
Wilmington, DE 19801

Northeast Treatment Centers Kirkwood Detox
3315 Kirkwood Highway
Wilmington, DE 19808

Pace, Inc.
5171 West Woodmill Drive
Suite 9
Wilmington, DE 19808

Psychotherapeutic Services, Inc. Relapse Prevention/ Continuous Treatment
5207 West Woodmill Drive
Suite 34
Wilmington, DE 19808

Sodat Counseling and Evaluation Center
625 Orange Street
Wilmington, DE 19801

Wilmington Veterans Affairs Med Center
1601 Kirkwood Highway 116B
Wilmington, DE 19805

DISTRICT OF COLUMBIA

WASHINGTON

APRA/Karick Hall/PPWI
1900 Massachusetts Avenue SE
Building 17
Washington, DC 20003

Adams Mill Alcohol Treatment Center
1808 Adams Mill Road NW
Washington, DC 20009

Addiction Prevention and Recovery Administration
Detox Center
1900 Massachusetts Avenue SE
Building 12
Washington, DC 20003

Minimal Services
1300 1st Street NE
2nd Floor
Washington, DC 20002

Andromeda Transcultural Hispanic Mental Health Center
1400 Decatur Street NW
Washington, DC 20011

Bureau of Rehabilitation, Inc. Community Care Center
3301 16 Street NW
Washington, DC 20010

Clean and Sober Streets
425 2nd Street NW
Washington, DC 20001

Concerned Citizens on Alcohol and Drug Abuse
Pregnant/Postpartum Outpatient Women's Program
311 Martin Luther King Avenue SE
Washington, DC 20032

Consulting Counseling Center
3000 Connecticut Avenue NW
Suite 439
Washington, DC 20008-2556

DC Employee Consultation and Counseling Service
33 N Street NE, 2nd Floor
Washington, DC 20009

DC General Hospital
Dept. of Psychiatry Substance Abuse Program
Detox Center
1900 Massachusetts Avenue SE
DC General Hospital Unit 42
Washington, DC 20003

DC Lifeline Addiction Treatment Program
1901 East Street SE
Washington, DC 20003

Demeter Northwest of Vanguard Services Unlimited
301 I Street NW
Washington, DC 20001

Executive Addictive Disease Programs, Inc.
4335 Wisconsin Avenue NW
Washington, DC 20016

Family and Medical Counseling Services
2041 Martin Luther King Avenue SE
Suite M-2
Washington, DC 20020

Foundation for Contemporary Mental Health Next Step
2112 F Street NW
Suite 404
Washington, DC 20037

Georgetown Medical Center Alcohol and Drug Abuse Program
3800 Reservoir Road NW
Washington, DC 20007

Girard Treatment Center (GTC)
1413 Girard Street NW
Washington, DC 20009

Holy Comforter/Saint Cyprian Community Action Group/ Carriage House
901 Pennsylvania Avenue SE
Washington, DC 20003

House of Ruth Mothers/Infants Program
700 6th Street NE
Washington, DC 20003

Howard University Drug Abuse Institute
2041 Georgia Avenue NW
Suite 6B07
Washington, DC 20059

Institute for Behavioral Change
34 O Street NW
Washington, DC 20001

Karrick Hall Pregnant and Postpartum Women and Infant Program
1900 Massachusetts Avenue SE
Building 17
Washington, DC 20003

Koba Associates Diagnostic Unit
1300 First Street NE
Suite 214
Washington, DC 20003

Kolmac Clinic
1411 K Street NW
Suite 703
Washington, DC 20005

La Clinica del Pueblo Inc
1470 Irving Street NW
Washington, DC 20010-2804

Latin American Youth Center Substance Abuse Program
1419 Columbia Road NW
Washington, DC 20009

Mary E. Herring Safe House
700 Monroe Street NE
Washington, DC 20000

Metropolitan Psychiatric Group/ Mars
2021 K Street NW, Suite 206
Washington, DC 20006

Model Treatment Program
1300 First Street NE
Washington, DC 20002

Necessary Intervention for Adolescents
2146 24th Place NE
Washington, DC 20002

New Risings Women's Center
2146 24th Place NE, Suite 111
Washington, DC 20018

Oasis
910 Bladensburg Road NE
Washington, DC 20002

Partners in Drug Abuse Rehabilitation and Counseling (PIDARC)
2112 F Street NW
Suite 101
Washington, DC 20037

Pirgrim Rest Baptist Therapeutics
4606 Sheriff Road NE
Washington, DC 20019-3703

Professional Guidance Associates
1314 18th Street NW, Suite 300
Washington, DC 20036-1803

Progressive Life Center
1129 11th Street NW
Washington, DC 20001

Providence Hospital Substance Abuse Services
1053 Buchanan Street NE
Washington, DC 20017

Psychiatric Institute of Washington New Directions Recovery Center
4228 Wisconsin Avenue
Washington, DC 20016

Rap, Inc.
1949 4th St NW
Washington, DC 20002

Second Genesis Residential Therapeutic Community DC Clinic
1320 Harvard Street NW
Washington, DC 20009

Shaw Abstinence Program
602 N Street NW
Washington, DC 20002

So Others May Eat, Inc. (SOME)
60 O Street NW
Washington, DC 20001

Supervised Living Program Phase II and III
221 Orange Street SE
Washington, DC 20002

Umoja Treatment Center
5140 Nannie Helen Burroughs Avenue NE
Washington, DC 20019

Unfoldment, Inc.
2605 Wade Road SE
Barry Farms Dwellings
Washington, DC 20020

Veterans Affairs Medical Center Substance Abuse Treatment Program
50 Irving Street NW, Suite 116-A
Washington, DC 20422

Walter Reed Army Medical Center Community Counseling Ctr
6825 16th Street NW
Building 6, 2nd Floor
Washington, DC 20307

Ward and Ward Associates
7600 Georgia Avenue NW
Suite 100
Washington, DC 20012-1616

Washington Area Council on Alcoholism and Drug Abuse Inc./Comp Counseling Center
2813 12 Street NE
Washington, DC 20017

Washington Assessment/ Therapy Services
4455 Connecticut Avenue NW
Suite A-400
Washington, DC 20008

Whitman Walker Clinic, Inc. Mental Health and Addiction Treatment Services
1407 S Street NW
Washington, DC 20009

Women's Services Clinic
1900 Massachusetts Avenue SE
Building 13
Washington, DC 20003

FLORIDA

ALTAMONTE SPRINGS

Cornerstone Institute, Inc.
400 Maitland Avenue
Altamonte Springs, FL 32701

Quest Counseling Centre, Inc.
401 whooping Loop, Suite 1549
Altamonte Springs, FL 32701

Serenity Center
378 Whooping Loop, Suite 1238
Altamonte Springs, FL 32701

APOPKA

Addictions Compulsions Treatment Center/ACT Center Inc
325 West Main Street, Suite A
Apopka, FL 32712

ARCADIA

Coastal Recovery Centers, Inc. Arcadia Office
14 East Oak Street
Arcadia, FL 34266

BARTOW

Tri-County Addictions Rehab Services, Inc.
Detoxification Unit
Women's Residential
2725 Highway 60 East
Bartow, FL 33830

BAY PINES

Veterans' Affairs Medical Center Substance Abuse Treatment Program
10000 Bay Pines Boulevard
Bay Pines, FL 33744

BELLE GLADE

New Beginnings
149 Southeast Avenue, Suite D
Belle Glade

West Palm Beach County
Outpatient Substance Abuse Services
1024 NW Avenue D
Belle Glade, FL 33430

Panda/Mental Health Clinic
816 Northwest Avenue, Suite D
Belle Glade, FL 33430

BOCA RATON

Alternatives in Treatment, Inc.
7601 North Federal Highway
Suite 100
Boca Raton, FL 33487

Counseling Services Institute, Inc.
1515 North Federal Highway
Suite 216
Boca Raton, FL 33432

BOYNTON BEACH

Atlantic Counseling
200 Knuth Road
Suite 238
Boynton Beach, FL 33436

BRADENTON

Center for Rational/Emotive Therapy
4303 1st Street East 265
Bradenton, FL 34208

Inpatient Addictions Treatment Service
2020 26th Avenue East
Bradenton, FL 34208

Manatee Glens Corporation
Adolescent Recovery Center
1819 5 Street West
Bradenton, FL 34205

Outpatient Detox
2020 26th Avenue East
Bradenton, FL 34208

PAR Narcotic Addiction Treatment Center
5105 26th Street West
Bradenton, FL 34207

BRANDON

Personal Growth Counseling
113 Lithia Pinecrest Road, Suite A
Brandon, FL 33511

BRONSON

Meridian Behavioral Healthcare Inc
100 NE 90th Street
Bronson, FL 32621

BROOKSVILLE

Eckerd Family Youth Services
397 Culbreath Road
Brooksville, FL 34602

BUNNELL

Flagler City Outpatient Services
302 1/2 Moody Boulevard
Bunnell, FL 32110

CAPE CORAL

Bill Bohs MA/DBA Omega
1443 Delprado Boulevard
Cape Coral, FL 33990

CASTLEBURY

Spellman Counseling and Consulting, Inc.
274 Wilshire Boulevard, Suite 253
Castlebury, FL 32707

CITRA

Phoenix Houses of Florida
15681 North Highway 301
Citra, FL 32113

CLEARWATER

Fairwinds Treatment Center Residential
1569 South Fort Harrison Street
Clearwater, FL 34756

Family Services Centers
Clearwater Clinical Services
2188 58th Street
Clearwater, FL 33760

Focus One Inc
11681 49th Street North, Suite 8
Clearwater, FL 33762

Operation PAR, Inc.
Narcotic Addiction Treatment Center
4900 Creekside Drive
Suite 4908-B
Clearwater, FL 34620

DOC Day/Night Program
4914-B Creekside Drive
Turtle Creek Office Park
Clearwater, FL 33760

Ryan White Facility
Juvenile addiction Recovery Facility
Short-term Residential
Adult Outpatient
6150 150 Avenue North
Clearwater, FL 34620

CLERMONT

Lake Correctional Institution
19225 Route 27
Clermont, FL 34711

CLEWISTON

Hendry/Glades Mental Health Clinic, Inc.
601 West Alverdez Avenue
Clewiston, FL 33440-3504

COCOA

Alco Hall
1215 Lake Drive
Cocoa, FL 32922

Alco Rest Inc
1050 West King Street
Cocoa, FL 32922

Central Florida Treatment Center
7 North Cocoa Boulevard
Cocoa, FL 32922

Wenz Education and Counseling, Inc.
690 Friday Road
Cocoa, FL 32926

COOPER CITY

Florida Cooper Health Services, Ltd. DBA High Point
5960 SW 106th Avenue
Cooper City, FL 33328

CRAWFORDVILLE

Disc Village, Inc.
Wakula Human Services
Juvenile Outpatient
Adult Outpatient
Crawfordville, FL 32326

CROSS CITY

Kansas City Community Center, Inc. Cross City Correctional Institution
Old Radar Road
Cross City, FL 32628

DADE CITY

Harbor Behavioral Healthcare Institute East Pasco Outpatient
14527 7th Street
Dade City, FL 33525

DAYTONA BEACH

ACT Corporation Reality House
1341 Indian Lake Road
Daytona Beach, FL 32114

Adolescent Outpatient
955-G Orange Avenue
Daytona Beach, FL 32114

Counseling Associates of Port Orange
3959 South Nova Road, Suite 5
Daytona Beach, FL 32127

Daytona Methadone Treatment Center
737 Volusia Avenue
Daytona Beach, FL 32114

Miles and Associates/Daytona Beach Alcohol/Drug Intervention/Prevention Services
308 South Martin Luther King Boulevard
Daytona Beach, FL 32114

Stewart/Marchman Treatment Center, Inc.
Adult ARF
Detox Unit
1200 Red John Road
Daytona Beach, FL 32114

Adult Clinical Services
330 North Street
Daytona Beach, FL 32114

Salvation Army Residential Program
560 Ballough Road
Daytona Beach, FL 32114

Serenity House of Volusia, Inc.
547 High Street
Daytona Beach, FL 32114

DE FUNIAK SPRINGS

Cope Alcohol and Drug Program
3686 U.S. Highway 331 South
De Funiak Springs, FL 32433

DELAND

ACT Corporation De Land Outpatient Treatment
803 Woodland Boulevard
Deland, FL 32720

Community Outreach Services, Inc. De Land Residential/ Outpatient Unit 1
245 South Amelia Street
Deland, FL 32724

Memorial Hospital/West Volusia Psychiatric Services/ Substance Abuse Services
701 West Plymouth Avenue
Deland, FL 32720

Miles and Associates/De Land Alcohol/Drug Intervention/ Prevention Services
620 East New York Avenue
Suite A
Deland, FL 32720

Serenity West Farm
2775 Big John Drive
Deland, FL 32773

DELRAY BEACH

Beachcomber Family Treatment Center
4493 North Ocean Boulevard
Delray Beach, FL 33483

Drug Abuse Foundation of Palm Beach County
Linton Blvd. Unit
400 South Swinton Avenue
Delray Beach, FL 33444

Intervention Strategies, Inc.
495 NE 4th Street, Suite 2
Delray Beach, FL 33444

Pathways to Recovery, Inc. Residential and Extended Care Facility
13132 Barwick Road
Delray Beach, FL 33445

South County Mental Health Center Substance Abuse Treatment Program Unit 1
16158 South Military Trail
Delray Beach, FL 33484

Wayside House
378 NE 6th Avenue
Delray Beach, FL 33483

DELTONA

West Volusia Outpatient
1200 Deltona Boulevard Suite 20
Deltona, FL 32738

DUNEDIN

Rational Steps Main Street Psychiatric Associates
1605 Main Street
Dunedin, FL 34698

EGLIN AIR FORCE BASE

Eglin Air Force Base Substance Abuse Program
96 MDOS/SGOHA
Eglin AFB, FL 32542-6832

Substance Abuse Recovery Center
307 Boatner Road Suite 114
Eglin AFB, FL 32542

FERNANDINA BEACH

Nassau County Mental Health Alcohol and Drug Abuse Council
1890 South 14th Street
Suite 312-320
Fernandina Beach, FL 32034-4740

FERN PARK

Seminole Community Mental Health Center
237 Fernwood Boulevard
Fern Park, FL 32730

FLORIDA CITY

Miami Dade Office of Rehab Services Diversion and Treatment Program South
1600 NW 6th Court
Florida City, FL 33034

FORT LAUDERDALE

Alternative Substance Abuse Systems, Inc.
208 SE 8th Street
Fort Lauderdale, FL 33301

Broward Addiction Recovery Center (BARC)
1000 SW 2 Street
Fort Lauderdale, FL 33312

ATACC
Drug Court Treatment
601 South Andrews Street
Fort Lauderdale, FL 33301

Broward House
Chemical Dependency Treatment Program
417 Southeast 18th Street
Fort Lauderdale, FL 33316

West/Lauderdale Lakes
4487 North State Road 7
Fort Lauderdale, FL 33319

Family Institute/Fort Lauderdale
1144 SE 3 Avenue
Fort Lauderdale, FL 33316

Fort Lauderdale Counseling Services
1215 SE 2 Avenue
Fort Lauderdale, FL 33316

Lifeline of Miami
6550 Griffin Road, Suite 104
Fort Lauderdale, FL 33314

South Florida Counseling
3015 North Ocean Boulevard
Suite 109
Fort Lauderdale, FL 33308

Spectrum Programs, Inc.
5910 Northwest 9th Avenue
Fort Lauderdale, FL 33309

Adult Residential Services
2301 Wilton Drive
Fort Lauderdale, FL 33305

Broward Outpatient
2800 West Oakland Park Boulevard
Suite 100
Fort Lauderdale, FL 33311

Sunrise Regional Medical Center
555 SW 148th Avenue
Fort Lauderdale, FL 33325

FORT MYERS

Bill Bohs MA Omega Centre
8695 College Parkway, Suite 252
Fort Myers, FL 33919

Charter Glade Hospital Chemical Dependency Unit
3550 Colonial Boulevard
Fort Myers, FL 33906

Ruth Cooper Center
4424 Michigan Avenue
Apartment 507
Fort Myers, FL 33916

Drug Abuse Unit
2789 Ortiz Avenue SE
Fort Myers, FL 33905

Serenity Center
2709 Second Street
Fort Meyers, FL 33916

Southwest Florida Addiction Services, Inc.
Detoxification
2562 Dixie Parkway
Fort Myers, FL 33901

Residential and Outpatient
2101 McGregor Boulevard
Fort Myers, FL 33901

Residential Level 2
2450 Prince Street
Fort Myers, FL 33901

FORT PIERCE

Alpha Health Services
1025 Orange Avenue
Fort Pierce, FL 34950

Drug Abuse Treatment Association, Inc. (DATA) Norman C. Hayslip Treatment Center
4590 Selvitz Road
Fort Pierce, FL 34981

New Horizons of the Treasure Coast, Inc.
Detoxification Unit
800 Avenue H
Fort Pierce, FL 33950

Saint Lucie County Outpatient Branch
709 South 5th Street
Fort Pierce, FL 34950

FORT WALTON BEACH

Bridgeway Center Addiction/ Substance Abuse Program
205 Shell Avenue SE
Fort Walton Beach, FL 32548

GAINESVILLE

Bridges of America Cross Creek Bridge
3361 NE 39th Avenue
Gainesville, FL 32609

Corner Drug Store, Inc.
Alachua Halfway House
3430 Northeast Avenue
Gainesville, FL 32601

Outpatient Services
1300 NW 6 Street
Gainesville, FL 32601

Diversified Human Services
2830 NW 41st Street
Thornbrook III Building M
Gainesville, FL 32606

Meridian Behavioral Healthcare, Inc.
Gainesville, FL 32608

Sid Martin Bridge Street
4400 Southwest 13th Street
Gainesville, FL 32608

Metamorphosis Alachua County Drug Abuse Program
4201 Southwest 21st Place
Gainesville, FL 32607

North Florida Evaluation and Treatment Center
1200 NE 55th Boulevard
Gainesville, FL 32641

North Florida/South Georgia Veterans Health System
1601 SW Archer Road
116A SATT
Gainesville, FL 32608

GREENVILLE

Greenville Hills Academy
SW 22nd Avenue
Greenville, FL 32331

GULF BREEZE

The Friary
4400 Hickory Shores Boulevard
Gulf Breeze, FL 32561

Twelve Oaks Alcohol and Drug Recovery Center Detox
2068 Healthcare Avenue
Route 1
Gulf Breeze, FL 32566

HIALEAH

ACF Counseling Center, Inc.
102 East 49th Street
Hialeah, FL 33013

Citrus Health Network, Inc.
4175 West 20th Avenue
Hialeah, FL 33012-5874

Dade Family Counseling Center
1490 West 49 Place
Suite 390
Hialeah, FL 33012

Substance Abuse Control Center, Inc.
Family Services/A New Life, Inc.
1095 East 4th Avenue
Hialeah, FL 33010

HILLARD

Nassau County Mental Health, Alcohol, and Drug Council, Inc. Outpatient/Prevention
333 Eastwood Road
Hilliard, FL 32046

HOLLY HILL

Milestones, Inc. Center for Substance Abuse Intervention
484 LPGA Boulevard
Holly Hill, FL 32117

HOLLYWOOD

Broward Addiction Recovery Center (BARC) South
6491–Taft Street
Hollywood, FL 33024

Lock Towns CMHC Sub. Arts Project/Dade County Dual Diagnosis
1000 SW 84 Avenue
Hollywood, FL 33025

Memorial Regional Hospital Share Program
801 SW Douglas Road
Hollywood, FL 33025

Phoenix Group, Inc. Advanced Behavioral Care
668 North Dixie Highway
Hollywood, FL 33020

Spectrum Programs, Inc.
2219 Hollywood Boulevard
Suite 102
Hollywood, FL 33020

The Starting Place, Inc.
2057 Coolidge Street
Hollywood, FL 33020

HOMESTEAD

Associates for Psychological Services Homestead Alcohol Abuse Program
225 NE 8 Street
Suite 3
Homestead, FL 33030

Coalition of Florida Farmworkers Organization (COFFO)
21 South Krome Avenue
Homestead, FL 33030

Jewish Family Service of Greater Miami
701 South Homestead Boulevard
Suite B-6
Homestead, FL 33030

Metro Dade Office of Rehab Services Jack Orr Ranch
31601 SW 197 Avenue
Homestead, FL 33030

HUDSON

Shell of Hope, Inc.
13825 U.S. Highway 19
Suite 307
Hudson, FL 34667

IMMOKALEE

Bridges of America Hendry Correctional Institution
12551 Wayne Wright Drive
Immokalee, FL 34142-9747

David Lawrence Center The Pines
425 North First Street
Immokalee, FL 33934

INDIALANTIC

Center for Nonaddictive Living
114 6th Avenue, Suite 2
Indialantic, FL 32903

INDIANTOWN

Martin Unit Treatment Center
1175 SW Allapattah Road
Indiantown, FL 34956

INTERCESSION CITY

Center for Drug Free Living Adolescent Residential Campus
5970 South Orange Blossom Trail
Intercession City, FL 33848

JACKSONVILLE

Addictions Rehabilitations Clinic
Naval Air Station Jax
Building 590, Keily Street
Jacksonville, FL 32212-0046

Counseling and Assistance Center
Naval Station
Jacksonville, FL 32228-0071

Davenport Center
8889 Corporate Square Court
Jacksonville, FL 32216

Gateway Community Services, Inc.
Adolescent Unit/Outpatient
Adult Intensive Residential Program
555 Stockton Street
Jacksonville, FL 32204

TPC Village
2671 Huffman Boulevard
Jacksonville, FL 32216

Outpatient/Edgewood
1105 West Edgewood Avenue
Jacksonville, FL 32208

Outpatient/University
1754 University Boulevard West
Jacksonville, FL 32217

Greenfield Center
1820 Barrs Street
Suite 640
Jacksonville, FL 32204

Help Center
743 West Ashley Street
Jacksonville, FL 32202

Jacksonville Metro Treatment Center
3609 Emerson Street
Jacksonville, FL 32208

Kerekes and Associates, Inc.
101 Century 21 Drive
Suite 119-F
Jacksonville, FL 32216

River Region Human Services, Inc.
330 West State Street
Jacksonville, FL 32202

Substance Abuse Program
451 Catherine Street
Jacksonville, FL 32202

Salvation Army
900 West Adams Street
Jacksonville, FL 32202

Substance Abuse Treatment Program
451 Catherine Street-CCD
Jacksonville, FL 32202

KEY WEST

Drug Court Treatment Division Project Outpatient
323 Fleming Street
Key West, FL 33041

Lower Florida Keys Health System, Inc.
1200 Kennedy Drive
Key West, FL 33040

Safe Port/Housing Authority/ Key West
301 White Street, Building 12
Key West, FL 33040

KISSIMMEE

Addictions Compulsions Treatment Center (ACT, Inc.) Kissimmee Outpatient
800 Office Plaza Boulevard
Suite 401
Kissimmee, FL 34744

Bridges of America Kissimmee CCC
2925 North Michigan Avenue
Kissimmee, FL 34744

Colonial Counseling Associates Outpatient/Kissimmee
3501 West Vine Street, Suite 290
Kissimmee, FL 34741

Osceola Counseling Center
Center for Drug Free Living
201 East Ruby Avenue
Building 9, Suite B
Kissimmee, FL 34741

Osceola Mental Health, Inc. Adult Outpatient Substance Abuse Services
230 East Monument Avenue
Kissimmee, FL 34741

LA BELLE

Hendry/Glades Mental Health Clinic, Inc. Mental Health Alcohol and Drug Abuse Treatment Program
80 Euclid Place
La Belle, FL 33935

LAKE BUTLER

Meridian Behavioral Healthcare, Inc. Union Office
395 West Main Street
Lake Butler, FL 32054

LAKE CITY

Bridges of America Lake City Community Correctional Center
1620 Lake Jeffery Street
Lake City, FL 32056

Meridian Behavioral Health Care, Inc.
3900 South First Street
Lake City, FL 32025

Turning Point Hospital
650 East Baya Avenue
Lake City, FL 32025

Veterans' Affairs Medical Center Substance Abuse Services
801 South Marion Street
Suite 116-A
Lake City, FL 32055

LAKELAND

Central Florida Human Services Centers
1325 George Jenkins Boulevard
Lakeland, FL 33802

Heart of Florida Behavioral Center
2510 North Florida Avenue
Lakeland, FL 33805

Lakeland Center
3506 Lakeland Hill Boulevard
Lakeland, FL 33805

Michiel W. Crawford LCSW
215 East Bay Street, Suite 2
Lakeland, FL 33801

Tri-County Human Services
1831 North Crystal Lake Drive
Lakeland, FL 33801

LAKE WORTH

Center for Alcohol and Drug Studies
3153 Canada Court
Lake Worth, FL 33461

Growing Together, Inc.
1000 Lake Avenue
Lake Worth, FL 33460

Quest Center
5700 Lake Worth Road, Suite 112
Lake Worth, FL 33406

LAND O LAKES

Alpha Counseling Services
6741 Land O Lakes Boulevard
Land O Lakes, FL 34639

LARGO

Boley Centers for Behavioral Healthcare, Inc.
12809 Wild Acres Road
Largo, FL 34643

Center for Behavioral Medicine
2025 Indian Rocks Road
Largo, FL 33774

Operation PAR, Inc.
13800 66th Street North
Largo, FL 34641

LECANTO

Marion/Citrus Mental Health
Center, Inc. Citrus Alcoholism Program
3238 South Lecanto Highway
Lecanto, FL 34461

Tri-County Rehab Center
1645 West Gulf to Lakes Highway
Lecanto, FL 32661

LIVE OAK

Meridian Behavioral Healthcare, Inc.
Nobles Ferry Road, Box 418
Live Oak, FL 32060

LONGWOOD

Families in Recovery
282 Short Avenue, Suite 116
Longwood, FL 32750

Human Service and Resources and Associates, Inc.
880 State Road 434 East
Suite 100
Longwood, FL 32750

MACCLENNY

Gateway Community Services, Inc.
U.S. Highway 90 West
Agricultural Building
MacClenny, FL 32063

MAITLAND

Orlando Health Care Group
2301 Lucien Way
Suite 145
Maitland, FL 32751

MARATHON

Comprehensive Psychiatric Center/Keys
11399 Overseas Highway
Marathon, FL 33050

Guidance Clinic of Middle Keys
3000 41st Ocean Street
Marathon, FL 33050

MARIANNA

Chemical Addictions Recovery Effort Jackson County Outpatient Office
4150 Hollis Drive
Marianna, FL 32446

Community Services of North Florida, Inc.
4878 Blue Springs Road
Marianna, FL 32446

MAYO

Mayo Correctional Institution
Highway 27
Mayo, FL 32066

MELBOURNE

Center for Drug Free Living Inc
1204 South Hickory Street
Melbourne, FL 32901

Circles of Care, Inc. Melbourne Detox/Residential
400 East Sheridan Road
Melbourne, FL 32901

Family Counseling Center of Brevard/Melbourne Outpatient and Prevention
507 North Harbor City Boulevard
Melbourne, FL 32935

Harbor City Counseling Center
668 West Eau Gallie Boulevard
Melbourne, FL 32935

MIAMI

Bayview Centers, Inc.
Division of Outpatient Services
12550 Biscayne Boulevard
Miami, FL 33150

Better Way of Miami, Inc.
800 NW 28 Street
Miami, FL 33127

Catholic Charities Bureau, Inc. Arch Diocese of Miami/DBA St. Luke's Center
7707 NW 2nd Avenue
Miami, FL 33150

Comprehensive Psychiatric Center/North
240 NW 183rd Street
Miami, FL 33169

Comprehensive Psychiatric Center/South
9735 East Fern Street
Miami, FL 33157

Concept House, Inc.
Maternal Addiction Program
162 NE 49th Street
Miami, FL 33137

Outpatient Services
4850 NE 2nd Street
Miami, FL 33137

Dade Family Counseling
8352 SW 8th Street
Miami, FL 33155

DUI Resolutions
7765 South West 87th Avenue
Suite 104
Miami, FL 33173

Extended Care, Inc. DBA Transitions Recovery Program
1928 NE 154th Street, Suite 100
Miami, FL 33162

Family Counseling Services
South Dade
10700 Caribbean Boulevard
Suite 412
Miami FL 33183

West Dade
8900 SW 107th Avenue
Suite 200
Miami, FL 33176

Family Resource Center Family Enhancement Program
4770 Biscayne Boulevard
Suite 610
Miami, FL 33137

Health Crisis Network
5050 Biscayne Boulevard
Miami, FL 33137

Health and Recovery Center at Jackson Memorial Hospital
1611 NW 12th Avenue, Annex 4
Miami, FL 33136

Here's Help, Inc.
9016 SW 152nd Street
Miami, FL 33156

Jewish Family Service of Greater Miami
1790 SW 27th Avenue
Miami, FL 33145

18999 Biscayne Boulevard
Suite 200
Miami, FL 33180

9700 South Dixie Highway
Suite 650
Miami, FL 33156

Kedem Counseling Center, Inc.
Outpatient Substance Abuse Treatment
2420 SW 27th Avenue
Miami, FL 33155

Miami Dade Office of Rehabilitation Services
3140 NW 76th Street
Miami, FL 33137

Central Receiving and Treatment Diversion and Treatment Program/Model Cities
8500 NW 27 Avenue
Miami, FL 33147

New Opportunity House
777 NW 30 Street
Miami, FL 33127

Rehab and Aftercare Center/North
3190 NW 116 Street
Miami, FL 33167

T/G/K Correctional Facility A/C Program
7000 NW 41 Street
Miami, FL 33166

Miami Counseling Services
13831 SW 59th Street
Suite 101
Miami, FL 33183

New Hope Corps
17130 SW 137th Avenue
Miami, FL 33177

Open Door Counseling Center, Inc.
515 SW 12th Avenue, Suite 521
Miami, FL 33130

Regis House Prevention Services
2010 NW 7 Street
Miami, FL 33125

South Florida Jail Ministries Agape Women's Center
22790 SW 112th Avenue
Miami, FL 33170

Spectrum Programs, Inc.
Administration and Outpatient Service
11031 NE 6th Avenue
Miami, FL 33161

Dade Residential
140 NW 59 Street
Miami, FL 33127

Outpatient South
8353 SW 124th Street
Suite 107
Miami, FL 33127

Substance Abuse Control Center, Inc. Outpatient Program
6850 SW 24th Street, Suite 503
Miami, FL 33155

Village South, Inc. Addiction Treatment Center
3180 Biscayne Boulevard
Miami, FL 33137

Total Rehab Services
4011 West Flagler Street
Miami, FL 33134

Veterans' Affairs Medical Center Substance Abuse Rehab Program
1201 NW 16 Street
Miami, FL 33125

Outpatient Program
5220 Biscayne Boulevard
Miami, FL 33137

MIAMI BEACH

Associates for Psychological Services Miami Beach Substance Abuse Services
2301 Collins Avenue, Suite M-113
Miami Beach, FL 33139

Jewish Family Service of Greater Miami
300 41st Street, Suite 216
Miami Beach, FL 33141

MIDDLEBURG

Clay County Behavioral Health Center
3292 County Road, Suite 220
Middleburg, FL 32068

MONTICELLO

Apalachee Center for Human Services Monticello
U.S. 19 South
Monticello, FL 32344

NAPLES

A Kind Ear
2900 14th Street North
Unit 7
Naples, FL 34103

Alternatives Chemical
Dependency Consultant Services, Inc.
3065 Terrace Avenue
Naples, FL 34103

David Lawrence Center
6075 Golden Gate Parkway
Naples, FL 34103

3400 North Tamiami Trail
Suite 204
Naples, FL 34103

Naples Research and Counseling Center Willough at Naples
9001 Tamiami Trail East
Naples, FL 34103

NARANJA

Metatherapy Institute, Inc.
27200 Old Dixie Highway
Naranja, FL 33032

NAVARRE

Twelve Oaks Alcohol and Drug Recovery Center
Intensive Day Treatment
Outpatient Program
2068 Healthcare Avenue
Navarre, FL 32566

NEW PORT RICHEY

Anglican Family Service Inc
3110 Florida Avenue
New Port Richey, FL 34653

Shell of Hope Inc
5254 State Road 54
New Port Richey, FL 34652

The Harbor Behavioral Healthcare Institute
5390 School Road
New Port Richey, FL 34653

Adolescent Residential Center Academy
6205 Trouble Creek Road
New Port Richey, FL 34652

Detox Program
8002 King Helie Boulevard
New Port Richey, FL 34653

NEW SMYRNA BEACH

Turning Point Hospital
237 North Causeway
New Smyrna Beach, FL 32169

NOKOMIS

Doctor Lynn Bernstein and Associate
2510 Tamiami Trail North
Nokomis, FL 34275

NORTH MIAMI BEACH

Holistic Counseling Services
16103 NE 11th Court
North Miami Beach, FL 33162

OCALA

CATS, Inc.
730 SE Osceola Avenue
Ocala, FL 34471

Marion/Citrus Mental Health Center, Inc.
Adult Residential Services
Children and Family Services
Women's Day Treatment
717 SW Martin Luther King Jr. Avenue
Ocala, FL 34474

MICA
Detox Unit
5664 SW 60th Avenue
Ocala, FL 34474

Quad County Treatment Center
913 East Silver Springs Boulevard
Ocala, FL 32670

OCHOPEE

Miccosukee Human Services Program
U.S. Route 41, Tamiami Trail Mile Marker 70
Ochopee, FL 34141

OKEECHOBEE

Okeechobee Outpatient Office
1600 SE 2nd Avenue
Okeechobee, FL 34972

OPA LOCKA

Dade Family Counseling, Inc.
2734 NW 183rd Street, Suite 206
Opa Locka, FL 33056

Here's Help, Inc. Residential
15100 NW 27 Avenue
Opa Locka, FL 33054

Lock Towns CMHC, Inc.
Opa Locka Substance Abuse Outpatient
15055 NW 27th Avenue
Opa Locka, FL 33054

Daybreak North
16555 NW 25nd Avenue
Opa Locka, FL 33054

ORLANDO

Access Behavioral Care Associates
7232 Sand Lake Road, Suite 302
Orlando, FL 32819-5255

Addictions Compulsions Treatment Center, Inc.
4300 South Semoran Boulevard
Suite 207
Orlando, FL 32822

5761 South Orange Blossom Trail
Suite 2
Orlando, FL 32810

4823 Silver Star Road, Suite 140
Orlando, FL 32808

Arise Counseling Associates
120 Gatlin Avenue
Orlando, FL 32806-6908

Barbara B Fuller, LCSW PA
1910 East Hillcrest Street
Orlando, FL 32803

Bridges of America/Orlando Bridge
2100 Brengle Avenue
Orlando, FL 32808

Center for Drug Free Living, Inc.
Aftercare
New Horizons
Orlando Counseling Center
100 West Columbia Street
Orlando, FL 32806

Harbor Halfway House
1405 West Michigan Street
Orlando, FL 32805

Women's Residential Program
1780 North Mercy Drive
Orlando, FL 32808

Central Florida Substance Abuse Treatment Centers, Inc. Outpatient Methadone Maintenance
1800 West Colonial Drive
Orlando, FL 32804

Colonial Counseling Associates
9318 East Colonial Drive
Suite A-15
Orlando, FL 32817

West Office
5600 West Colonial Drive
Suite 305
Orlando, FL 32819

Outpatient/Central Office
710 East Colonial drive
Orlando, FL 32803

Department of Veterans Affairs Satellite Outpatient Clinic
5201 Raymond Street
Orlando, FL 32806

Florida Hospital
Outpatient Addictions Treatment Services
615 East Princeton Street
Orlando, FL 32803

Florida Psychiatric Associates Orlando Outpatient
7300 Sandlake Commons Boulevard
Suite 112
Orlando, FL 32819

Human Services Associates, Inc. Juvenile ARF
823 West Central Boulevard
Orlando, FL 32

Lakeside Alternatives, Inc.
434 West Kennedy Boulevard
Orlando, FL 32810

Lisa Merlin House, Inc.
3101 North Pine Hills Road
Orlando, FL 32808

Medical Services Methadone
712 West Gore Street
Orlando, FL 32806

Prucare Orlando Health Care Group
21 West Columbia Street
Orlando, FL 32806

Short Term Adult Residential Women's Residential II
5609 Claracona/Ocoee Road
Orlando, FL 32801

Specialized Treatment Education and Prevention Services, Inc.
2917 North Pine Hills Road
Orlando, FL 32808

OVIEDO

Human Service and Resources and Associates, Inc.
120 North Central Avenue
Oviedo, FL 32765

PALATKA

Putnam Behavioral Healthcare Residential Program
320 Kay Lakin Drive
Palatka, FL 32177

PALM HARBOR

Elliot and Worley Counseling
1022 Nebraska Avenue
Palm Harbor, FL 34683

PANAMA CITY

Chemical Addictions Recovery Effort
A Woman's Addiction Recovery Effort (AWARE)
4000 East 3rd Street
Panama City, FL 32404

Bay County Outpatient Office
Starting Over Straight
School Prevention
4000 East 3 Street
Suite 200
Panama City, FL 32404

PEMBROKE PINES

Bridges of America Broward Correctional Institution
20421 Sheridan Street
Pembroke Pines, FL 33084

PENSACOLA

Community Drug and Alcohol Commission
Women's Intervention Services and Education
222-A West Cervantes Street
Pensacola, FL 32501

Cordova Counseling Center
4400 Bayou Boulevard, Suite 8-D
Pensacola, FL 32503

Lakeview Center, Inc.
Adolescent Overlay
Adult Residential
Outpatient Counseling
Pathway
1221 West Lakeview Avenue
Building D
Pensacola, FL 32501

Naval Air Station Addictions Treatment Facility
499 South Avenue
Pensacola, FL 32508

Pavillion Chemical Dependency
8383 North Davis Highway
Pensacola, FL 32514

PERRY

Apalachee Center for Human Services
301 Industrial Park Drive
Perry, FL 32347

PINELLAS PARK

Bay Area Treatment Center (BATC)
6328 Park Boulevard North
Suite 4
Pinellas Park, FL 33781

Center for Rational Living Inc
Avenue North Suite 5
Pinellas Park, FL 33781

Personal Enrichment Through Mental Health Services
Crisis Stabilization Unit
11254 58 Street North
Pinellas Park, FL 33782

PLANT CITY

Drug Abuse Comp. Coord. Office (DACCO)
1308 Larrick Lane
Plant City, FL 33566

POLK CITY

Central Florida Human Services Center Polk Correctional Facility
3876 Evans Road
Polk City, FL 33868

POMPANO BEACH

Alcohol and Drug Abuse Services Division Residential Services
3275 NW 99th Way
Pompano Beach, FL 33065

Bridges of America/Turning Point
400 SW 2nd Street
Pompano Beach, FL 33060

Broward County Sheriffs Office DUI Program Unit
3900 North Powerline Road
Pompano Beach, FL 33073

Center for Positive Growth
1500 University Drive, Suite 201
Pompano Beach, FL 33071

Pompano Treatment Center, Inc. Methadone Maintenance
380 SW 12 Avenue
Pompano Beach, FL 33069

Spectrum Programs, Inc.
Outpatient Broward North
450 East Atlantic Boulevard
Pompano Beach, FL 33060

PORT CHARLOTTE

Life Transitions, Inc.
2450 Tamiami Trail
Port Charlotte, FL 33952

PORT RICHEY

Alpha Counseling Service
10730 U.S. Highway 19, Suite 4
Port Richey, FL 34668

PORT SAINT LUCIE

Recovery Associates, Inc.
8000 South U.S. 1, Suite 202
Port Saint Lucie, FL 34952

PUNTA GORDA

Charlotte Community Mental Health Services, Inc.
1700 Education Avenue
Punta Gorda, FL 33950

Coastal Recovery Centers/Kelly Hall Residential Treatment Center
2208 Castilla Avenue
Punta Gorda, FL 33950

Riverside Behavioral Center
733 East Olympic Street
Punta Gorda, FL 33950

QUINCY

Disc Village, Inc.
Gadsden Adult Outpatient
Gadsden Juvenile Outpatient
Quincy, FL 32351

RIVERVIEW

Tampa Bay Academy Youth and Family Centered Services, Inc.
12012 Boyette Road
Riverview, FL 33569

ROCKLEDGE

Family Counseling Center of Brevard, Inc.
220 Coral Sands Drive
Rockledge, FL 32955

Wuesthoff Hospital Sunrise Substance Abuse Program
110 Longwood Avenue
Rockledge, FL 32956

SAFETY HARBOR

Behavioral Sciences Center Structured Outpatient Chemical Dependency Treatment Program
727 2nd Street South
Safety Harbor, FL 34695

SAINT AUGUSTINE

Epic Community Services I
88 Riberia Street, Suite 300
Saint Augustine, FL 32084

Psychological Services of Saint Augustine Inc
28 Clark Street
Saint Augustine, FL 32095

Mental Health Resource Center, Inc. Saint John's County Community Mental Health Services
179 Marine Street
Saint Augustine, FL 32084

SAINT PETERSBURG

Behavioral Sciences Center
5100 First Avenue North
Saint Petersburg, FL 33710

Boley Behavioral Health Care
1147 16th Street North
Saint Petersburg, FL 33705

Goodwill Industries Suncoast
10596 Gandy Boulevard
Saint Petersburg, FL 33733-4456

Operation PAR, Inc.
Adolescent Residential Center
6720 54th Avenue North
Saint Petersburg, FL 33707

Children of Substance Abusers (COSA)
2000 4th Street South
Saint Petersburg, FL 33705

Juvenile Outpatient
6720 54th Avenue North
Saint Petersburg, FL 33709

SAINT PETERSBURG BEACH

Stepping Stone of Tampa, Inc.
Dolphin Village, Suite 213
Saint Petersburg Beach, FL 33706

SANFORD

Bridges of America/Sanford Bridge
500 South Holly Avenue
Sanford, FL 32771

Crossroads of Sanford
300 South Bay Avenue
Sanford, FL 32771

Grove Counseling Center, Inc.
Adolescent Outpatient Program
1550 South French Road
Sanford, FL 32771

Specialized Treatment Education and Prevention Services
1019 Oleander Avenue
Sanford, FL 32771

SARASOTA

Another Level of Recovery
310 South Osprey Street
Sarasota, FL 34236

Coastal Recovery Centers
3830 Bee Ridge Road
Sarasota, FL 34233

Doctors' Hospital of Sarasota, Ltd. Genesis Center
2750 Bahia Vista Street
Sarasota, FL 34239

First Step of Sarasota, Inc.
Residential Center
4613 North Washington Boulevard
Sarasota, FL 34234

Pregnant SA Women's Program
1726 18 Street
Sarasota, FL 34234

Outpatient Program
2800 Bahia Vista Street
Suite 300
Sarasota, FL 34239

SATELLITE BEACH

Brevard Outpatient Alternative Treatment (BOAT)
1127 South Patrick Drive
Suite 24
Satellite Beach, FL 32937

SEBRING

Tri-County Human Services
155 U.S. Highway 27 North
Sebring, FL 33870

SHARPES

Brevard Correctional Institution
Juvenile TASC Program
870 Camp Road
Sharpes, FL 32959

STARKE

Bridges of America Florida State Prison Work Camp
Highway 26 West
Starke, FL 32091

Meridian Behavioral Health Care Bradford Guidance Clinic
945 Grand Street
Starke, FL 32091

STUART

New Horizons of the Treasure Coast, Inc.
2440 SE U.S. Highway 1
Stuart, FL 34994

TALLAHASSEE

A Life Recovery Center
449 West Georgia Street
Tallahassee, FL 32304

Addiction Recovery Center
2626 Care Drive, Suite 202
Tallahassee, FL 32308

Disc Village, Inc.
Adult Outpatient
603 Martin Luther King Boulevard
Tallahassee, FL 32301

Juvenile Outpatient
3333 West Pensacola Street
Suite 140
Tallahassee, FL 32304

Salvita, Inc.
419 East Georgia Street
Tallahassee, FL 32301

Turn About, Inc.
2771 Miccosukee Road
Tallahassee, FL 32308

TAMPA

Agency for Community Treatment Services, Inc. (ACTS)
Outpatient Treatment Services
1815 West Sligh Avenue
Tampa, FL 33604

Transitional Housing
4403 West Martin Luther King Jr. Boulevard
Tampa, FL 33614

W. T. Edwards Group Home
3810 West Martin Luther King Jr. Boulevard
Tampa, FL 33614

DACCO Inc.
74402 North 56th Street
Building 500 and 600
Tampa, FL 33617

Chemotreatment Center
Methadone Maintenance
Detox
74402 North 56th Street
Building 600
Tampa, FL 33617

50th Street Outpatient
3630 North 50th Street
Tampa, FL 33619

Inner City Residential Program
4422 East Columbus Drive
Tampa, FL 33605

Male and Female Residential
4422 East Columbus Drive
Tampa, FL 33605

Residential Treatment Facility
3636 North 50 Street
Tampa, FL 33619

Daytop Village, Inc.
1718 West Cass Street
Tampa, FL 33606

Healthcare Connection of Tampa, Inc.
107 West 131st Avenue
Tampa, FL 33614

Hillsborough Community Correctional Center Day/Night Intensive Treatment
4102 West Hillsborough Avenue
Tampa, FL 33614

James A. Haley Veterans' Hospital Alcohol and Drug Abuse Treatment Program
13000 Bruce Downs Boulevard
Tampa, FL 33612

Larry Garvin Outpatient Program
13701 Bruce B Downs Boulevard, Suite 110
Tampa, FL 33613

Project Recovery Center for Women
305 South Hyde Park Avenue
Tampa, FL 33606

Tampa Crossroads
202 West Columbus Drive
Tampa, FL 33602

Tampa Metro Treatment Center
5202-C East Busch Boulevard
Tampa, FL 33617

Town and Country Hospital Addictions Recovery Unit
6001 Webb Road
Tampa, FL 33615

Turning Point of Tampa
5439 Beaumont Center Boulevard
Suite 1010
Tampa, FL 33634

TARPON SPRINGS

Agency for Community
Treatment Services, Inc. (ACTS) Pinellas Domiciliary
3575 Old Keystone Road
Tarpon Springs, FL 34689

TAVARES

Counseling Associates and Treatment Services
102 East Alfred Street
Tavares, FL 32778

TAVERNIER

Guidance Clinic of the Upper Keys Outpatient
92140 Overseas Highway
Suite 5
Tavernier, FL 33070

TYNDALL AFB

Tyndall Air Force Base Substance Abuse Program
325 MDOS/SGOMH 340 Magnolia Circle
Tyndall AFB, FL 32403-5612

VENICE

Coastal Recovery Centers South County Clinic III
119 Corporation Way
Venice, FL 34292

First Step of Sarasota, Inc. Venice Office
2210 South Tamiami Trail
Suite 9
Venice, FL 34293

VERO BEACH

Alcohope
5925 37th Street
Vero Beach, FL 32968-4920

Center for Counseling and Addiction Recovery
1434 21st Street
Vero Beach, FL 32961

Indian River County Outpatient Branch
2300 3rd Court, Suite C
Vero Beach, FL 32960

New Life
5925 37th Street
Vero Beach, FL 32968-4920

WEST PALM BEACH

Bridges of America West Palm Beach Community Corrections
261 Fairgrounds Road
West Palm Beach, FL 33411

Center for Family Services
471 Spencer Drive
West Palm Beach, FL 33409

Comprehensive AIDS Program
2580 Metrocentre Boulevard
Suite 2
West Palm Beach, FL 33407

Drug Abuse Foundation of Palm Beach County/Sheriffs Drug Farm
673 Fairground Road
West Palm Beach, FL 33411

Drug Abuse Treatment Association, Inc. (DATA)
Outpatient
1720 East Tiffany Drive
Suite 102
West Palm Beach, FL 33407

Forest Hill Counseling Center, Inc.
3101 Forest Hill Boulevard
West Palm Beach, FL 33406

Glenbeigh Hospital of Palm Beach, Inc.
4700 Congress Avenue
West Palm Beach, FL 33407

Gratitude House
317 North Lakeside Court
West Palm Beach, FL 33407

Hanley Hazelden Center at Saint Mary's
5200 East Avenue
West Palm Beach, FL 33407

Lee Ballard, RN. CD. CAP
1408 North Killian Drive
Suite 208
West Palm Beach, FL 33403

Nina de Gerome MSW/F. Edward
McCabe Substance Abuse Services
333 Southern Boulevard
Suite 204
West Palm Beach, FL 33405

Palm Beach Treatment Center
1771 South Congress Avenue
Congress Plaza Unit 7
West Palm Beach, FL 33406

Parent and Child Team Inc
1195 North Military Trail
West Palm Beach, FL 33409

Professional Educational Consultants, Inc.
4623 Forest Hill Boulevard
Suite 110
West Palm Beach, FL 33415

Saint Marys Hospital Institute for Mental Health
901 45th Street
West Palm Beach, FL 33407

WINTER HAVEN

Tri-County Human Services
Adolescent Outpatient
Adult Outpatient
37 3rd Street SW
Winter Haven, FL 33880

WINTER PARK

Another Chance Counseling Center, Inc.
709 Executive Drive
Winter Park, FL 32789

Florida Psychiatric Associates, Inc.
1276 Minnesota Avenue
Winter Park, FL 32789

Lakeside Alternatives, Inc.
807 Morse Boulevard
Winter Park, FL 3278

Maureen R. Traynor Enterprises, Inc.
1347 Palmetto Avenue, 1st Floor
Winter Park, FL 32789

New Leaf Center, Inc.
1850 Lee Road, Suite 236
Winter Park, FL 32789-2106

Psychiatric Care Center
1600 Dodd Road
Winter Park, FL 32792

WINTER SPRINGS

Grove Counseling Center, Inc.
Adolescent
Adult Outpatient
580 Old Sanford Oviedo Road
Winter Springs, FL 32708

WOODVILLE

Disc Village, Inc.
Adolescent Treatment Program
Natural Bridge Treatment Center
2967 Natural Bridge Road
Woodville, FL 32362

ZEPHYRHILLS

Alpha Counseling Services
5040 Mission Square
Zephyrhills, FL 33541

Bridges of America Zephyrhills Corrections Institute
2739 Gall Boulevard
Zephyrhills, FL 33541

GEORGIA

ADEL

Behavioral Health Services of South Georgia
105 North Parrish Avenue
Adel, GA 31620

ALBANY

Albany Area Community Service Board/Crisis Stabilization Program and Detox
601 West 11th Avenue
Albany, GA 31701

Phoebe Putney Memorial Hospital Recovery Centers
417 3rd Avenue
Albany, GA 31703

Substance Abuse Counseling Center Family Service Center
Marine Corps Logistics Base
Code 170
Albany, GA 31704-5000

AMERICUS

Addiction Recovery Program (ARC)
696 McMath Mill Road
Americus, GA 31709

Middle Flint Behavioral Health Care Substance Abuse Detoxification Unit
425 North Lee Street
Americus, GA 31709

Sumter County Substance Abuse Outpatient Program
425 North Lee Street
Americus, GA 31709

Sumter Regional Hospital
100 Wheatley Drive
Americus, GA 31709

ASHBURN

Behavioral Health Services of South Georgia
259 East Washington Avenue
Ashburn, GA 31714

ATHENS

Athens Regional Medical Center Commencement Center
1199 Prince Avenue
Athens, GA 30613

Northeast Georgia Center Community Alcohol and Drug Abuse Prevention and Treatment
250 North Avenue
Athens, GA 30601

ATLANTA

Atlanta West Treatment Center
3201 Atlanta Industrial Parkway NW
Building 100, Suite 101
Atlanta, GA 30331

Charter Anchor Behavioral Health System
5454 Yorktowne Drive
Atlanta, GA 30349

Charter Behavioral Health Systems of Atlanta
811 Juniper Street NE
Atlanta, GA 30308

2151 Peachford Road
Atlanta, GA 30338

Choices
505 Fairburn Street SW
Atlanta, GA 30312

Columbia West Paces Medical Center Behavioral Health Unit
3200 Howell Mill Road
Unit 3 East
Atlanta, GA 30327

Dekalb Community Services Board Kirkwood Substance Abuse Clinic
30 Warren Street SE, Suite 5
Dekalb/Atlanta Human Services Center
Atlanta, GA 30317

Emory University Hospital Dept of Psychiatry and Behavioral Science
1639 Pierce Drive
Atlanta, GA 30322

GPA Treatment, Inc.
4255 Chamblee-Tucker Road
Atlanta, GA 30340

Grady Health System Drug Dependence Unit
60 Coca Cola Place SE
Atlanta, GA 30335

Grady Memorial Hospital Drug Dependence Unit
60 Coca Cola Place SE
Atlanta, GA 30303

Kirkwood Substance Abuse Clinic
66 Howard Street
Atlanta, GA 30317

Marr
2801 Clearview Place
Atlanta, GA 30340

New Start Drug Treatment Center
30 Warren Street SE
Atlanta, GA 30317

Northside Hospital Substance Abuse Center
1000 Johnson Ferry Road
Atlanta, GA 30342

Northside Mental Health Center
5825 Glenridge Drive, Building 4
Atlanta, GA 30342

Outreach Inc.
3030 Campbellton Road SW
Atlanta, GA 30311

649 Ashby Street NW
Atlanta, GA 30318-6644

Piedmont House Project Assist
761 Piedmont Avenue
Atlanta, GA 30309

Plasmetics, Inc. Apollo Addiction Recovery Center
275 Carpenter Drive, Suite 101
Atlanta, GA 30328

Private Clinic
1447 Peachtree Street NE
Suite 900
Atlanta, GA 30309

Renaissance Family Center for Women
3201 Atlanta Industrial Parkway
Building 100, Suite 101
Atlanta, GA 30305

Saint Judes Recovery Center, Inc.
139 Renaissance Parkway
Atlanta, GA 30308

Southside Healthcare Substance Abuse Unit
1660 Lakewood Avenue SW
Atlanta, GA 30315

Talbott Recovery Campus
5448 Yorktowne Drive
Atlanta, GA 30349

UJIMA Continuing Care Program
3201 Atlanta Industrial Parkway
Building 100, Suite 101
Atlanta, GA 30331

AUGUSTA

Augusta Metro Treatment Center
3171 Washington Road
Augusta, GA 30907

Charter Augusta Behavioral Health System
3100 Perimeter Parkway
Augusta, GA 30909-6423

CMHC of East Central Georgia Alcohol and Drug Services
3421 Mike Padgett Highway
Augusta, GA 30906

Medical College of Georgia Hospital and Clinics
1120 15th Street
Augusta, GA 30912

University Hospital Behavioral Health Center
1350 Walton Way
Augusta, GA 30902

Veterans' Affairs Medical Center Substance Abuse Treatment Program
Uptown Division
One Freedom Way
Augusta, GA 30904

BAINBRIDGE

Decatur County Mental Health Center
200 West Broughton Street
Bainbridge, GA 31717

BARNESVILLE

McIntosh Trail MH/MR/SA Community Services Board
700 Highway 341 South
Barnesville, GA 30204

BLAIRSVILLE

Georgia Mountains Community
55 Hughes Street, Suite B
Blairsville, GA 30512-3551

BLOOMINGDALE

Tidelands CSB Adolescent Program
Route 1, Box 280
Bloomingdale, GA 31302

BRUNSWICK

Gateway Center for Human Development Crisis Stabilization Unit
3045 Scarlet Street
Brunswick, GA 31302

Gateway Community Service Board Residential Substance Abuse Program
1609 Newcastle Street
Winchester Center
Brunswick, GA 31520

CARROLLTON

Carroll County Mental Health
Pathways/Carroll IDR Male Substance Abuse Center
527 Tanner Street
Carrollton, GA 30117

Pathway Center/Sunshine House
107 Park Place Way
Carrollton, GA 30117

CHICKAMAUGA

Lookout Mountain Community Services Youth Substance Abuse Program
4909 West Highway 136
Chickamauga, GA 30707

CLARKSTON

Marr, Inc. Women's Recovery Center
3700-D Market Street
Clarkston, GA 30021

CLEVELAND

White County Mental Health Substance Abuse Program
1241 Helen Highway, Suite 240
Cleveland, GA 30528

CEDARTOWN

Cosa Valley Center for MH/MR/SA Services Residential Treatment Unit
180 Water Oak Drive
Cedartown, GA 30125

COCHRAN

Middle Georgia Adolescent Residential Center
408 Peacock Street
Cochran, GA 31014

COLQUITT

Miller County Mental Health Center
250 West Pine Street
Colquitt, GA 31737

COLUMBUS

New Horizons MH/MR/SA Community Service Board Women's Program
1727 Boxwood Place
Columbus, GA 31906

New Horizons Outpatient Alcohol and Drug Services
2100 Comer Avenue
Columbus, GA 31901

Saint Francis Hospital Inc.
The Bradley Center of Saint Francis
Columbus, GA 31904

CONYERS

GRN Community Service Board
977A Taylor Street
Conyers, GA 30012

CORDELE

Crisp County Outpatient Services
112 23rd Avenue East
Cordele, GA 31015

COVINGTON

Newton Mental Health Clinic
6119 Adams Street NE
Covington, GA 30014

CUMMING

Forsyth County Mental Health
125 North Corners Parkway
Cumming, GA 30040

DAHLONEGA

Georgia Mountains Community
266-B Mechanicsville Road
Dahlonega, GA 30533

DALTON

Georgia Highlands Treatment Services
900 Shugart Road
Dalton, GA 30720

DAWSON

Terrell County Mental Health Center
638 Forrester Drive
Dawson, GA 31742

DECATUR

Alliance Recovery Center
209-B Swanton Way
Decatur, GA 30030-3271

Comprehensive Addiction Rehabilitation Programs of Georgia Inc (CARP)
2145 Candler Road
Decatur, GA 30032

Dekalb Community Service Board
3110 Clifton Springs Road
Suite A
Decatur, GA 30034

Dekalb Medical Center Behavioral Health Services
2701 North Decatur Road
Decatur, GA 30033

Fox Recovery Center Alcohol and Drug Abuse Program
3100 Clifton Springs Road
Decatur, GA 30034

Our Common Welfare Inc
4289 Memorial Drive, Suite I
Decatur, GA 30032

Veterans' Affairs Medical Center Substance Abuse Treatment Program
1670 Clairmont Road
Decatur, GA 30033

DEMOREST

Habersham Mental Health Center
196 Scroggins Drive
Demorest, GA 30535-5354

DORAVILLE

Turn Around Recovery Residences
5455 Buford Highway
Suite 105-A
Doraville, GA 30340

DOUGLAS

Satilla Community Mental Health Substance Abuse Clinic
1005 Shirley Avenue
Douglas, GA 31533

DUBLIN

Mental Health/Alcohol and Drug Outpatient Services
2121-A Bellevue Avenue
Dublin, GA 31021

Twin Oaks Recovery Center
2121A Belevue Street
Dublin, GA 31021

Veterans' Affairs Medical Center
Substance Abuse Treatment Program
1826 Veterans Boulevard
Dublin, GA 31021

EASTMAN

Community Mental Health Center Eastman Annex
107 Plaza Drive
Eastman, GA 31023-2223

EISENHOWER ARMY MEDICAL CENTER

Fort Gordon Community Counseling Center
CCC Eisenhower Medical Center 12 W Bldg 300
Eisenhower Army Med Center, GA 30905

ELBERTON

Elbert County Mental Health Center
230 Tate Street
Elberton, GA 30635

FITZGERALD

Behavioral Health Services of South Georgia
124 South Grant Street
Fitzgerald, GA

FORT BENNING

U.S. Army MEDAC MCXB/AD
Building 324
Fort Benning, GA 31905-6100

FORT MCPHERSON

U.S. Army Health Clinic
Building 171
Fort McPherson, GA 30330-5000

FORT OGLETHORPE

Metro Treatment of Georgia LP Northwest Georgia Treatment Center
65 White Street
Fort Oglethorpe, GA 30742

FORT STEWART

U.S. Army MEDAC MSUB/ ADAPCP
Fort Stewart, GA 31314-5000

FORT VALLEY

Phoenix Center Behavioral Health Services
503 Camellia Boulevard
Fort Valley, GA 31030

GAINESVILLE

Georgia Mountain Community Services Lakewinds Recovery Program
472 South Enota Street
Gainesville, GA 30501

Northeast Georgia Medical Center
743 Spring Street NE
Gainesville, GA 30505

GREENSBORO

Greene County Mental Health Center
502 Martin Luther King Boulevard
Box 9
Greensboro, GA 30642

502 South Walnut Street
Suite 101
Greensboro, GA 30642

GREENVILLE

Pathways Center Meriweather County Mental Health Substance Abuse Center
756 Woodbury Highway
Greenville, GA 30222

GRIFFIN

McIntosh Trail Substance Abuse Services
Substance Abuse Outpatient Treatment
141 West Solomon Street
Griffin, GA 30223

Adolescent Substance Abuse Day Treatment
1435 North Expressway
Griffin, GA 30223

Midway Recovery Systems, Inc.
119 South 10th Street
Griffin, GA 30223

HAHIRA

Behavioral Health Services of South Georgia Lowndes Substance Acute Detox
204 East Lawson Street
Hahira, GA 31632

HAPEVILLE

Odyssey Family Counseling Center
3578 South Fulton Avenue
Hapeville, GA 30354

HARTWELL

Hart Mental Health Substance Abuse Clinic
520 West Franklin Street
Hartwell, GA 30643

HINESVILLE

Fraser Recovery Center
203 Mary Lou Drive
Hinesville, GA 31313

JACKSON

Butts County Counseling Center
463 Kennedy Drive, Suite B
Jackson, GA 30233

JEFFERSON

Jackson County Mental Health Center
67 Athens Street
Jefferson, GA 30549

Potter's House Christian Rehabilitation Center
655 Potters House Road, Route 2
Jefferson, GA 30549

JONESBORO

Clayton Mental Health Center Substance Abuse Program
853 Battle Creek Road
Jonesboro, GA 30236

KINGS BAY

Counseling and Assistance Center Clinical Services Department
881 USS James Madison Road
Kings Bay, GA 31547

LAFAYETTE

Lookout Mountain Community Services
501 Mize Street
LaFayette, GA 30728

LAGRANGE

Troup County MH/SA Clinic
122 Gordon Commercial Drive
LaGrange, GA 30240

West Georgia Health System
1514 Vernon Street
LaGrange, GA 30240-4130

LAKELAND

Behavior Health Services of South Georgia
Lanier County Health Department Clinic
422 West Bostick Street
Lakeland, GA 31635

Cook Outpatient Services
422 West Bostick Street
Lakeland, GA 31635

LAWRENCEVILLE

Gwinnett/Rockdale/Newton Alcohol and Drug Abuse Program
175 Gwinnett Drive
Lawrenceville, GA 30044

Summitridge
250 Scenic Highway
Lawrenceville, GA 30045

LEESBURG

The Anchorage, Inc.
162 Hampton Lane
Leesburg, GA 31763

LITHIA SPRINGS

Cobb/Douglas County Community Service Board
Parkway Medical Center, 9th Floor
Lithia Springs, GA 30122

Columbia Parkway Medical Center
1000 Thornton Road
Lithia Springs, GA 30122

LOUISVILLE

Jefferson County Mental Health Clinic
408 Green Street
Louisville, GA 30434

MACON

Charter Behavioral Health Systems
3500 Riverside Drive
Macon, GA 31209

Macon New Start Substance Abuse Program
175 Emery Highway
Macon, GA 31201

River Edge BHC Addictive Disease Outpatient Program
175 Emery Highway
Macon, GA 31201

River Edge Project Connect
543 2nd Street, Lower Level
Macon, GA 31201

River Edge Recovery Center
3575 Fulton Mill Road
Macon, GA 31206

MARIETTA

Cobb/Douglas County Community Service Board Adult Substance Abuse Outpatient
3411 Austell Road
Marietta, GA 30060

Kennestone Hospital Mental Health Unit
677 Church Street
Marietta, GA 30060

Mothers Making a Change
Marietta Parkway
Marietta, GA 30060

MCDONOUGH

Henry County Counseling Center
139 Henry Parkway
McDonough, GA 30253-6636

MIDLAND

Alchemy Therapeutic Community Columbus TC
9067 Veterans Parkway
Midland, GA 31820

MILLEDGEVILLE

Bridges Outpatient Center Inc
540 West Thomas Street, Suite E
Milledgeville, GA 31061

Oconee Alcohol and Drug Program
900 Barrows Ferry Road
Milledgeville, GA 31061

Oconee Mental Health Center Day Treatment Program
430 North Jefferson Street
Milledgeville, GA 31061

MONROE

Walton County Mental Health Center
226 Alcova Street, Suite D-11
Monroe, GA 30655

MOODY AFB

Moody Air Force Base Substance Abuse Program
347 MDOS/SGOMH
3278 Mitchell Boulevard
Moody AFB, GA 31699

MOULTRIE

Georgia Pines Community Service Board Colquitt County Mental Health Center
615 North Main Street
Moultrie, GA 31768

Turning Point Hospital
319 East Bypass
Moultrie, GA 31768]

NASHVILLE

Behavioral Health Services of South Georgia/Berrien Outpatient
201 Hazel Avenue
Nashville, GA 31639

NEWNAN

Pathways Center Coweta Substance Abuse Center
12 Savannah Street
Newnan, GA 30263-2503

OCILLA

Behavioral Health Services of South Georgia Irwin Outpatient Program
310 Vocational Tech Drive
Ocilla, GA 31774

RIVERDALE

Riverwoods Southern Regional Psychiatric Center
11 Upper Riverdale Road SW
Riverdale, GA 30274

ROBERTA

Crossroads Substance Abuse Day Treatment
278 Wright Avenue
Roberta, GA 31078

ROBINS AFB

Robins Air Force Base Substance Abuse Program
78 MDOS/SGOMH
655 7th Street
Robins AFB, GA 31098-2227

ROME

Northwest Georgia Regional Hospital
1305 Redmond Street
Rome, GA 30165

Star House, Inc. Halfway House
212 1/2 North 5th Avenue
Rome, GA 30161

Three Rivers Behavioral Health Services
43 Chateau Court SE
Rome, GA 30161-7238

Windwood
306 Shorter Avenue
Rome, GA 30165

ROSSVILLE

Private Clinic North
312 East Lake Avenue
Rossville, GA 30741

SAINT SIMONS ISLAND

Charter By The Sea Behavioral Health System
2927 Demere Road
Saint Simons Island, GA 31522

SANDERSVILLE

Oconee Center Adult Services
151 East Church Street
Sandersville, GA 31082

Washington County Satellite Clinic
153 East Church Street
Sandersville, GA 31082

SAVANNAH

Tidelands Community Service Board
516 Drayton Street
Savannah, GA 31401

SMYRNA

Ridgeview Institute Adult Addictions Medicine
3995 South Cobb Drive
Smyrna, GA 30080

Value Mark Browner Behavior Health Care System
3180 Atlanta Street SE
Smyrna, GA 30080

SNELLVILLE

GRN Recovery Center
3005-D Lenora Church Road
Snellville, GA 30078

SPRINGFIELD

Tidelands Community Mental Center
204 East Madison Street
Springfield, GA 31329-1086

STATESBORO

Pineland MA/MR/SA Services
508 Gentilly Road
Statesboro, GA 30458

Willingway Hospital Substance Abuse Services
311 Jones Mill Road
Statesboro, GA 30458

Women's Place
131 North College Street
Statesboro, GA 30458

SUMMERVILLE

Lookout Mountain Community Services
83 Highway 48
Summerville, GA 30747

SWAINSBORO

Ogeechee Substance Abuse Center
223 North Anderson Drive
Swainsboro, GA 30401

SYLVANIA

Ogeechee Area Mental Health Clinic
302 East Ogeechee Street
Sylvania, GA 30467

SYLVESTER

Worth County Mental Health Center Day Treatment Program
504 East Price Street
Sylvester, GA 31791

THOMASVILLE

Archbold Northside
401 Old Albany Road
Thomasville, GA 31799

Southwestern State Hospital
Gateway Dual Diagnosis
Community Residential Program
400 Pinetree Boulevard
Thomasville, GA 31792-1378

THOMSON

McDuffie County Mental Health Center
306 Greenway Street
Thomson, GA 30824

TIFTON

Behavioral Health Services of South Georgia
Tift Outpatient
334 Tifton-El Dorado Road
Tifton, GA 31794

Lakeside Addiction Recovery Center
340 Tifton-El Dorado Road
Tifton, GA 31794

TOCCOA

Stephens County MH/SA Center
1020 East Tugalo Street
Toccoa, GA 30577

TRENTON

Lookout Mountain Community Services
9622 Highway 11
Trenton, GA 30752-4621

VALDOSTA

Behavioral Health Services of South Georgia
Lowndes Project Light for Women
256 North Saint Augustine Road
Valdosta, GA 31602

Greenleaf Center, Inc. Substance Abuse Treatment Program
2209 Pineview Drive
Valdosta, GA 31602

Lowndes County Service Center
1664 East Park Avenue
Valdosta, GA 31601

Moody Air Force Base
3278 Mitchell Boulevard
347 Medical Group
Valdosta, GA 31699

WARNER ROBINS

Air Force Robins Mental Health Office
Warner Robins, GA 31098

Houston Medical Center Behavioral Science and Psychiatry
1601 Watson Boulevard
Warner Robins, GA 31093

Phoenix Center Behavioral Health Services
202 North Davis Drive
Warner Robins, GA 31093

WAYCROSS

New Visions Counseling Services
2100 Riverside Avenue
Waycross, GA 31501-7072

Saint Illa Center
3455 Harris Road
Waycross, GA 31503

WINDER

Project Adam Community Assistance Center, Inc.
112 Lanthier Street
Winder, GA 30680

HAWAII

AIEA

YMCA Outreach Services School-Based Program
Aiea High School
98-1276 Ulune Street
Aiea, HI 96701

EWA BEACH

Kahi Mohala Chemical Dependency Services
91-2301 Fort Weaver Road
Ewa Beach, HI 96706

YMCA Outreach Services School-Based Program
Campbell High School
91-980 North Road
Ewa Beach, HI 96706

HILO

Corporate Office Outpatient Treatment
1420 Kilauea Avenue
Hilo, HI 96720

Drug Addiction Services of Hawaii
305 Wailuku Drive, Suite 5
Hilo, HI 96720

HONOLULU

Attorneys and Judges Assistance Program of The Supreme Court of Hawaii
801 Alakea Street, Suite 202
Honolulu, HI 96813

Drug Addiction Services of Hawaii, Inc. (DASH)
Methadone Maintenance
1031 Auahi Street
Honolulu, HI 96814

Hawaii Alcoholism Foundation Sand Island Treatment Center
Residential Program
12–40 Sand Island Access Road
Honolulu, HI 96819

Hina Mauka/Teen Care
Kalani High School
4680 Kalanianaole Highway
Honolulu, HI 96821

Kalihi Palama Health Clinic
Health Care for Homeless Project
350 Sumner Street
Honolulu, HI 96817

Kokua Kalihi Valley Family Services
1846 Gulick Avenue
Honolulu, HI 96819

Queen's Medical Center Day Treatment Services
1301 Punchbowl Street
Honolulu, HI 96813

Salvation Army Addiction Treatment Services
Continuum of Care Program
Social Detox Unit
3624 Waokanaka Street
Honolulu, HI 96817

Salvation Army Family Treatment Services
Day Treatment Program
Women's Way
845 22nd Avenue
Honolulu, HI 96822

Veterans' Affairs Substance Abuse Treatment Program
300 Ala Moana Boulevard
Suite 1126
Honolulu, HI 96813

Women's Addiction Treatment Services (WATCH) Saint Francis Medical Center
2230 Liliha Street
Honolulu, HI 96817

YMCA Kaimuki-Waialae Palolo Youth Program
4835 Kilauea Avenue
Honolulu, HI 96816

YMCA Outreach Services
1335 Kalihi Street
Honolulu, HI 96819

YMCA Outreach Services School-Based Program
Farrington High School
1564 North King Street
Honolulu, HI 96819

Leilehua High School
Waialua Intermediate/High School
1335 Kalihi Street
Honolulu, HI 96819

Moanalua High School
2825 Ala Ilima Street
Honolulu, HI 96819

Roosevelt High School
1120 Nehoa Street
Honolulu, HI 9681

KAHUKU

Bobby Benson Center
50-660 Kamehameha Highway
Kahuku, HI 96731

Hina Mauka/Teen Care
Kahuku Intermediate and High School
56-490 Kamehameha Highway
Kahuku, HI 96731-2200

KAHULUI

Malama Na Makua A Keiki
388 Ano Street
Kahului, HI 96732

KAILUA

Hawaii Counseling and Education Center Inc. Chemical Dependency Outpatient Treatment
970 North Kalaheo Avenue
Suite C-214
Kailua, HI 96734

Hina Mauka/Teen Care
Kalaheo High School
730 Iliaina Street
Kailua, HI 96734

Olomana High School
42-471 Kalanianaole Highway
Kailua, HI 96734

YMCA Outreach Services School-Based Program
Kailua High School
451 Ulumanu Drive
Kailua, HI 96734

KAILUA KONA

Drug Addiction Services of Hawaii, Inc. (DASH) Kona Office
74-5620-A Polani Road
Kailua Kona, HI 96740

Outpatient Treatment
74-5467 Kaiwi Street
Kailua Kona, HI 96745-2077

KANEOHE

Alcoholic Rehab Services of Hawaii, Inc. DBA Hina Mauka Adult Continuum
45-845 Pookela Street
Kaneohe, HI 96744

Habilitat Inc
45-035 Kuhonu Place
Kaneohe, HI 96744

Hina Mauka/Teen Care
Castle High School
45-386 Kaneohe Bay Drive
Kaneohe, HI 96744

KANEOHE BAY

Substance Abuse Counseling Center Marine Corps Base Hawaii
Kaneohe Bay, HI 96863

KAUNAKAKAI

Hale Hookupaa
Ala Malamalama Street
Kaunakakai, HI 96748

LAHAINA

Teen Challenge Hawaii, Inc.
Olowalu Village
Lahaina, HI 96761

LIHUE

Child and Family Service Kauai Office
4375 Puaole Street, Building B
Lihue, HI 96766

Ke Ala Pono Recovery Center
4371 Puaole Street, Suite B
Lihue, HI 96766

MAKAWAO

Aloha House Adult Residential/ Outpatient Treatment
4593 Ike Drive
Maunaolu Campus
Makawao, HI 96768

MILILANI

Hina Mauka/Teen Care
Milliani High School
95-1200 Meheula Parkway
Mililani, HI 96789

PAHOA

Drug Addiction Services of Hawaii, Inc.
Hui Hoola
15-2927 Government Main Road
Pahoa, HI 96778

PAIA

Maui Youth and Family Services, Inc.
Adolescent Residential Program
1931 Baldwin Avenue
Paia, HI 96779

PEARL CITY

Hina Mauka Teen Care
Pearl City High School
2100 Hookiekie Street
Pearl City, HI 96782

PEARL HARBOR

Naval Counseling and Assistance Center
Comnavbase Pearl Harbor
Pearl Harbor, HI 96860-5020

SHOFIELD BARRACKS

Schofield Barracks Alcohol and Drug Abuse Prevention and Control Program
Building T-695A
Schofield Barracks, HI 96857-5000

TRIPLER ARMY

Tri-Service Addictions Recovery Facility (TRISARF)
1 Jarrett White Road
Tripler Army, HI 96859-5000

WAIANAE

New Horizons Learning Center
98-211 Poli Momi Street
Waianae, HI 96792

Waianae Coast Community Mental Health Center School-Based Program
Nanakuli and Waianae High Schools
86-226 Farrington Highway
Waianae, HI 96792

Waianae Coast Comprehensive Health Center Malama Recovery Services
89-188 Farrington Highway
Waianae, HI 96792

WAIPAHU

Alcohol Rehab Services of Hawaii, Inc.
Hina Mauka/Waipahu Site
94-216 Farrington Highway
Suite B2-306
Waipahu, HI 96797

YMCA Outreach Services School Based Program
Waipahu High School
94-1211 Farrington Highway
Waipahu, HI 96797

IDAHO

BLACKFOOT

Road to Recovery, Inc.
583 West Sexton Street
Blackfoot, ID 83221

BOISE

Aerie Addictions Recovery Center, Inc.
9600 West Brookside Lane
Boise, ID 83703

Alcoholism Intervention Services
4477 Emerald Street
Boise, ID 83706

Boise Center for Recovery Outpatient Services
410 South Orchard Street, Suite 132
Boise, ID 83705

Crossroads Counseling Services
1010 North Orchard Street
Suite 2
Boise, ID 83706-2255

First Step for Women/First Step for Men
1818 West State Street
Boise, ID 83702

Healing Center, Inc.
2503 West State Street
Boise, ID 83702

Nelson Institute
1088 North Orchard Street
Suite 1
Boise, ID 83706

Port of Hope Centers, Inc.
710 North 6th Street
Boise, ID 83706

Saint Alphonsus Addiction Recovery Center
6148 Emerald Street
Boise, ID 83704

Veterans' Affairs Medical Center Substance Abuse Treatment Programs
500 West Fort Street
Boise, ID 83702

YWCA Womens Services
720 West Washington Street
Boise, ID 83702

BONNERS FERRY

Kootenai Tribe Substance Abuse Services
County Road 38-A
Bonners Ferry, ID 83805

CALDWELL

Bell Chemical Dependency Counseling, Inc.
111 East Logan Street
Caldwell, ID 83605

COEUR D'ALENE

Comprehensive Clinical Services
401 1/2 Sherman Avenue
Suite 207
Coeur d'Alene, ID 83814

Idaho Youth Ranch Anchor House
1609 Government Way
Coeur d'Alene, ID 83814

North Idaho Behavioral Health
2301 North Ironwood Place
Coeur d'Alene, ID 83814

Port of Hope Center North
218 North 23 Street
Coeur d'Alene, ID 83814

COTTONWOOD

North Idaho Correctional Institution Road to Recovery
Star Route 3
Cottonwood, ID 83522

EMMETT

Bell Chemical Dependency Counseling, Inc.
621 South Washington Street
Emmett, ID 83617

FORT HALL

Shoshone Bannock Tribal Chemical Dependency Program
Agency Road
Fort Hall, ID 83203

GOODING

Walker Center
1120A Montana Street
Gooding, ID 83330

IDAHO FALLS

Alcohol Rehabilitation Association Phoenix Center
163 East Elva Street
Idaho Falls, ID 83401

Community Alcohol and Drug Treatment Services
589 North Water Avenue
Idaho Falls, ID 83402-3712

LAPWAI

Nez Perce Tribe Alcohol and Substance Abuse
Agency Road Bia Campus
Lapwai, ID 83540

LEWISTON

Port of Hope Family Treatment Centers
828 8th Avenue
Lewiston, ID 83501

Riverside Recovery
1720 18th Avenue
Lewiston, ID 83501

Saint Joseph's Regional Medical Center, Inc.
415 6th Street
Lewiston, ID 83501

NAMPA

Port Of Hope Centers, Inc.
508 East Florida Street
Nampa, ID 83686

Mercy Medical Center for Recovery
1512 12th Avenue
Nampa, ID 83686

OROFINO

State Hospital North Chemical Dependency Unit
300Hospital Drive
Orofino, ID 83544

PAYETTE

Bell Chemical Dependency Counseling
14 South Main Street, Suite 106
Payette, ID 83661

PLUMMER

Coeur D'Alene Tribe Family Healing Center
1115 B Street
Plummer, ID 83851

POCATELLO

Road to Recovery Inc
343 East Bonneville Street
Pocatello, ID 83201-6434

600 East Oak Street
Pocatello, ID 83201

SALMON

Carroll Counseling and Consulting
1301 Main Street Suite 8
Salmon, ID 83467

TWIN FALLS

Canyon View Psychiatric and Addiction Services of Magic Valley Regional Medical Center
228 Shoup Avenue West
Twin Falls, ID 83301

Port of Hope Centers, Inc.
425 2 Avenue North
Twin Fails, ID 83301

Walker Center
263 2nd Avenue North
Twin Falls, ID 83301

WEISER

Bell Chemical Dependence Counseling, Inc.
270 East 7th Street, Suite G
Weiser, ID 83672

ILLINOIS

ADDISON

Serenity House, Inc.
891 South Route 53
Addison, IL 60101

ALBION

Southeastern/Edwards Family Counseling Center/DUI Program
254 South 5th Street
Albion, IL 62806

ALGONQUIN

Alternative Pathways
1107 South Main Street
Algonquin, IL 60102

ALSIP

Southwest YMCA
YMCA of Metropolitan Chicago
Adolescent Outpatient Treatment
3801 West 127th Street
Alsip, IL 60803

ALTON

Chestnut Health Systems, Inc. Outpatient Program
1639 Main Street
Alton, IL 62002

Community Counseling Center of Northern Madison County, Inc.
2615 Edwards Street
Alton, IL 62002

Saint Clare's Hospital Chemical Dependency Treatment Center
915 East 5 Street
Alton, IL 62002

ANNA

Fellowship House
800 North Main Street
Anna, IL 62906

ARLINGTON HEIGHTS

Arlington Center for Recovery LLC
2010 South Arlington Heights Road
Suite 210
Arlington Heights, IL 60005

Comprehensive Behavioral Services
3345-K Arlington Heights Road
Arlington Heights, IL 60004

Hakuju Counseling Center
2010 South Arlington Heights Road
Arlington Heights, IL 60005

Mercy Counseling at Arlington Heights
115 South Wilke Road
Suite 100
Arlington Heights, IL 60005

Omni Youth Services
1616 North Arlington Heights Road
Arlington Heights, IL 60004

AURORA

Association for Individual Development
400 North Highland Avenue
Aurora, IL 60506

Breaking Free, Inc. Family Support
120 Gale Street
Aurora, IL 60506

Community Counseling Center of the Fox Valley, Inc.
400 Mercy Lane
Aurora, IL 60506

El Primer Paso
325 East Galena Boulevard
Aurora, IL 60505

Comprehensive Behavioral Services, Inc.
4260 Westbrook Drive Suite 109
Aurora, IL 60504

Dreyer Medical Clinic Department of Psychiatry
1877 West Downers Place
Aurora, IL 60506

Family Guidance Centers, Inc.
751 Aurora Avenue
Aurora, IL 60505

Opportunity House
469 North Lake Street
Aurora, IL 60506

Project Safe Women's Residential
400 Mercy Lane
Aurora, IL 60505

Provena Mercy Center Alcoholism/Drug Dependency Center
1325 North Highland Avenue
Aurora, IL 60506

Reese Clinical and Consulting Services
205 North Lake Street
Suite 103
Aurora, IL 60506

BATAVIA

Sunrise Growth Center
10 East Wilson Street
Batavia, IL 60510

BEARDSTOWN

Cass County Mental Health
Center Alcoholism Treatment Program
121 East 2 Street
Beardstown, IL 62618

BELLEVILLE

Gateway Foundation, Inc. Belleville Unit
7 North High Street, 3rd Floor
Belleville, IL 62220

Mid-America Behavioral Healthcare Alcohol and Substance Abuse Programs
5 Executive Woods Court
Belleville, IL 62226

Saint Elizabeth's Hospital Addiction Services
211 South 3 Street
Belleville, IL 62222

BENSENVILLE

Bensenville Home Society Lifelink
331 South York Road
Bensenville, IL 60106

BERWYN

McNeal Hospital Behavioral Health Services
3249 South Oak Park Avenue
Berwyn, IL 60402

Youth in Crisis, Inc.
7139 West 34th Street
Berwyn, IL 60402

BLOOMINGDALE

Accurate Caring Therapy Services
201 East Army Trail Road
Bloomingdale, IL 60108

BLOOMINGTON

Alcohol-Impaired Motorists Program (AIM)
505 North Center Street
Bloomington, IL 61701

Chestnut Health Systems, Inc.
Lighthouse/Bloomington Youth
702 West Chestnut Street
Bloomington, IL 61701

Lighthouse/Adult
1003 Martin Luther King Jr. Drive
Bloomington, IL 61701

Countermeasures, Inc.
110 North Center Street
Bloomington, IL 61701

BLUE ISLAND

Guildhaus Halfway House
2413 South Canal Street
Blue Island, IL 60406

2413 West Canal Street
Blue Island, IL 60406

BOLINGBROOK

Interventions/Lifeworks
4040 west Boughton Road
Bolingbrook, IL 60440

BUFFALO GROVE

Compsych Substance Abuse Programs
1130 Lake Cook Road, Suite 280
Buffalo Grove, IL 60089

Leslie S. Berkley and Associates
1207 McHenry Road
Buffalo Grove, IL 60089-1371

Omni Youth Services Substance Abuse Treatment Program
1111 Lake Cook Road
Buffalo Grove, IL 60089

BURR RIDGE

Heritage Corridor Counseling Services, Inc.
60 Shore Drive
Burr Ridge, IL 60521

CAIRO

Community Health Emergency Services
Rural Route 1, Box 11
Cairo, IL 62914

Delta Center, Inc.
1001 Washington Street
Cairo, IL 62914

CALUMET CITY

Comprehensive Counseling and DUI Services
536 Pulaski Road
Calumet City, IL 60409

Gutierrez and Associates
613 Wentworth Avenue
Calumet City, IL 60409-4222

CAMBRIDGDE

Bridgeway Adapt Services DUI
117 South East Street
Cambridge, IL 61238

CANTON

Alcohol and Drug Professionals of Fulton County
401 West Locust Street
Canton, IL 61520

Community Mental Health Center of Fulton and McDonough Counties
229 Martin Avenue
Canton, IL 61520

CARBONDALE

Carbondale DUI and Counseling Program
2015 West Main Street, Suite B
Carbondale, IL 62901

Gateway Foundation
318 East Walnut Street
Carbondale, IL 62901

Gateway Youth Care Foundation Carbondale
1080 East Park Street
Carbondale, IL 62901

Southern Illinois Regional Social Services
604 East College Street
Carbondale, IL 62901

CARLINVILLE

Macoupin County Mental Health
Center Alcoholism Outpatient Center
100 North Side Square
Carlinville, IL 62626

CARLYLE

Community Resource Center
580 8th Street
Carlyle, IL 62231

CARMI

Egyptian Public and Mental Health Dept
200 North Main Cross Street
Carmi, IL 62821

CAROL STREAM

CSTO Counseling Centers
350 South Schmale Road
Suite 180
Carol Stream, IL 60188

CARPENTERSVILLE

Renz Addiction Counseling Center Outpatient Substance Abuse Services
211 West Main Street, Suite 218
Carpentersville, IL 60110

CARTHAGE

Hancock County Mental Health Center, Inc. Substance Abuse Program
607 Buchanan Street
Highway 136
Carthage, IL 62321

CARY

Advantage Group Foundation, Ltd.
400 Habler Road
Cary, IL 60013

CASEYVILLE

Gateway Foundation, Inc.
Caseyville Facility
600 West Lincoln Street
Caseyville, IL 62232

CENTRALIA

Community Resource Center
101 South Locust Street
Centralia, IL 62801

Psychiatric Services
838 East McCord Street
Centralia, IL 62801

Saint Mary's Hospital Alcohol and Substance Abuse Programs
400 North Pleasant Avenue
Centralia, IL 62801

CHAMPAIGN

Carle Clinic Association New Choice Adult Outpatient/ Alcohol/Drug Recovery
809 West Church Street
Champaign, IL 61820

Centerpoint Division of Mental Health Center of Champaign County
1801 Fox Drive
Champaign, IL 61824

LWS Place Alcohol/Drug Education and Outpatient Counseling
605 North Neil Street
Champaign, IL 61820

Prairie Center Health Systems
122 West Hill Street
Champaign, IL 61820

University of Illinois at Chicago Counseling Center
610 East John Street
212 Student Services Building
Champaign, IL 61820

CHARLESTON

Central East Alcohol and Drug Council
Substance Abuse Program
635 Division Street
Charleston, IL 61920

Women's Chemical Dependency Project
726 4th Street
Charleston, IL 61920

Women's Project
1501 1/2 18 Street
Charleston, IL 61920

CHESTER

Chester Memorial Hospital The Newark Center
1900 State Street
Chester, IL 62233

Human Services Center of Southern Metro East
800 Servant Street
Chester, IL 62233

CHICAGO

Academy for Counseling, Inc.
810 East 81st Street
Chicago, IL 60619

Addiction Counseling and Education Services of Catholic Charities of Chicago
721 North Lasalle Street
Chicago, IL 60610

Aftercare Inc
10459 South Kedzie Avenue
Chicago, IL 60655

Alternatives, Inc.
1126 West Granville Avenue
2nd Floor
Chicago, IL 60660

Anixter Center
2001 North Clybourn Street
Chicago, IL 60614

6610 North Clark Street
Chicago, IL 60626

Addiction Recovery for the Deaf
1706 North Kedzie Street
1st Floor
Chicago, IL 60647

Outpatient Substance Abuse Treatment
1401 South California Boulevard
3 East Room 360
Chicago, IL 60608

Association House of Chicago
116 North Kodzie Street
Chicago, IL 60622

Bobby E Wright (CMHC) Alcoholism and Substance Abuse Services
9 South Kedzie Avenue
Chicago, IL 60612

Brass Foundation, Inc.
Substance Abuse Program
1223 West Marquette Road
Chicago, IL 60636

8000 South Racine Avenue
Chicago, IL 60620

Behavioral Health Center
340 East 51st Street
Chicago, IL 60615

Cathedral Shelter of Chicago Higgins Halfway House
207 South Ashland Boulevard
Chicago, IL 60607

Adult Outpatient
1668 West Ogden Avenue
Chicago, IL 60607

Center for Addictive Problems
609 North Wells Street
Chicago, IL 60610

Catholic Health Partners Project Hope
3047 West Cermack Road
Chicago, IL 60623

Center for Alcoholism/Project Coat
9415 South Western Avenue
Chicago, IL 60619

Center for New Horizons
551 East 36th Place
Chicago, IL 60653

Chicago Department of Health Alcohol/Substance Abuse Program
140 North Ashland Avenue
Chicago, IL 60607

Chicago Lakeshore Hospital Chemical Dependence Program
4840 North Marine Drive
Chicago, IL 60640

Chicago Treatment and Counseling Center, Inc. (CTCCI)
555C West Roosevelt Road
Chicago, IL 60607

4453 North Broadway Street
Chicago, IL 60640

Columbia Grant Hospital Chemical Dependence Program
550 West Webster Street
Suite 5-SE
Chicago, IL 60614

Community Counseling Center of Chicago
5710 North Broadway
Chicago, IL 60660

Progressions
4740 North Clark Street
Chicago, IL 60640

Comprehensive Behavioral Services, Inc.
455 North Cityfront Plaza Drive
Chicago, IL 60611

Counseling Center/Lake View Substance Abuse Services
3225 North Sheffield Avenue
Chicago, IL 60657

DUI Counseling Center Bayrach Counseling Services
4059 West 47th Street
Chicago, IL 60632

2334 West Lawrence Street
Chicago, IL 60625

Dimensions of Recovery
2240 South Michigan Avenue
Chicago, IL 60616

El Rincon Community Clinic
1874 North Milwaukee Avenue
Chicago, IL 60647

Englewood Comm. Health Organization (ECHO)
845 West 69th Street
Chicago, IL 60621

Recovery Home Program
1503-05 West 68 Street
Chicago, IL 60636

Erie Family Health Center
1701 West Superior Street
Chicago, IL 60622

Family Guidance Center, Inc.
310 West Chicago Avenue
Chicago, IL 60610

Family Link, Inc.
10 West 35th Street, 2nd Floor
Chicago, IL 60616

Garfield Counseling Center
4132 West Madison Street
Chicago, IL 60624

Gateway Foundation, Inc.
2615 West 63 Street
Chicago, IL 60629

Cook County Jail/SATC
1859 South Ashland Avenue
Chicago, IL 60608

DCFS Case Coordination
4301 West Grand Avenue
Chicago, IL 60651

West Side Treatment Center
3828 West Taylor Street
Chicago, IL 60624

Genesis Family Prevention and Intervention Programs
900 North Franklin Street
Chicago, IL 60610

Great Lakes Psychological Services Substance Abuse Services
111 North Wabash Avenue
Suite 1400
Chicago, IL 60602

Gutierrez and Associates
505 North Lasalle Street Suite 400
Chicago, IL 60610

Habilitative Systems Inc
5930 West Washington Street
Chicago, IL 60644

4350 West 16th Street
Chicago, IL 60623

Haymarket Center
4910 South King Drive
Chicago, IL 60615

108 North Sangamon Street
Chicago, IL 60607

Athey Hall
932 West Washington Street
Chicago, IL 60607

The McDermott Center/Maryville
750 West Montrose Avenue
Chicago, IL 60613-3608

Hazelden/Chicago
867 North Dearborn Street
Chicago, IL 60610

Healthcare Alternative Systems, Inc.
4534 South Western Avenue
Chicago, IL 60609

1942 North California Avenue
Chicago, IL 60647

2755 West Armitage Street
Chicago, IL 60647

1949 North Humboldt Avenue
Chicago, IL 60647

Howard Brown Health Center
4025 North Sheridan Road
Chicago, IL 60613

Human Resources Development Institute
Alcohol/Substance Abuse Program
Womens Outpatient Treatment Services
33 East 114th Street
Chicago, IL 60628

11352 South State Street
Chicago, IL 60628

Englewood Counseling Services
6241 South Halstead Avenue
Chicago, IL 60621

Pre-Release Center
3026 South California Street
Building 3 and 4
Chicago, IL 60608

Women's Residential Services
2311 East 98th Street
Chicago, IL 60617

Humana Health Plan, Inc. Evergreen Center
9415 South Western Avenue
Suite 202
Chicago, IL 60620

Interventions
Central Intake
1234 South Michigan Avenue
Suite 100
Chicago, IL 60605

Crossroads
3401 West 111th Street
Chicago, IL 60655

Northside Clinic
2723 North Clark Street
1st and 2nd Floors
Chicago, IL 60614

South Wood
5701 South Wood Street
Chicago, IL 60636

Kedzie Center
1706 North Kedzie Avenue
Chicago, IL 60647

King Drive Counseling and Referral Services
6252 South Martin Luther King Drive
Chicago, IL 60637

Latino Treatment Center Chicago Outpatient
2608 West Petersen Avenue
Chicago, IL 60659

Loretto Hospital
645 South Central Avenue
Chicago, IL 60644

Lutheran Social Services of Illinois
Edgewater
1758 West Devon Street
Chicago, IL 60660

Kathy Dwyers
1764 West Devon Avenue
Chicago, IL 60660

Men's Residence
1640 West Morse Avenue
Chicago, IL 60626

Mount Greenwood
3220 West 115 Street
Chicago, IL 60655

South Residence
7843 South Essex Avenue
Chicago, IL 60649

Women's Residence
5517 North Kenmore Avenue
Chicago, IL 60640

McDermott Center
108 North Sangamon Street
Chicago, IL 60607

810 West Montrose Street
Chicago, IL 60613-3608

120 North Sangamon Street
Chicago, IL 60607

Mercy Hospital and Medical Center
Alcoholism and Drug Dependency Program
2525 South Michigan Avenue
Chicago, IL 60616

Mercy Medical at Presidential Towers
614 West Monroe Tower 3
Chicago, IL 60606

Mercy Medical on Pulaski
5635 South Pulaski Road
Chicago, IL 60629

Mount Sinai Hospital/Medical Center
California and 15 Streets
Chicago, IL 60608

Near North Health Services Winfield Moody Health Center
1276 North Clybourn Street
Chicago, IL 60610

Nearwest Professional Counseling Residential Services
2207 West 18th Street
Chicago, IL 60608

New Age Services Corporation
701-709 West Roosevelt Road
Chicago, IL 60607

New Pathways Counseling Services, Inc.
4419 North Kedzie Avenue, 3rd Floor
Chicago, IL 60625

NIA Comprehensive Center for Developmental Disabilities
1808 South State Street
Chicago, IL 60616

Northwestern Memorial Hospital Chemical Dependence Program
446 East Ontario Street, 8th Floor
Chicago, IL 60611

Norwod Park Township Family Services
4600 North Harlem Avenue
Chicago, IL 60656

Pilsen Little Village CMHC
3113 West Cermack Road
Chicago, IL 60623

Polish American Association Starting Point
3834 North Cicero Avenue
Chicago, IL 60641

Polish American Addictions Counseling
6901 West Archer Avenue
Chicago, IL 60638

Reed Treatment Clinic III
4004 West Division Street
Chicago, IL 60651

Rogers Park Substance Abuse Center, Ltd.
6926 North Glenwood Street
Chicago, IL 60626

Rosemoor Assessment Substance Abuse Program, Inc.
123 East 103rd Street, Suite 1
Chicago, IL 60628

Rush/Presbyterian St. Luke's Medical Center Alternate Behavior Consultation
1720 West Polk Street
Marshall Field IV Center
Chicago, IL 60612

Saint Elizabeth's Hospital Substance Abuse Treatment Center
1431 North Claremont Avenue
Chicago, IL 60622

Saint Joseph's Hospital Partners Recovery Program
2900 North Lake Shore Drive
Chicago, IL 60657

Salvation Army Harbor Light Center
1515 West Monroe Street
Chicago, IL 60607

Southeast Alcohol and Drug Abuse Center
8640 South Chicago Avenue
Chicago, IL 60617

Substance Abuse Services, Inc.
Outpatient Unit
2101 South Indiana Avenue
Chicago, IL 60616

Substance Abuse Services, Inc. Outpatient
2101 South Indiana Avenue
Chicago, IL 60616

Tarnowski Counseling and Clinical Services
5642 West Diversey Street
Room 107
Chicago, IL 60639

Thresholds Rowan Trees Vincennes House
500 West Englewood Street
Chicago, IL 60621

University of Illinois at Chicago Addiction Services
1740 West Taylor Street, Room C-600
Chicago, IL 60612

Urban Life Line
2149-53 East 83rd Street
Chicago, IL 60617

Veterans' Affairs Medical Center
Alcohol/Substance Abuse Program
820 South Damen Avenue
Chicago, IL 60612

West Side Holistic Family Center
4909 West Division Street
Chicago, IL 60651

Woodlawn Organization, The (TWO)
1447 East 65th Street
Chicago, IL 60637

York Behavioral Health Care
1525 East Hyde Park Boulevard
Chicago, IL 60615

Yos/Albany Park
4751 North Kedzie Avenue
Chicago, IL 60625

Yos/Austin
5912 West Division Street
Chicago, IL 60651

Youth Outreach Services, Inc.
Northwest Youth Outreach/Irving Park
6417 West Irving Park Road
Chicago, IL 60634

Youth Service Project, Inc.
3942 West North Avenue
Chicago, IL 60647

CHICAGO HEIGHTS

Aunt Marthas Youth Service Center
1526 Otto Boulevard
Chicago Heights, IL 60411

CICERO

Chicago Treatment and Counseling Center, Inc. (CTCCI)
1849 South Cicero Avenue
Cicero, IL 60804

Pro Health Advocates, Inc.
5929 West Roosevelt Road
Cicero, IL 60402

Racing Industry Charitable Foundation (RICF)
Hawthorne Racecourse
3701 South Laramie Street
Cicero, IL 60804

Youth Outreach Services
6117 West Cermak Road
Cicero, IL 60804

CLINTON

Dewitt County Human Resource
Center Substance Abuse Treatment Program
1150 Route 54 West
Clinton, IL 61727

CRYSTAL LAKE

Comprehensive Behavioral Services, Inc.
333 Commerce Drive
Crystal Lake, IL 60014

Counseling Center
735-C McArdle Drive
Crystal Lake, IL 60014

Northwest Community Counseling Services
111 South Virginia Avenue
Crystal Lake, IL 60014-5936

Professional Consultations, Inc.
Ambutal Medical Center
4900 South Route 31, Suite 117
Crystal Lake, IL 60012

DANVILLE

Prairie Center Health Systems, Inc.
3545 North Vermilion Street
Danville, IL 61832-1337

Provena United Samaritans Medical Center
Bridgeway Recover Center
600 Sager Avenue
Danville, IL 61832

Veterans' Affairs Medical Center
Alcohol/Drug Dependence Treatment Program
1900 East Main Street
Danville, IL 61832

DECATUR

Behavioral Advocate Group, Inc.
1900 East Lake Shore Drive
Suite 340
Decatur, IL 62521

Chestnut Health Systems, Inc.
2130 North 27th Street
Decatur, IL 62526

Heritage Behavioral Health Center
151 North Main Street
Decatur, IL 62523

Saint Mary's Treatment Center
1800 East Lakeshore Drive
Decatur, IL 62521

DEKALB

Ben Gordon Center Substance Abuse Services Program
12 Health Services Drive
DeKalb, IL 60115

Kishwaukee Hospital Alcohol and Chemical Dependency Treatment Center
626 Bethany Road
DeKalb, IL 60115

DES PLAINES

Family Guidance Centers Inc
1689 Elk Boulevard
Des Plaines, IL 60016

Forest Healthy System, Inc.
555 Wilson Lane
Des Plaines, IL 60016

Holy Family Medical Center Keys to Recovery
100 North River Road
Des Plaines, IL 60016

Relapse Prevention Counseling Center
1330 Webford Street
Des Plaines, IL 60016

DIXON

Adult Education Associates
748 Timbercreek Road
Dixon, IL 61021

Sinnissippi Centers, Inc.
325 Illinois Route 2
Dixon, IL 61021

DOWNERS GROVE

CAP of Downers Grove
5329 Main Street
Downers Grove, IL 60515

Rush Behavioral Health Center
2001 Butterfield Road, Suite 320
Downers Grove, IL 60515

DUNDEE

Professional Consultations
302 West Main Street
Dundee, IL 60118

DUQUOIN

Impact Incarceration Program
Rural Route 1
DuQuoin, IL 62832

EAST HAZELCREST

South Suburban Council on Alcoholism and Substance Abuse
1909 Cheker Square
East Hazelcrest, IL 60429

EAST PEORIA

Ripper and Associates, Ltd.
204 Pinecrest Drive
East Peoria, IL 61611

EAST SAINT LOUIS

Comp. Mental Health Center of Saint Clair County, Inc.
913 Martin Luther King Drive
East Saint Louis, IL 62201

402 North 9th Street
East Saint Louis, IL 62201

129 North 9th Street
East Saint Louis, IL 62201

EDWARDSVILLE

Intensive Outpatient Care
315 North Main Street
Edwardsville, IL 62025

EFFINGHAM

Foil Counseling and DUI Services, Inc.
1901 South 4th Street, Suite 28
Effingham, IL 62401

Heartland Human Services
Guidance and Counseling Center
1108 South Willow Street
Effingham, IL 62401

ELDORADO

Egyptian Public and Mental
Health Dept. Alcohol Outpatient
1412 Highway 45 North
Eldorado, IL 62930

ELGIN

Abacus Program
555 Tollgate Street, Suite A
Elgin, IL 60120

CSTO Counseling Centers, Inc.
115 South Grove Street, Suite 201
Elgin, IL 60120

Latino Treatment Center
54 Fountain Square Plaza
Elgin, IL 60120

Lutheran Social Services of Illinois
675 Varsity Drive
Elgin, IL 60120

Renz Addiction Counseling Center
Substance Abuse Services
76/80 South Grove Avenue
Elgin, IL 60120

Saint Joseph Hospital Cornerstone Program
77 North Airlite Street
Elgin, IL 60120

ELK GROVE VILLAGE

ABLC Behavioral Health Resources
901 Biesterfield Road, Suite 400
Elk Grove Village, IL 60007

Alexian Brothers Medical Center
800 Biesterfield Road
Elk Grove Village, IL 60007

ELMHURST

Elmhurst Memorial Hospital Behavioral Health Services
200 Berteau Avenue
Elmhurst, IL 60126

Kevin and Associates, Inc.
110 Cottage Hill Street, Suite 305
Elmhurst, IL 60126

EVANSTON

Behavioral Health Center
500 Davis Street
Evanston, IL 60201

Evanston Hospital Chapman Center
2650 North Ridge Avenue
Evanston, IL 60201

Peer Services, Inc.
906 Davis Street
Evanston, IL 60201

Saint Francis Hospital Outpatient Addiction Services
355 Ridge Avenue
Evanston, IL 60202

EVERGREEN PARK

Little Company of Mary Hospital Behavioral Health Services
2800 West 95th Street
Evergreen Park, IL 60805

FAIRFIELD

Southeastern/Wayne Family Counseling Center
407 North Basin Drive
Fairfield, IL 62837

FLORA

Clay County Counseling Center
118 West North Avenue
Flora, IL 62839

Southeastern/Clay Family Counseling Center
901 West 3rd Street
Flora, IL 62839

FLOSSMOOR

Family Link, Inc. Counseling and Assessment Center
3608 West Vollmer Road
Flossmoor, IL 60422

FOREST PARK

Riveredge Hospital/One South
8311 West Roosevelt Road One South
Forest Park, IL 60130

FOX LAKE

Western Lake County Alcohol and Drug Dependency Treatment Program
17 West Grand Avenue
Fox Lake, IL 60020

FRANKLIN PARK

Leyden Family Services
Mental Health Center Alcoholism Services
10001 West Grand Avenue
Franklin Park, IL 60131

Leyden Youth Outreach Services
10013-15 West Grand Avenue
Franklin Park, IL 60131

FREEPORT

Alpine Park Center
773 West Lincoln Boulevard
Suite 101
Freeport, IL 61302

Sojourn House, Inc.
565 North Turner Avenue
Freeport, IL 61032

GALENA

Sojourn House, Inc. DUI Program
706 South West Street
Galena, IL 61036

GALESBURG

Bridgeway, Inc. Adapt Services
2323 Windish Drive
Galesburg, IL 61401

Galesburg Cottage Hospital
695 North Kellogg Street
Galesburg, IL 61401

GENESEO

Good Shepherd Foundation
4166 South Oakwood Avenue
Geneseo, IL 61254

GENEVA

Attitude Behavior Modification Systems, Inc.
324 West State Street
Geneva, IL 60134

Comprehensive Behavioral Services, Inc.
825 West State Street, Suite 109
Geneva, IL 60134

GOLCONDA

Family Counseling Center, Inc.
Market and Washington Streets
Golconda, IL 62938

GRANITE CITY

Alcoholic Rehab Community Home Arch House
1313 21st Street
Granite City, IL 62040

Chestnut Health Systems, Inc.
50 Norhtgate Industrial Drive
Granite City, IL 62040-6805

Saint Elizabeth Medical Center/ BHS
2100 Madison Avenue
Granite City, IL 62040

GREAT LAKES

Naval Hospital Alcohol Rehabilitation Department
2705 Sheridan Road
Great Lakes, IL 60088-5234

GREENVILLE

Bond County Health Department Prairie Counseling Center
503 South Prairie Street
Greenville, IL 62246

GURNEE

Michael L Klestinski and Associates
68 Ambrogio Drive
Gurnee, IL 60031

HANOVER PARK

Renz Addiction Counseling Center
7431 Astor Street
Hanover Park, IL 60103

HARVARD

Lutheran Social Services
Division Street, Unit 3
Harvard, IL 60033

HARVEY

Foundation I Center for Human Development Methadone Treatment Unit
15400 South Page Avenue
Harvey, IL 60426

Ingalls Memorial Hospital Health Management Center
1 Ingalls Drive
Wyman Gordon Pavillion, Room 207
Harvey, IL 60426

HAZEL CREST

Mercy Counseling at Hazel Crest
17577 South Kedzie Street
Hazel Crest, IL 60429

Recovery Concepts
17065 Dixie Highway
Hazel Crest, IL 60429

Highland Park Hospital Chemical Dependency Services
718 Glenview Avenue
Highland Park, IL 60035

HILLSBORO

Gateway Foundation, Inc.
Graham Correctional Center Substance Abuse Treatment Center
I55 and Highway 185
Hillsboro, IL 62049

Montgomery County Prevention and Treatment Program
Route 185
Hillsboro, IL 62049

HINSDALE

Interventions Du Page
11 South 250 Route 83
Hinsdale, IL 60521

New Day Center of Hinsdale Hospital
120 North Oak Street
Hinsdale, IL 60521

HOFFMAN ESTATES

Leyden Family Service MH Center Share Program
1776 Moon Lake Road
Hoffman Estates, IL 60194

HOPEDALE

Hopedale Hall Chemical Dependency Program for Older Adults
Railroad and Tremont Streets
Hopedale Medical Complex
Hopedale, IL 61747

INA

Jefferson County Comp Services, Inc. Vantage Program
BMRCC
Ina, IL 62846

JACKSONVILLE

Park Place Center
201 East Morgan Street
Jacksonville, IL 62651

Wells Center
1300 Lincoln Avenue
Jacksonville, IL 62650

Wells Center Department of Corrections
Jacksonville, IL 62650

JERSEYVILLE

Tri-County Counseling Center
220 East County Road
Jerseyville, IL 62052

JOLIET

Healy and Associates
2317 West Jefferson Street
Suite 204
Joliet, IL 60455

Interventions/Lifeworks
214 North Ottawa Street
Joliet, IL 60431

Joliet Counseling Center
54 North Ottawa Street, Suite 120
Joliet, IL 60432

Paramos Counseling Center
815 North Larkin Avenue, Suite 204
Joliet, IL 60435

Saint Joseph Medical Center Substance Abuse Program
333 North Madison Street
Joliet, IL 60435

Silver Cross Hospital Chemical Dependency Unit
1200 Maple Road
Joliet, IL 60432

Stepping Stones, Inc.
1621 Theodore Street
Joliet, IL 60435

Will County Health Department Addiction Services
407 West Jefferson Street
Joliet, IL 60433

William Reid Group
68 North Chicago Street
Joliet, IL 60432

JUSTICE

Mercy Medical In Justice
81 Street and Kean Avenue
Justice, IL 60458

KANKAKEE

Aunt Martha's Youth Service Center, Inc.
335 NorthSchuyler Street
Suite 420
Kankakee, IL 60901

Duane Dean Prevention and Treatment Center
700 East Court Street
Kankakee, IL 60901

New Hope Counseling Center
150 North Schulyer Avenue
Suite 1002
Kankakee, IL 60901

Saint Mary's Hospital of Kankakee
500 West Court Street
Kankakee, IL 60901

LAGRANGE

Elder and Associates Inc
475 West 55th Street
LaGrange, IL 60525

LAKE FOREST

Rush Behavioral Health Center at Lake Forest Hospital
Westmoreland Road
Lake Forest, IL 60045

LAKE VILLA

Gateway Foundation, Inc.
25480 West Cedarcrest Lane
Lake Villa, IL 60046

Lake County Health Dept. Mental
Health Division Outpatient Substance Abuse NW Satellite
121 East Grand Avenue
Lake Villa, IL 60046

Victory Outpatient Chemical Dependency Programs
2031 Grand Avenue, Suite 200
Lake Villa, IL 60046

LAKE ZURICH

Omni Youth Services Ela Township Office
157 East Main Street
Lake Zurich, IL 60047

LA SALLE

North Central Behavioral Health System
2960 Chartres Street
LaSalle, IL 61301

LAWRENCEVILLE

Southeastern Illinois Counseling Centers, Inc.
1501 Olive Street
Lawrenceville, IL 62439

LIBERTYVILLE

Addictions Associates, Inc.
322 Peterson Road
Libertyville, IL 60048

Alliance Institute for the Treatment of Chemical Dependency
501 West Peterson Road
Libertyville, IL 60048

Condell Medical Center Living
Free/Outpatient Addiction Recovery Program
345 North Milwaukee Avenue
Libertyville, IL 60048

LINCOLN

Alcohol and Related Counseling
1411 North Kickapoo Street
Lincoln, IL 62656

Mental Health Centers of Central Illinois
304 8th Street
Lincoln, IL 62656

LOMBARD

Alexian Brothers/Lake Cook Behavioral Health
2 East 22nd Street, Suite 301
Lombard, IL 60148

Elmhurst Memorial Hospital Guidance Center
470 East Roosevelt Road
Lombard, IL 60148

Catholic Charities Diocese of Joliet
26 West Saint Charles Road
Lombard, IL 60148

MACHESNEY PARK

Alpine Park Center
7507 North 2nd Street
Machesney Park, IL 61115-2815

MACOMB

CMHC of Fulton/McDonough
Counties Substance Abuse Services
301 East Jefferson Street
Macomb, IL 61455

McDonough District Hospital Recovery Center
525 East Grant Street
Macomb, IL 61455

MANTENO

Kankakee Minimum Security Unit
37040 South Illinois Street
Route 102
Manteno, IL 60950

Riverside Resolve Center
411 Division Street
Manteno, IL 60950

Substance Abuse Services, Inc. Branden House
800 Bramble Street
Manteno, IL 60950

MARSHALL

Human Resources Center of Edgar and Clark Counties
1006 South 6th Street
Marshall, IL 62441

MARYVILLE

Chestnut Health Systems, Inc.
21487 Vadalabene Road
Maryville, IL 62062

MATTOON

Central East Alcohol and Drug Council
Adolescent Outpatient Services
513 North 13 Street
Mattoon, IL 61938

Outpatient Services
416 North 19 Street
Mattoon, IL 61938

MAYWOOD

Substance Abuse Operations
308 South 5 Avenue
Maywood, IL 60153

The Way Back Inn, Inc.
104 Oak Street
Maywood, IL 60153

201 South 2 Avenue
Maywood, IL 60153

MCHENRY

Family Service and CMHC For McHenry County
5320 West Elm Street
McHenry, IL 60050

Michael L Klestinski and Associates
5400 West Elm Street, Suite 200
McHenry, IL 60050

MELROSE PARK

Procare Recovery Center
1414 West Main Street
Melrose Park, IL 60160

Westlake Community Hospital Substance Abuse Center
1225 Lake Street
Melrose Park, IL 60160

Yos/North Avenue
2140 West North Avenue
Melrose Park, IL 60160

MENDOTA

Mendota Community Hospital DUI/Outpatient Services
1315 Memorial Drive
Mendora, IL 61342

METROPOLIS

MASSAC County Mental Health
206 West 5th Street
Metropolis, IL 62960

MONMOUTH

Bridgeway Adapt Services
219 Euclid Street
Monmouth, IL 61462

MONTICELLO

Piatt County Mental Health Center
1921 North Market Street
Monticello, IL 61856

MORRIS

Grundy County Health Department
1320 Union Street
Morris, IL 60450-2426

Institute for Personal Development
1401 Lakewood Drive, Suite A
Morris, IL 60450

MOUNT CARMEL

Southeastern Counseling Center
Wabash Family
311 West 5th Street
Mount Carmel, IL 62863

MOUNT CARROLL

Sinnissippi Centers, Inc.
1122 Healthcare Drive
Mount Carroll, IL 61053

MOUNT STERLING

Brown County Mental Health Center Alcoholism Services
111 West Washington Street
Mount Sterling, IL 62353

MOUNT VERNON

Jefferson County Comp. Services, Inc. Vantage Point
Route 37 North
Mount Vernon, IL 62864

MUNDELEIN

Omni Youth Services Mundelein Libertyville
505 East Hawley Street
Mundelein, IL 60060

NAPERVILLE

Alpha Counseling Center Inc
25 West 550 Royce Road
Naperville, IL 60565

Linden Oaks Hospital
852 West Street
Naperville, IL 60540

NASHVILLE

Washington County Vocational Workshop
781 East Holzhauer Drive
Nashville, IL 62263

NEWTON

Jasper County Counseling Services
106 East Edwards Street
Newton, IL 62448

Southeastern Illinois Counseling Centers, Inc.
902 West Jourdan Street
Newton, IL 62448

NORTH CHICAGO

Lake County Health Department Behavioral Health Alcoholism Treatment Center
3001 Green Bay Road
Building 126
North Chicago, IL 60064

Northern Illinois Council on Alcohol and Substance Abuse(NICASA) Women's and Children's Program
2031 Dugdale Road
North Chicago, IL 60064

Veterans' Affairs Medical Center Substance Abuse Program
3001 Greenbay Road
Building B-11
North Chicago, IL 60064

NORTHFIELD

Adolescent Substance Abuse Program
405 Central Avenue
Northfield, IL 60093

OAK BROOK

Patricia Ely and Associates
2625 Butterfield Road
Oak Brook, IL 60523

OAK BROOK TERRACE

Alexander Zubenko and Associates
17 West 620 14th Street
Suite 202
Oak Brook Terrace, IL 60181

OAK FOREST

Bremen Youth Services
15350 Oak Park Avenue
Oak Forest, IL 60452

OAK LAWN

Associates in Alcohol and Drug Counseling
8938 South Ridgeland Avenue
Suite 100
Oak Lawn, IL 60453

Christ Hospital and Medical Center Substance Abuse Services
4440 West 95th Street 5 West
Oak Lawn, IL 60453

Crossmont and Associates, Inc.
10522 South Cicero Avenue
Suite 4-A
Oak Lawn, IL 60453

OAK PARK

Education and Intervention, Inc.
1515 North Harlem Avenue, Suite 202
Oak Park, IL 60302-1205

Family Services Center and Mental Health Center of Oak Park and River Forest
120 South Marion Street
Oak Park, IL 60302

Grateful Hand Foundation, Inc. Grateful House
412 South Wesley Avenue
Oak Park, IL 60302

Procare Recovery Center
723 South Boulevard, 1st Floor
Oak Park, IL 60302

York Behavioral Health
1 Erie Court, 4th Floor
Oak Park, IL 60302

Yos/Oak Park
723 South Boulevard
Oak Park, IL 60302

OLNEY

Southeastern Illinois Counseling
Centers, Inc. Alcohol Outpatient Services
4 Micah Drive
Olney, IL 62450

OLYMPIA FIELDS

Intercept Programs, Inc.
20200 Governors Drive
Suite 104-5
Olympia Fields, IL 60461

Olympia Fields
2400 West Lincoln Highway
Suite 107
Olympia Fields, IL 60461

ONARGA

Nexus-Adolescent Chemical Health
212 East Seminary Road
Onarga, IL 60955-0003

ORLAND PARK

William Reid Group
62 Orland Square Drive, Suite 605
Orland Park, IL 60462

OTTAWA

Choices at Community Hospital of Ottawa
1100 East Norris Drive
Ottawa, IL 61350

DUI Assessments and Services
417 West Madison Street
Suite 205
Ottawa, IL 61350

James R. Gage and Associates
1784 Dhessie Lane
Ottawa, IL 61350

PALATINE

Lutheran Social Services
4811 Emerson Avenue
Suite 112
Palatine, IL 60067

The Bridge Youth and Family
Services Comprehensive Prevention
721 South Quintin Road
Palatine, IL 60067

PALOS HEIGHTS

7270 College Drive, Suite 101
Palos Heights, IL 60463

PALOS HILLS

Baxter and Sheehan, Inc.
Palos Hills, IL 60465

PARIS

Human Resources Center of Edgar and Clark Counties
118 East Court Street
Paris, IL 61944

PARK RIDGE

Lutheran General Hospital Addiction Treatment Program
1700 Luther Lane, 2 North Unit
Park Ridge, IL 60068

Maine Township for Addiction
1400 North Northwest Highway
Suite 100
Park Ridge, IL 60068

PAWNEE

Pawnee Counseling Center
528 Douglas Street
Pawnee, IL 62558

PEKIN

Behavioral Medicine at Pekin Hospital Lifeway ACDU
600 South 13th Street
Pekin, IL 61554

Tazwood Center for Human Services
3223 Griffin Avenue
Pekin, IL 61554

PEORIA

Alcohol and Related Counseling
416 Main Street, Suite 619
Peoria, IL 61602

Human Service Center
218 NE Jefferson Avenue
Peoria, IL 61603

New Leaf Retreat
3500 West New Leaf Lane
Peoria, IL 61614

White Oaks Center
3400 New Leaf Lane
Peoria, IL 61615

White Oaks Knolls
2101 West Willow Knolls Drive
Peoria, IL 61615

Proctor Hospital
5409 North Knoxville Street
Peoria, IL 61614

Professional Consultants
411 Hamilton Boulevard
Suite 1000
Peoria, IL 61602

T. W. Mathews and Associates
7501 North University Street
Suite 215
Peoria, IL 61614

PITTSFIELD

Counseling Center of Pike County
121 South Madison Street
Pittsfield, IL 62363

PONTIAC

Institute for Human Resources
310 Torrance Avenue
Pontiac, IL 61764

PRAIRIE VIEW

NICASA
2900 North Main Street
Prairie View, IL 60069

PRINCETON

North Central Behavioral Health Systems
530 Park Avenue East
Princeton, IL 61356

QUINCY

Family Therapy Associates
200 North 8 Street
Suite 111
Quincy, IL 62301

Great River Recovery Resource
428 South 36 Street
Quincy, IL 62301

Newman Clinic
Broadway at 14th Street
Quincy, IL 62305-7005

Park Place Center
301 Oak Street
Quincy, IL 62301

RED BUD

Human Service Center of Southern Illinois
East Substance Abuse Services
10257 State Route 3
Red Bud, IL 62278

RIDGEWAY

Egyptian Public and Mental Health Dept.
711 Main Street
Ridgeway, IL 62979

ROBINSON

Southeastern Counseling Center Crawford Family Counseling Center
204 West Highland Street
Robinson, IL 62454

ROCHELLE

Sinnissippi Centers, Inc.
417 North 6th Street
Rochelle, IL 61068

ROCK FALLS

KSB Hospital Recovery Center
1503 First Avenue
Suite A
Rock Falls, IL 61071

ROCKFORD

Al Tech, Inc. Drug and Alcohol Outpatient
3415 North Main Street
Rockford, IL 61103

Alpine Park Center
5411 East State Street
Suite 212
Rockford, IL 61108

Comprehensive Behavioral Services, Inc.
6016 Fincham Street
Rockford, IL 61108

Family Addiction Instruction Recovery Treatment Center
5301 East State Street
Suite 101
Rockford, IL 61108

PHASE, Inc.
319 South Church Street
Rockford, IL 61101-1316

Rockford Memorial Hospital Addiction Treatment and Education Program
950 South Mulford Road
Rockford, IL 61108

Rosecrance Center
1505 North Alpine Road
Rockford, IL 61107

3815 Harrison Avenue
Rockford, IL 61108

420 East State Street
Rockford, IL 61104-1015

Youth Outpatient Program
1021-23 West Jefferson Street
Rockford, IL 61101

ROCK ISLAND

Alcohol and Drug Education Services
1705 2nd Avenue, Suite 100
Rock Island, IL 61201-8718

Center for Alcohol and Drug
Services Freedom House Clinic I
4230 11th Street
Rock Island, IL 61201

Paul A. Hauck, PhD., Ltd. Substance Abuse Services
1800 3 Avenue
Suite 302
Rock Island, IL 61201

Robert Young Center for Community Mental Health
2701 17 Street
Rock Island, IL 61201

ROLLING MEADOWS

Rolling Meadows Counseling Services
1645 Hicks Road
Rolling Meadows, IL 60008

ROUND LAKE

Northern Illinois Council on
Alcoholism and Substance Abuse
31979 North Fish Lake Road
Round Lake, IL 60073

RUSHVILLE

Schuyler Counseling and Health Services
127 South Liberty Street
Rushville, IL 62681

SAINT CHARLES

Human Resources Development Institute Illinois Youth Center/Valley View
34 W 826 Villa Maria Road
Saint Charles, IL 60174

Renz Counseling Center
230 West River Drive
Saint Charles, IL 60174

SALEM

Community Resource Center
1325-C West Whitaker Street
Salem, IL 62881

SCHAUMBURG

Haymarket Center
1990 East Algonquin Road
Schaumburg, IL 60173

Professional Consultations, Inc.
1650 Moon Lake Boulevard
Schaumburg, IL 60194

Wendy Stebbins and Associates
1701 East Woodfield Road
Suite 415
Schaumburg, IL 60173

SCOTT AFB

375th Medical Group SGOHS Substance Abuse Control Program
310 West Losey Street
Scott AFB, IL 62225-5252

SHELBYVILLE

Central East Alcohol and Drug Council
155 South Morgan Street
Shelbyville, IL 62565

SHERIDAN

Gateway Foundation, Inc.
Sheridan Correctional Facility Substance Abuse Treatment Center
4017 East 2603 Road
Sheridan, IL 60551

SKOKIE

Alon Treatment Center
9150 North Crawford Avenue
Skokie, IL 60076

SPARTA

Human Service Center of Southern Illinois
104 Northtown Road
Sparta, IL 62286

SPRINGFIELD

Alcohol and Addictions Outpatient Center
550 North Street
Springfield, IL 62704

Gateway Foundation, Inc. Springfield Facility
2200 Lake Victoria Drive
Springfield, IL 62703

Midwest Psychological Systems DUI and Substance Abuse Services
987 Clock Tower Drive
Springfield, IL 62704

Personal Consultants
1945 South Spring Street
Springfield, IL 62704

1430 South 8th Street
Springfield, IL 62703

Saint John's Hospital Libertas Program
800 East Carpenter Street
Springfield, IL 62769

Stillmeadow Counseling Center
706 South Grand Avenue West
Springfield, IL 62704

Triangle Center
120 North 11 Street
Springfield, IL 62703

SPRING VALLEY

Spring Valley Outpatient Services
213 East Saint Paul Street
Spring Valley, IL 61362

STERLING

Community Employee Assistance Agency
2804 West Lefevre Road
Sterling, IL 61081

Lutheran Social Services Sterling
1901 First Avenue
Sterling, IL 61081

Sinnissippi Centers, Inc.
2611 Woodlawn Road
Sterling, IL 61081

STREAMWOOD

Streamwood Behavioral Health Center
1400 East Irving Park Road
Streamwood, IL 60107

STREATOR

North Central Behavioral Health Systems
104 6 Street
Streator, IL 61364

Saint Mary's Hospital Behavioral Health Service
111 East Spring Street
Streator, IL 61364

SULLIVAN

Moultrie County Counseling Center Moultrie County DUI Referral
2 West Adams Street
Sullivan, IL 61951

SUMMIT

Des Plaines Valley Community Center Family Outpatient Addiction
7355 West Archer Avenue
Summit, IL 60501

SYCAMORE

Attitude/Behavioral Modification Systems, Inc.
134 West State Street
Sycamore, IL 60178

TAYLORVILLE

Gateway Foundation, Inc. Taylorville Correctional Center
Route 29 South, Box 1000
Taylorville, IL 62568

Triangle Center
320 North Western Avenue
Taylorville, IL 62568

TINLEY PARK

Medical Control Centers
7060 Centennial Drive, Suite 104
Tinley Park, IL 60477

TUSCOLA

Douglas County Drug Alcohol Evaluation and Remedial Education Program
114 West Houghton Street
Tuscola, IL 61953

URBANA

Creative Consultations
302 West Elm Street
Urbana, IL 61801

Prairie Center Health Systems, Inc. Killarney Street Unit
718 Killarney Drive
Urbana, IL 61801

VANDALIA

Community Resource Center
421 West Main Street
Vandalia, IL 62471

Helm DUI Services
716 School Street
Vandalia, IL 62471

Wells Center Vandalia Correctional Center
Vandalia, IL 62471

VERNON HILLS

Lake County Health Department Behavioral Health Women's Residential Services
24647 North Milwaukee Avenue
Vernon Hills, IL 60061

VIENNA

Family Counseling Center, Inc.
408 East Vine Street
Vienna, IL 62995

VILLA PARK

Life Awareness Center, Inc. Adult Outpatient Treatment
335 South Ardmore Street
Villa Park, IL 60181

WATERLOO

Human Support Services of Monroe County Substance Abuse Alternatives
988 North Illinois Route 3
Waterloo, IL 62298

WATSEKA

Iroquois Mental Health Center
Outpatient Alcoholism Program
908 East Cherry Street
Watseka, IL 60970

WAUCONDA

Interventions Contact
26991 Anderson Road
Wauconda, IL 60084

WAUKEGAN

Lake County Health Dept. Behavioral Health Division
MISA Case Management
3012 Grand Avenue
Waukegan, IL 60085

Northern Illinois Council on Alcoholism and Substance Abuse
1113 Greenwood Avenue
Waukegan, IL 60087

Bridge House
3016 Grand Avenue
Waukegan, IL 60085

Victory Memorial Hospital Chemical Dependency Programs
1324 North Sheridan Road
Waukegan, IL 60085

WESTCHESTER

Procare Recovery Center
9855 Roosevelt Road
Westchester, IL 60154

WEST FRANKFORT

Franklin/Williamson Human Services, Inc.
902 West Main Street
West Frankfort, IL 62896

WHEATON

DuPage County Health Department
111 North County Farm Road
Wheaton, IL 60187

Du Page County Psychological Services
421 North County Farm Road
Wheaton, IL 60187

Pape and Associates
618 South West Street
Wheaton, IL 60187

William Reid Group
2100 Manchester Road
Building A, Suite 303
Wheaton, IL 60187

WHEELING

Omni Youth Services
222 East Dundee Road
Wheeling, IL 60090

Scott Bayrach, Ltd.
925 North Milwaukee Avenue
Suite 1016
Wheeling, IL 60090

WINFIELD

Behavioral Health Services of Central Du Page Hospital
27 West 350 High Luke Road
Winfield, IL 60190

WOODRIDGE

Interventions/Woodridge
2221 West 64 Street
Woodridge, IL 6051

New Visions Counseling Services, Inc.
8263 Janes Avenue, Suite I
Woodridge, IL 60517

WOOD RIVER

Wood River Township Hospital Flex Care Program
101 East Edwardsville Road
Wood River, IL 62095

WOODSTOCK

McHenry County Youth Service
Bureau Outpatient Substance Abuse Treatment
101 South Jefferson Street
Woodstock, IL 60098

Centegra/Memorial Medical Center Chemical Dependency Services
527 West South Street
Woodstock, IL 60098

YORKVILLE

Kendall County Health and Human Servs
500-A Countryside Center
Yorkville, IL 60560

ZION

Zion Township Crew
2800 Sheridan Road
Zion, IL 60099

INDIANA

ALBION

Addiction Recovery Centers of Indiana
100 West Main Street
Albion, IN 46701

ANDERSON

Center for Mental Health, Inc.
2020 Brown Street
Anderson, IN 46015

1808 Main Street
Anderson, IN 46015

Community Hospital of Anderson and Madison County
1515 North Madison Avenue
Anderson, IN 46011

Crestview Center
2201 Hillcrest Drive
Anderson, IN 46012

House of Hope of Madison County, Inc.
902 High Street
Anderson, IN 46012

ANGOLA

Cameron Memorial Community Hospital Substance Abuse Services
416 East Maumee Avenue
Angola, IN 46703

Northeastern Center Steuben County Satellite
200 Hoosier Drive
Angola, IN 46703

ATTICA

Wabash Valley Hospital Outpatient Services
101 Suzie Lane
Attica, IN 47918

AUBURN

Northeastern Center Dekalb County Satellite
1800 Wesley Road
Auburn, IN 46706

BATESVILLE

Community Mental Health Center, Inc.
215 East George Street
Batesville, IN 47006

BEDFORD

Center for Behavioral Health, Inc.
Lawrence County Services
1315 Hillcrest Road
Bedford, IN 47421

BEECH GROVE

Saint Francis Hospital and Health Centers Behavioral Health Services
1600 Albany Street
Beech Grove, IN 46107

BLOOMINGTON

BHC Meadows Hospital
3800 North Prow Road
Bloomington, IN 47404

Bloomington Hospital Chemical Dependency Unit
601 West 2nd Street
Bloomington, IN 47402

Partners in Recovery DBA Sunrise Counseling Centers
924 West 17th Street
Bloomington, IN 47404

South Central Community Mental Health Center DBA Center for Behavioral Health
645 South Rogers Street
Bloomington, IN 47403

BLUFFTON

CAP, Inc.
122 Lamar Street
Bluffton, IN 46714

Park Center, Inc. Bluffton Counseling Services
1115 South Main Street
Bluffton, IN 46714

BOONVILLE

Southwest Indiana Mental Health Center, Inc.
315 South 3rd Street
Boonville, IN 47601

BROOKVILLE

Community Mental Health Center, Inc.
Highway 101 and Cooley Road
Brookville, IN 47012

BROWNSBURG

Hill and Associates
23 Boulevard Motif
Brownsburg, IN 46112

CARMEL

Behavior Corporation Substance Abuse Outpatient Services
697 Pro Med Lane
Carmel, IN 46032

CEDAR LAKE

Awakenings
10800 West 133rd Avenue
Suite 2
Cedar Lake, IN 46303

CHURUBUSCO

Wise Choices, Inc.
209 South Main Street
Churubusco, IN 46723

CLINTON

Hamilton Center at Vermillion County Center
825 South Main Street
Suite 207
Clinton, IN 47842

COLUMBIA CITY

Otis R Bowen Center for Human Services, Inc.
201 North Line Street
Columbia City, IN 46725

COLUMBUS

Brumbaugh and Associates
2209 Central Avenue
Columbus, IN 47201

Quinco Consulting Center
806 Jackson Avenue
Columbus, IN 47201

SOAR LLC/Steps of Addiction Recovery
1601 Orinoco Avenue
Columbus, IN 47201

Tara Treatment Center, Inc.
3985 Williamsburg Street
Columbus, IN 47203

CONNORSVILLE

Fayette Memorial Hospital DBA Whitewater Valley Care Pavilion
450 Erie Street
Connersville, IN 47331

CORYDON

Lifespring Mental Health Services Harrison County Office
Corydon/New Middletown Road
Corydon, IN 47112

Recovery Care Center, Inc.
109 North Elm Street
Corydon, IN 47112

CRAWFORDSVILLE

Wabash Valley Hospital, Inc. Outpatient Services
1480 Darlington Avenue
Crawfordsville, IN 47933

DANVILLE

Cummins Mental Health Center, Inc. Addictions Program
6655 East U.S. 36
Danville, IN 46122

Hendricks Community Hospital Mental Health Inpatient Services
1000 East Main Street
Danville, IN 46122

Lebanon Hospital LLC DBA BHC Lebanon Hospital
5250 East U.S. 36
Danville, IN 46122

DECATUR

Park Center, Inc. Decatur Counseling Services
809 South High Street
Decatur, IN 46733

Adams County Memorial Hospital Stress Center
805 High Street
Decatur, IN 46733

DELPHI

Wabash Valley Hospital Outpatient Services
108 North Washington Street
Delphi, IN 46923

DYER

Saint Margaret Mercy Healthcare Centers
24 Joliet Street
Dyer, IN 46311

EAST CHICAGO

Tri-City Comprehensive Mental Health Center, Inc. Substance Abuse Services
3903 Indianapolis Boulevard
East Chicago, IN 46312

4522 Indianapolis Boulevard
East Chicago, IN 46312

ELKHART

Center for Problem Resolution
211 South 5th Street
Elkhart, IN 46516

Renewal Center
401 West Lexington Street
Elkhart, IN 46516

ENGLISH

Southern Hills Counseling Center, Inc. Crawford County Services
523 North Main Street
English, IN 47118-0400

EVANSVILLE

Charter Behavioral Health System
7200 East Indiana Street
Evansville, IN 47715

Chrysalis Addiction Services, Inc.
Outpatient Program
Women's Program
501 John Street, Suite 7
Evansville, IN 47713

Chrysalis Women's Addiction Services
35 East Chandler Streetm Suite 7
Evansville, IN 47713

Evansville State Hospital Addiction Service Unit
3400 Lincoln Avenue
Evansville, IN 47714

Saint Mary Medical Center Behavioral Science Services
3700 Washington Avenue
Evansville, IN 47750

Southwest Indiana Mental Health Center Inc.
Eiseman Annex
12 East Chandler Avenue
Evansville, IN 47713

Moulton Center
1 North Barker Street
Evansville, IN 47712

Robert M Spear Building
415 Mulberry Street
Evansville, IN 47713

Stepping Stone
30 South Stockwell Road
Evansville, IN 47714

Stockwell Center
60 South Stockwell Road
Evansville, IN 47714

Welborn Memorial Baptist Hospital Parkside Addiction Services
500 4th Street
Evansville, IN 47713

FORT WAYNE

Addictive Behaviors Counseling Center, Inc.
6070-B East State Boulevard
Fort Wayne, IN 46815

Allen County Community Corrections Day Reporting Center
109 East Superior Street
Fort Wayne, IN 46802

Alternatives Counseling and Learning Center
3024 Fairfield Avenue, M-404A
Fort Wayne, IN 46807

Alternatives Outreach
2030 Inwood Drive
Fort Wayne, IN 46815

Brown and Associates Consulting, Inc.
2324 Lake Avenue
Fort Wayne, IN 46805-5404

CAP, Inc. Counseling Service
1417 North Anthony Boulevard
Fort Wayne, IN 46805

6001 South Anthony Street
Suite 100
Fort Wayne, IN 46806

Charter Beacon Behavioral Health Systems
1720 Beacon Street
Fort Wayne, IN 46805

Family and Children's Services, Inc.
2712 South Calhoun Street
Fort Wayne, IN 46807

Fort Wayne Women's Bureau, Inc.
Transitions
2435 Oliver Street
Fort Wayne, IN 46802

2440 Bowser Street
Fort Wayne, IN 46803

Hope House
1115 Garden Street
Fort Wayne, IN 46802

1129 Garden Street
Fort Wayne, IN 46802

Lutheran Hospital of Indiana, Inc. Chemical Dependency Services
3024 Fairfield Avenue
Fort Wayne, IN 46804

Park Center, Inc.
909 East State Boulevard
Fort Wayne, IN 46805

Parkview Behavioral Health
1909 Carew Street
Fort Wayne, IN 46805

7230 Engle Road, Suite 240
Fort Wayne, IN 46804

Phoenix Chemical Dependency Program
2200 Lake Avenue, Suite 260
Fort Wayne, IN 46805

Ray of Light Counseling Centers, Inc.
1315 West Main Street
Fort Wayne, IN 46808

Transitions Program
303 East Washington Boulevard
Fort Wayne, IN 46802

Washington House, Inc.
2720 Culbertson Street
Fort Wayne, IN 46802

Wise Choices, Inc.
916 West Coliseum Street
Fort Wayne, IN 46808

FRANKFORT

Howard Community Hospital Community Counseling Center
250 Alhambra Avenue
Frankfort, IN 46041

FRANKLIN

Brumbaugh and Associates
200 East Jefferson Street
Franklin, IN 46131-4450

Tara Treatment Center, Inc.
Alcohol and Drug Treatment Center
6231 South U.S. Highway 31
Franklin, IN 46131

GARRETT

DSM Group/DBA Dekalb Professional Counseling
1202 West Quincy Street
Garrett, IN 46738

GARY

Choices Counseling Service
475 Broadway Street, Suite 404
Gary, IN 46402

Discovery House, Inc.
4195 South Cleveland Street
Gary, IN 46408

Edgewater System for Balanced Living Inc
1100 West 6th Avenue
Gary, IN 46402

Holliday Health Care Professional Corporation
8410 Maple Avenue
Gary, IN 46403

Serenity House of Gary, Inc.
5157 Harrison Street
Gary, IN 46408

GASTON

Interventions, Inc. Muncie Program
6951 North Creek 700 West
Gaston, IN 47342

GOSHEN

Addiction Recovery Centers of Indiana Goshen Addictions Program
114 North Main Street
Goshen, IN 46526

Center for Problem Resolution, Inc.
117 West Washington Street
Goshen, IN 46526

Oaklawn
330 Lakeview Drive
Goshen, IN 46527

GRANGER

Charter Behavioral Health System
6407 North Main Street
Granger, IN 46530

GREENCASTLE

Cummins Mental Health Center, Inc. Greencastle Clinic
308 Medic Way
Greencastle, IN 46135

Discover Recovery LLC
110 South Indiana Street
Greencastle, IN 46135

GREENFIELD

Community Hospitals of Indiana, Inc.
145 Green Meadows Drive, Suite 1
Greenfield, IN 46140

GREENSBURG

Community Mental Health Center, Inc.
1033-B East Freeland Road
Greensburg, IN 47240

GREENWOOD

BHC Valle Vista Health System
898 East Main Street
Greenwood, IN 46013

CPC Valle Vista Health System
896 East Main Street
Professional Building
Greenwood, IN 46142

Indy Interventions
500 Polk Street
Greenwood, IN 46143

Tara Treatment Center, Inc. United Way Center for Human Services
500 South Polk Street Suite 18
Greenwood, IN 46142

HAMMOND

Burgos Counseling Services Inc
6431 Kennedy Avenue
Hammond, IN 46323

HIGHLAND

Relapse Prevention and Recovery Center
2331 45th Street
Highland, IN 46322

HOBART

Charter Behavioral Health System
101 West 61 Avenue and SR 51
Hobart, IN 46342

Southlake Center for Mental Health, Inc.
Southlake Center Associates
1348 South Lake Park Avenue
Hobart, IN 46342

HUNTINGTON

Otis R Bowen Center for Human Services, Inc.
1340 Etna Avenue
Huntington, IN 46750

Parkview Behavioral Health
1215 Etna Avenue
Huntington, IN 46750

INDIANAPOLIS

Adult and Child Mental Health
Center Substance Abuse Services
8320 Madison Avenue
Indianapolis, IN 46227

Alpha Resources
4822 West 34th Street
Indianapolis, IN 46224

Behavior Corporation
Outpatient Services
6100 North Keystone Avenue
Suite 360
Indianapolis, IN 46220

2506 Willowbrook Parkway
Indianapolis, IN 46220

Broad Ripple Counseling Center
6208 North College Street
Indianapolis, IN 46220

1115 Prospect Street
Indianapolis, IN 46203

Charter Behavioral Health System
5602 Caito Drive
Indianapolis, IN 46226

7212 North Shadeland Avenue
Indianapolis, IN 46256

Community Addiction Services of Indiana, Inc.
Mirage Center
4615 North Michigan Road
Indianapolis, IN 46208

Prevention, Intervention and Treatment Services
1040 East New York Street
Indianapolis, IN 46202

5110 Madison Avenue
Indianapolis, IN 46227

Community Hospitals of Indiana, Inc.
6919 East 10th Street, Building C
Indianapolis, IN 46219

Fairbanks Hospital
8102 Clearvista Parkway
Indianapolis, IN 46256

Fallcreek Counseling Service
2511 East 46 Street
Building P
Indianapolis, IN 46205

3500 Lafayette Street
Suite 305
Indianapolis, IN 46222

Family Service Assoc. of Indianapolis Substance Abuse Services
615 North Alabama Street
Room 220
Indianapolis, IN 46204

Life Effectiveness Training
147 East Maryland Street
Indianapolis, IN 46204

520 East 12th Street
Indianapolis, IN 46202

Magellan Behavioral Health
5420 Southern Avenue, Room 401
Indianapolis, IN 46241

Methodist Hospital Substance Abuse Services
1701 NorthSenate Street
Indianapolis, IN 46206

Midtown Community Mental Health Center
832 North Meridian Street
Indianapolis, IN 46204

Project Home
850 North Meridian Street
Indianapolis, IN 46202

Riverside Residential Center
1415 North Pennsylvania Street
Indianapolis, IN 46202

Saint Vincent Hospital and Health Care Center, Inc.
Assisted Living Program
1661 Handball Lane
Indianapolis, IN 46260

Salvation Army Harbor Light Center
927 North Pennsylvania Street
Indianapolis, IN 46204

Volunteers of America, Inc.
611 North Capitol Avenue
Indianapolis, IN 46204

Winona Memorial Hospital Behavioral Health Services
3232 North Meridian Street
Indianapolis, IN 46208

JASPER

Southern Hills Counseling Center
480 Eversman Drive
Jasper, IN 47546

JEFFERSONVILLE

Charter Behavioral Health System
2700 River City Park Road
Jeffersonville, IN 47130

Lifespring Mental Health Services
207 West 13 Street
Jeffersonville, IN 47130

Dual Diagnosis
1401 Mitchell Avenue
Jeffersonville, IN 47130

KENDALLVILLE

Northeastern Center Substance Abuse Services
220 South Main Street
Kendallville, IN 46755

Wise Choice, Inc.
671-B Dowling Street
Kendallville, IN 46755

KNOX

Porter Starke Services, Inc.
1003 Edgewood Drive
Knox, IN 46534

KOKOMO

Howard Community Hospital
Mental Health Center
3500 South LaFountain Street
Kokomo, IN 46902

Psychiatric Services
3548 South LaFountain Street
Kokomo, IN 46902

New Choices
2705 South Berkley Street
Suite 1B
Kokomo, IN 46901

Saint Joseph Hospital Trinity House
1907 West Sycamore Street
Kokomo, IN 46901

LAFAYETTE

Charter Behavioral Health System
3700 Rome Drive
Lafayette, IN 47905

Home With Hope, Inc. Transitional Halfway House
1001 Ferry Street
Lafayette, IN 47901

New Directions, Inc.
360 North 775 East Street
Lafayette, IN 47905

Wabash Valley Hospital, Inc. Outpatient Service
610 Main Street
Lafayette, IN 47901

LAGRANGE

Addiction Recovery Centers of Indiana Cornerstone of Recovery
400 Union Street
LaGrange, IN

Northeastern Center
2155 North Street
LaGrange, IN 46761

LAWRENCEBURG

Community Mental Health Center
427 Eads Parkway
Lawrenceburg, IN 47025

Substance Abuse Services
285 Bielby Road
Lawrenceburg, IN 47025

LEBANON

Behavior Corp Boon County Offices Outpatient Services
602 Ransdell Road
Lebanon, IN 46052

LIGONIER

Northeastern Center
Lincolnway South
Ligonier, IN 46767

LINTON

Hamilton Center, Inc.
1815 North Meridian Street
Linton, IN 47441

Greene County Center
Lonetree Road
Linton, IN 47441

LOGANSPORT

Affiliated Service Providers of Indiana, Inc.
1015 Michigan Avenue
Logansport, IN 46947

Four County Counseling Center
1015 Michigan Avenue
Logansport, IN 46947

LOOGOOTEE

Knox County Hospital Martin County Office
200 John F Kennedy Avenue
Loogootee, IN 47553

MADISON

Lifespring Mental Health Services
319 West 2nd Street
Madison, IN 47250

Madison State Hospital Lou Scalo Center for Adult Addiction
711 Green Road
Madison, IN 47250

MARION

Grant/Blackford Mental Health, Inc.
Branson Place
925 South Branson Street
Marion, IN 46953

Cornerstone
505 Wabash Avenue
Marion, IN 46952

Community Support Program
206 West 8th Street
Marion, IN 46953

Milestone Counseling Services
701 Wabash Avenue
Marion, IN 46952

Trinity House/Saint Joseph Hospital and Health Center
417 South Branson Street
Marion, IN 46953

MARTINSVILLE

Center for Behavioral Health, Inc.
2222 Burton Lane
Martinsville, IN 46151-9405

MERRILLVILLE

Anglican Social Services of Northern Indiana
8555 Grand Boulevard
Merrillville, IN 46401

B Gutierrez and Associates, Inc.
200 East 80th Street, Suite 200
Merrillville, IN 46410

Methodist Hospitals of Gary, Inc. Inpatient Addiction Treatment
8701 Broadway Street
Merrillville, IN 46410

Southlake Center for Mental Health, Inc.
290 A East 90th Drive
Merrillville, IN 46410

8555 Taft Street
Merrillville, IN 46410

MICHIGAN CITY

Saint Anthony Memorial Health Center
301 West Homer Street
Michigan City, IN 46360-3370

Swanson Center Satellite Outpatient
450 St. John Road, Suite 501
Michigan City, IN 46360

MISHAWAKA

Charter Behavioral Health System
2410 Grape Road
Mishawaka, IN 46545

Children's Campus, Inc.
1411 Lincolnway West
Mishawaka, IN 46544

MONTICELLO

Wabash Valley Hospital, Inc. Outpatient Services
207 North Bluff Street
Monticello, IN 47960

MOUNT VERNON

Southwest Indiana Mental Health Center, Inc. Posey Regional Services
100 Vista Drive
Mount Vernon, IN 47620

MUNCIE

AMH, Inc. DBA Associates in Mental Health
3111 West Jackson Street
Muncie, IN 47304

Ball Memorial Hospital
Middletown Center for Chemical Dependency
2401 University Avenue
Muncie, IN 47303

Comprehensive Mental Health Services
240 North Tillotson Avenue
Muncie, IN 47304

MUNSTER

Saint Margaret Mercy Healthcare Centers, Inc. Behavioral Medical Outpatient
312 Ridge Road
Munster, IN 46321

NASHVILLE

Quinco Consulting Center Brown County Consulting Associates
Jefferson and Mound Streets
Nashville, IN 47448

NEW ALBANY

Hedden House
801 Vincennes Street
New Albany, IN 47150

Lifespring Mental Health Services
904 East Spring Street
New Albany, IN 47150

NEW CASTLE

Christian Counseling and Addiction Services, Inc.
New Castle Church of Christ
11th Street
New Castle, IN 47362

502 South Main Street
New Castle, IN 47362

Comprehensive Mental Health Services, Inc.
930 North 14th Street
New Castle, IN 47362

NINEVEH

Tara Treatment Center, Inc.
Ninevah Square
7919 South 100 East
Nineveh, IN 46164

NOBLESVILLE

Behavior Corporation
Noblesville Outpatient Services
54 North 9th Street, Suite 205
Noblesville, IN 46060

Community Addiction Services of Indiana, Inc.
942 North 10th Street
Noblesville, IN 46060

NORTH VERNON

Quinco Consulting of North Vernon
1260 East Buckeye Street
North Vernon, IN 47265

OSGOOD

Community Mental Health Center, Inc.
240 West Craven Street
Osgood, IN 47037

PAOLI

Southern Hills Counseling Center, Inc. Orange County Services
488 West Hospital Road
Paoli, IN 47454

PERU

Four County Counseling Center Miami County Satellite
16 South Broadway Street
Peru, IN 46970

PETERSBURG

Knox County Hospital Pike County Office
400 Main Street
Petersburg, IN 47567

PLYMOUTH

Northern Indiana Hospital LLC DBA BHC of Northern Indiana
1800 North Oak Road
Plymouth, IN 46563

Otis R Bowen Center for Human Services, Inc.
990 Illinois Street
Plymouth, IN 46563

PORTAGE

Porter Starke Services, Inc. Substance Abuse Program
3220 Lancer Street
Portage, IN 46368

PORTLAND

Comprehensive Mental Health Services, Inc.
931 West Water Street
Portland, IN 47371

PRINCETON

Southwest Indiana Mental Health Center, Inc. Gibson Regional Services
310 South 5th Avenue
Princeton, IN 47670

RENSSELAER

Wabash Valley Hospital, Inc. Outpatient Services
1207 East Grace Street
Rensselaer, IN 47978

RICHMOND

Dunn Mental Health Center, Inc.
809 Dillon Drive
Richmond, IN 47375

Addiction Services
831 Dillon Drive
Richmond, IN 47374

Behavioral Health Care Associates
600 Promenade Street
Richmond, IN 47375

Reid Hospital Health Care Services
1401 Chester Boulevard
Richmond, IN 47374

Richmond State Hospital Adult Chemical Dependency
498 NW 18th Street
Richmond, IN 47374

RISING SUN

Community Mental Health Center, Inc.
315 Industrial Access Road
Rising Sun, IN 47040

ROCHESTER

Four County Counseling Center
321 East 8th Street
Rochester, IN 46975

Wayfarer Addictions Counseling
816 1/2 Main Street
Rochester, IN 46975

ROCKPORT

Southern Hills Counseling Center, Inc.
107 North 2nd Street
Rockport, IN 47635

ROCKVILLE

Hamilton Center, Inc. Parke County Center
205 North Jefferson Street
Rockville, IN 47872

RUSHVILLE

Dunn Mental Health Center, Inc.
119 East 3rd Street
Rushville, IN 46173

Rush County Substance Abuse Services
246 North Main Street
Rushville, IN 46173

SALEM

Lifespring Mental Health Services
Highway 60 East
Salem, IN 47167

SCHERERVILLE

Southlake Center for Mental Health, Inc.
2001-A South U.S. Highway 41
Schererville, IN 46375

SCOTTSBURG

Lifespring Mental Health Services
40 East Cherry Street
Scottsburg, IN 47170

SEYMOUR

Quinco Consulting Center Preferred Counseling Associates
321 West Bruce Street, Suite C
Seymour, IN 47274

SHELBYVILLE

Community Hospitals of Indiana, Inc. Gallahue Mental Health Center
7 East Hendricks Street
Shelbyville, IN 46176

SOUTH BEND

Addiction Recovery Centers of Indiana Michiana Addictions Recovery Center
127 West Wayne Street
South Bend, IN 46601

Life Treatment Centers, Inc.
1402 South Michigan Street
South Bend, IN 46613-2214

Madison Center, Inc.
813 South Michigan Street
South Bend, IN 46613

Madison Center for Children
701 North Niles Avenue
South Bend, IN 46617

Madison Hospital
403 East Madison Street
South Bend, IN 46617

Quietcare Building
712 North Niles Avenue
South Bend, IN 46617

Mother Earths Counseling Center for Addictions
1211 Vasaar Avenue
South Bend, IN 46624-0688

Options Institute, Inc.
116 South Taylor Street
South Bend, IN 46601-1522

Pathways Center for Behavioral Health
615 North Michigan Street
South Bend, IN 46601

Victory Clinic Services II
4218 Western Avenue
South Bend, IN 46619

YWCA of Saint Joseph County Mother and Child Program
802 Lafayette Boulevard
South Bend, IN 46601

SPENCER

Center for Behavioral Health, Inc.
751 East Franklin Street
Spencer, IN 47460-1829

Hamilton Center, Inc.
51 South Main Street
Spencer, IN 47460

SULLIVAN

Hamilton Center, Inc. Sullivan County Center
201 West Graysville Street
Sullivan, IN 47882

TELL CITY

Lake Cumberland Regional Hospital 30outhern Hills Counseling Center, Inc.
1443 9th Street
Tell City, IN 47586

TERRE HAUTE

A P and C Clinic PC DBA Associated Psychologists, Inc.
1801 North 6th Street, Suite 600
Terre Haute, IN 47804

Discover Recovery LLC
1509-B Wabash Avenue
Terre Haute, IN 47807

Hamilton Center, Inc.
500 8th Avenue
Terre Haute, IN 47804

Recovery Associates, Inc. Fellowship House
2940 Jefferson Street
Terre Haute, IN 47802

Terre Haute Regional Hospital Lamb Center
3901 South 7 Street
Terre Haute, IN 47802

VALPARAISO

Christian Service Center, Inc.
791 Juniper Street
Valparaiso, IN 46385

Joseph Corporation DBA Care Counseling Services
793-2 Juniper Road
Valparaiso, IN 46385

Porter Memorial Hospital Mother and Child Detox
814 Laporte Avenue
Valparaiso, IN 46383

Porter/Starke Services, Inc.
601 Wall Street
Valparaiso, IN 46383

600 North Vale Park Road
Valparaiso, IN 46383

VERNON

Jennings County Alcohol and Drug Program
28 Perry Street
Vernon, IN 47282

VEVAY

Community Mental Health Center, Inc.
205 West Main Street
Vevay, IN 47043

VINCENNES

Knox County Hospital DBA Samaritan Center
515 Bayou Steet
Vincennes, IN 47591

400 North 1st Street
Vincennes, IN 47591

WABASH

Otis R. Bowen Center for Human Services, Inc.
710 North East Street
Wabash County Hospital Lower Level
Wabash, IN 46992

Parkview Behavioral Health
216 Manchester Avenue
Wabash, IN 46992

WARSAW

Kosciusko Community Hospital Med Park Center
2101 East Center Street
Warsaw, IN 46580

Otis R. Bowen Center for Human Services, Inc.
850 North Harrison Street
Warsaw, IN 46580

WASHINGTON

Knox County Hospital
2007 State Street
Washington, IN 47501

WEST LAFAYETTE

Wabash Valley Hospital, Inc. Riverside
2900 North River Road
West Lafayette, IN 47906

WINAMAC

Four County Counseling Center Pulaski County Satellite
616 West 11th Street
Winamac, IN 46996

WINCHESTER

Dunn Mental Health Center, Inc.
132 North Main Street
Winchester, IN 47394

IOWA

AMES

Center for Addictions Recovery, Inc.
511 Duff Avenue
Ames, IA 50010

Seven 12 House Youth Recovery House
712 Burnett Street
Ames, IA 50010

ANAMOSA

Anamosa State Penitentiary Substance Abuse Program
North High Street
Anamosa, IA 52205

ATLANTIC

Alcohol and Drug Assistance Agency, Inc.
320 Walnut Street
Atlantic, IA 50022

AUDUBON

New View Substance Abuse Center
212 Market Street
Audubon, IA 50025-1136

BURLINGTON

Alcohol and Drug Dependency Services of Southeast Iowa
1340 Mount Pleasant Street
Lincoln Center
Burlington, IA 52601

Burlington Medical Center Riverview Rehabilitation Center
602 North 3 Street
Burlington, IA 52601

Woodlands Treatment Center
4715 Sullivan Slough Road
Burlington, IA 52601

CARROLL

New Vision Substance Abuse Treatment and Prevention Center
322 West 3 Street
Carroll, IA 51401

CEDAR FALLS

Daniel J Murphy, MD
310 West 4th Street
Cedar Falls, IA 50613

CEDAR RAPIDS

Area Substance Abuse Council, Inc.
3601 16 Avenue SW
Cedar Rapids, IA 52404

Mercy Medical Center Sedlacek Treatment Center
701 10 Street SE
Cedar Rapids, IA 52403

Saint Lukes Methodist Hospital Chemical Dependency Services
1030 5th Avenue SE
Cedar Rapids, IA 52403

CHARITON

Southern IA Economic Development Association
115 South Main Street
City Hall
Chariton, IA 50049

Lucas County Health Center
1200 North 7th Street
Chariton, IA 50049

CHEROKEE

Behavioral Health Management Services Synergy Center
1231 West Cedar Loop, Suite 210
Cherokee, IA 51012

CLARINDA

Clarinda Correctional Facility The Other Way Substance Abuse Treatment Program
2000 North 16 Street
Clarinda Treatment Complex
Clarinda, IA 51632

CLINTON

New Directions, Inc. Center for Alcohol and Other Chemical Dependency
217 6th Avenue South
Clinton, IA 52732

2219 Garfield Street
Clinton, IA 52732

Samaritan Health Systems The Bridge
638 South Bluff Boulevard
Clinton, IA 52732

CORYDON

Southern IA Economic Development Association
Courthouse Room 302
Corydon, IA 50060

COUNCIL BLUFFS

Jennie Edmundson Memorial Hospital Addictions Treatment Program
933 East Pierce Street
Council Bluffs, IA 51501

Alegent Health/Mercy Hospital Chemical Dependency Services
800 Mercy Drive
Council Bluffs, IA 51503

DAVENPORT

Center for Alcohol and Drug Services
1523 South Fairmount Street
Davenport, IA 52802

Prevention and Adolescent Services
1601 Harrison Street and Forest Grove Street
Davenport, IA 52802

Country Oaks
12160 Utah Avenue
Davenport, IA 52804

Family Resources, Inc. Wittenmyer Youth Center
2800 Eastern Avenue
Davenport, IA 52803

Genesis Medical Center West Campus Addictions Recovery Programs
West Central Park at Marquette
Davenport, IA 52804

DECORAH

Northeast Iowa Mental Health
Center Alcohol and Related Problems Service Center
905 Montgomery Street
Decorah, IA 52101

DES MOINES

Bernie Lorenz Recovery House, Inc.
4014 Kingman Boulevard
Des Moines, IA 50311

Des Moines General Hospital Gateway Centers
603 East 12th Street
Des Moines, IA 50309

First Step Mercy Recovery Center
1818 48th Street
Des Moines, IA 50310

House of Mercy
1409 Clark Street
Des Moines, IA 50314-1964

Iowa Methodist Medical Center Powell Chemical Dependency Center
700 East University Street
Des Moines, IA 50309

United Community Services
1301 19th Street
Des Moines, IA 50314

VA Central Iowa Health Care System
3600 30th Street
Des Moines, IA 50310-5774

DUBUQUE

Mercy Turning Point Treatment Center
Professional Arts Plaza, Suite 206
Dubuque, IA 52001

Substance Abuse Services Center, Inc.
Nesler Centre
Town Clock Plaza, Suite 270
Dubuque, IA 52001 [ELDORA]

Addiction Management Systems, Inc.
West Edgington Avenue State Training School
Eldora, IA 50627

EMMETSBURG

Marian Behavioral Care Chemical Dependency Services
2508 West Main Street
Emmetsburg, IA 50536

FORT DODGE

Community and Family Resources, Inc.
726 South 17 Street
Fort Dodge, IA 50501

New Life Associates, Inc.
809 Central Avenue, Suite 315
Fort Dodge, IA 50501-4732

Trinity Regional Hospital
802 Kenyon Road
Fort Dodge, IA 50501

FORT MADISON

Iowa State Penitentiary Substance Abuse Program
31 Avenue G
Fort Madison, IA 52627

River Center for Community Mental Health
815 Avenue, Suite H
Fort Madison, IA 52627

HUMBOLDT

Community and Family Resources
19 6th Street South
Humboldt, IA 50548

IDA GROVE

Gordon Recovery
106 Main Street
Ida Grove, IA 51445

INDEPENDENCE

Pathways Behavioral Services, Inc.
209 2 Avenue NE
Independence, IA 50644

IOWA CITY

Mid Eastern Council on Chemical Abuse (MECCA)
430 Southgate Avenue
Iowa City, IA 52240

University of Iowa Hospitals and Clinics Chemical Dependency Center
200 Hawkins Drive
Iowa City, IA 52242

Veterans' Affairs Medical Center Substance Abuse Treatment Program
Highway 6 West, 116A
Iowa City, IA 52246

IOWA FALLS

Freedom House
210 Iowa Street
Iowa Falls, IA 50126

KEOKUK

Alcohol and Drug Dependency Services
5 North 13th Street
Keokuk, IA 52632

River Center for Community Mental Health
208 Bank Street
Keokuk, IA 52632

KEOSAUQUA

Sieda Drug and Alcohol Services
Courthouse/Magistrates Office
115 South Main Street
Keosauqua, IA 52565

LE MARS

Gordon Recovery Center, Inc.
22 First Street NE
Le Mars, IA 51031

MARSHALLTOWN

Substance Abuse Treatment Unit of Central Iowa
9 North 4th Avenue
Marshalltown, IA 50158

MASON CITY

Prairie Ridge
320 North Eisenhower Avenue
Mason City, IA 50401

MITCHELLVILLE

Iowa Correctional Institution for Women
300 Elm Street SW
Mitchellville, IA 50169

MOUNT PLEASANT

Alcohol and Drug Dependency Services
207 South Harrison Street, Suite 4
Mount Pleasant, IA 52641

Mental Health Institute Iowa Residential Treatment Center
1200 East Washington Street
Mount Pleasant, IA 52641

Mount Pleasant Correctional Facility Therapeutic Community Program
1200 East Washington Street
Mount Pleasant, IA 52641

MUSCATINE

Community Health Resources New Horizons Outpatient Substance Abuse Program
1616 Cedar Street
Muscatine, IA 52761

NEW HAMPTON

Pathways Behavioral Services, Inc.
951 North Linn Avenue, Suite 3
New Hampton, IA 50659

NEWTON

Capstone Center, Inc. Substance Abuse Division
306 North 3rd Avenue East
Newton, IA 50208

Newton Correctional Facility Substance Abuse Treatment Program
1203 South 60th Avenue West
Newton, IA 50208

ONAWA

Gordon Recovery Center, Inc.
22 First Street NE
Le Mars, IA 51031

ORIENT

Zion Brown Treatment Center
Rural Route 1, Box 287
Orient, IA 50858-9609

OTTUMWA

Ottumwa Regional Health Center Family Recovery Center
312 East Alta Vista Avenue
Ottumwa, IA 52501

Southern Iowa Economic
Development Assoc. Drug and Alcohol Services
226 West Main Street
Ottumwa, IA 52501

PELLA

Capstone Center, Inc.
712 Union Street
Pella, IA 50219-1768

POCAHONTAS

Community and Family Resources, Inc.
218 1/2 North Main Street
Pocahontas, IA 50574-1624

ROCKWELL CITY

Community and Family Resources
515 Court Street Courthouse Annex
Rockwell City, IA 50579

Tree Substance Abuse Program Trinity Recovery Center Affiliate
North Central Correctional Facility
313 Lanedale Street
Rockwell City, IA 50579

SAC CITY

New View Substance Abuse Center
100 South State Street
Sac City, IA 50583

SIOUX CITY

Gordon Recovery Center, Inc.
Adult Residential
2309 Jackson Street
Sioux City, IA 51104

Outpatient
800 5th Street, Suite 200
Sioux City, IA 51101

Women and Children's Center
2720 Stone Park Boulevard
Sioux City, IA 51104

Mercy Behavioral Care Chemical Dependency Services
4301 Sergeant Road
Sioux City, IA 51106

SPENCER

Northwest Iowa Alcoholism and Drug Treatment Unit, Inc.
1900 Grand Avenue North
Suite E-8
Spencer, IA 51301

STORM LAKE

Vista Addiction and Recovery Center North Campus of Buena Vista County Hospital
1305 West Milwaukee Street
Storm Lake, IA 50588

TAMA

Meskwaki Alcohol/Drug Abuse Center
Tama, IA 52339

WAPELLO

Alcohol Drug Dependency Services
214 Prairie Street
Wapello, IA 52653

WATERLOO

Allen Memorial Hospital Counseling Center
1825 Logan Avenue
Waterloo, IA 50703

Pathways Behavioral Services, Inc.
2222 Falls Avenue
Waterloo, IA 50701

1221 Franklin Street
Waterloo, IA 50703

WAVERLY

Pathways Behavioral Services, Inc.
123 2nd Street NE
Waverly, IA 50677-1763

WEBSTER CITY

Community and Family Resources
914 Willson Street
Webster City, IA 50595

WINTERSET

Madison County Memorial Hospital The Bridge Counseling Center
300 Hutchings Street
Winterset, IA 50273

KANSAS

ABILENE

Dickinson County Council on Alcohol and Other Drugs, Inc.
400 NW 3 Street
Abilene, KS 67410

ARKANSAS CITY

Cowley County Mental Health and Counseling Center
115 East Radio Lane
Arkansas City, KS 67005

Curo Populus Alcohol and Drug Treatment Program
325 North First Street
Arkansas City, KS 67005-1012

ATCHISON

Atchison Valley Hope Alcoholism Treatment Center
1816 North 2 Street
Atchison, KS 66002

New Freedom, Inc. Counseling Services
1600 Skyway Street
Atchison, KS 66002

Northeast Kansas Mental Health Center
1301 North 2nd Street
Atchison, KS 66002

AUGUSTA

Valley Hope at Augusta Medical Complex
2101 Dearborn Street
Augusta, KS 67010

BELOIT

Beloit Juvenile Correctional Facility
1720 North Hersey Street
Beloit, KS 67420

BONNER SPRINGS

Mainstream Inc of Kansas City
12215 State Avenue
Bonner Springs, KS 66012

COLBY

Citizens Medical Center Substance Abuse Services
100 East College Drive
Colby, KS 67701

Thomas County Council on Alcohol/Drug Abuse, Inc.
775 East College Drive
Colby, KS 67701

COLUMBUS

Elm Acres Youth Home for Girls
501 Central Avenue
Columbus, KS 66725

Family Life Center Alcohol and Drug Abuse Program
201 West Walnut Street
Columbus, KS 66725

CONCORDIA

Kerrs Counseling
135 East 6th Street
Concordia, KS 66901

DODGE CITY

New Chance, Inc.
500 East Wyatt Earp Boulevard
Dodge City, KS 67801

Unlimited Recovery Opportunities of Kansas
1111 6th Avenue
Dodge City, KS 67801

EL DORADO

South Central Mental Health, Inc. Counseling Center
2365 West Central Street
El Dorado, KS 67042

ELLSWORTH

Ellsworth Correction Facility
1607 State Street
Ellsworth, KS 67439

EMPORIA

Corner House, Inc.
418 Market Street
Emporia, KS 66801

Counseling and Psychological Services
1512 West 6th Avenue
Emporia, KS 66801

Henderson/Simmons Counseling Services
517 Merchant Street, Suite 200
Emporia, KS 66801

Mental Health Center of East
Central Kansas Alcohol and Drug Services
1000 Lincoln Street
Emporia, KS 66801

Newman Memorial County Hospital Recovery Road
1320 C of E Drive, Suite 5
Emporia, KS 66801

EUREKA

Eureka Substance Abuse Program
612 East 3rd Street
Eureka, KS 67045

FORT LEAVENWORTH

US Army MEDDAC ADAPCP
550 Pope Avenue
Fort Leavenworth, KS 66027-2332

GARDEN CITY

Area Mental Health Center Substance Abuse Services
1111 East Spruce Street
Garden City, KS 67846

Western Kansas Foundation for Alcohol and Chemical Dependency Inc.
811 North Main Street
Garden City, KS 67846

GARNETT

Southeast Kansas Mental Health Center Alcohol and Drug Abuse Services
318 East 6th Street
Garnett, KS 66032

GOODLAND

Northwest Kansas Medical Center Substance Abuse Unit
First and Sherman Streets
Goodland, KS 67735

GREAT BEND

Central Kansas Psychological Services Eldean Kohrs
925 Patton Street
Great Bend, KS 67530

GREENSBURG

Iroquois Center for Human Development
103 South Grove Street
Greensburg, KS 67054

HAYS

Dream, Inc.
765 East 41st Street
Hays, KS 67601

Hays Medical Center Hays Behavioral Health Center
201 East 7th Street
Hays, KS 67601

High Plains Mental Health Center Alcohol and Drug Abuse Services
208 East 7 Street
Hays, KS 67601

Smoky Hill Foundation for Chemical Dependency, Inc.
1106 East 27th Street, Suite 10
Hays, KS 67601

Kelly Center/Fort Hays State University
600 Park Street
Hays, KS 67601

Peters and Associates
1503 Vine Street, Suite B
Hays, KS 67601

HOISINGTON

Women's Recovery Center
1410 North Vine Street
Hoisington, KS 67544

HORTON

Kickapoo Substance Abuse Program
Four Winds Halfway House
Route 1
Horton, KS 66439

HUMBOLDT

Southeast Kansas Mental Health Center
1106 South 9th Street
Humboldt, KS 66748

HUCHINSON

Charter Hutchinson Counseling Center
400 West 2nd Street, Suite C
Hutchinson, KS 67501

Horizons Mental Health Center, Inc. Substance Abuse Services
1715 East 23rd Avenue
Hutchinson, KS 67502-1188

Mirror, Inc.
2100 West Jackson Street
Hutchinson, KS 67501

Reno Alcohol and Drug Services
112 North Poplar Street
Hutchinson, KS 67501

Reno County Community Corrections
400 West 2nd Street, Suite B
Hutchinson, KS 67501

INDEPENDENCE

Four County Mental Health
Center Alcohol and Drug Program
3701 West Main Street
Independence, KS 67301

JUNCTION CITY

Geary Community Hospital Substance Abuse Services
1102 Saint Mary's Road
Junction City, KS 66441

KANSAS CITY

Addiction Stress Center
1330 North 78 Street
Kansas City, KS 66112

Associated Youth Services, Inc.
16205 37th Street
Kansas City, KS 66106

Heart of America Family Services, Inc.
5424 State Avenue
Kansas City, KS 66102

Kansas City Treatment Center
1404 Minnesota Avenue
Kansas City, KS 66102

Kansas City Metro Methadone Program
3901 Rainbow Boulevard
Kansas City, KS 66160

Kansas Multicultural Alcohol and Drug Treatment Center
2940 North 17 Street
Kansas City, KS 66104

Project Turn Around
739 Minnesota Avenue
Kansas City, KS 66101

Salvation Army Shield of Service
1203 Minnesota Avenue
Kansas City, KS 66101

Substance Abuse Center of Eastern Kansas, Inc.
3505 Rainbow Boulevard
Kansas City, KS 66103

Wyandotte Mental Health Center, Inc. Alcohol and Drug Abuse Services
3615 Eaton Street
Kansas City, KS 66103

LANSING

Gateway Foundation Lansing Correctional Facility
Kansas Avenue and Highway 7
Lansing, KS 66043

LARNED

Larned Correctional Mental
Health Facility Substance Abuse Treatment Program
Mental Health Consortium
Route 3
Larned, KS 67550

Larned State Hospital CDRP/ SSH Jung Building
Route 3
Larned, KS 67550

Sunrise, Inc.
523 North Main Street
Larned, KS 67550

LAWRENCE

Alpha Recovery
5020 West 15th Street Suite B
Lawrence, KS 66049

Bert Nash Community Mental Health Center Substance Abuse Services
336 Missouri Street Suite 202
Lawrence, KS 66044

Cedar Branch Recovery Systems PA
14 Westwood Road
Lawrence, KS 66044

DCCCA, Inc. First Step House
345 Florida Street
Lawrence, KS 66044

Haskell Health Center
2415 Massachusetts Street
Lawrence, KS 66049

LEAVENWORTH

Addiction Recovery Services
520 South 4th Street
Leavenworth, KS 66048

Northeast Kansas Mental Health
and Guidance Center Recovery Services of Northeast Kansas
818 North 7 Street
Leavenworth, KS 66048

VA Medical Center Dwight D Eisenhower Substance Abuse Treatment Program
4101 South 4th Street, Suite A-6
Leavenworth, KS 66048

LENEXA

First Things First
9230 Pflumm Road
Lenexa, KS 66215

LIBERAL

Alcohol and Drugs Counseling Services
504 North Kansas Street, Suite B
Liberal, KS 67901

Family Alcohol and Drug Services, Inc.
316 West 7 Street
Liberal, KS 67905

Fernandez/Martin Addiction Counselors
317 North 7th Street, Suite B-5
Liberal, KS 67901

Southwest Kansas Alcoholism and Drug Addiction Services
529 North New York Street
Liberal, KS 67901-0797

MANHATTAN

Edelman Associates
404 Humboldt Street Suite C
Manhattan, KS 66502

Pawnee Mental Health Center Substance Abuse Services
2001 Claflin Street
Manhattan, KS 66502

Peak, Larry M.
1133 College Avenue
Building B Upper Level
Manhattan, KS 66502

Potter, Greg, Ph.D. Alcohol and Drug Abuse Services
714 Poyntz Street, Suite A
Manhattan, KS 66502

MCCONNELL AFB

McConnell Air Force Base Substance Abuse Program
22 MDOS/SGOMH 57950
Leavenworth Street Suite 6E-4
McConnell AFB, KS 67221-3506

MCDONALD

Cheyenne County AB/AO
502 Decatur Avenue
McDonald, KS 67745

MISSION

Mission Valley Hope
5410 West 58th Terrace
Mission, KS 66205

NEWTON

Mirror, Inc.
130 East 5 Street
Newton, KS 67114

Prairie View Mental Health Center Chemical Dependency Treatment
1901 East First Street
Newton KS 67114

OLATHE

Choices
540 East Santa Fe Street
Olathe, KS 66061

Community Outreach Services, Inc.
226 South Kansas Avenue
Olathe, KS 66061

Cypress Recovery, Inc.
230 South Kansas Street
Olathe, KS 66061

Johnson County Adolescent Center for Treatment (ACT)
301 North Monroe Street
Olathe, KS 66061

Total Wellness Center
14161 South Mur Len Street
Olathe, KS 66062

OSKALOOSA

Northeast Kansas Mental Health Center
1102 Walnut Street
Oskaloosa, KS 66066

OTTAWA

Franklin County Mental Health
Clinic, Inc. Substance Abuse Program
204 East 15 Street
Ottawa, KS 66067

OVERLAND PARK

Bridge Way Recovery, Inc.
6800 College Boulevard, Suite 520
Overland Park, KS 66211

Cindy Parkans LSCSW
8100 Marty Street, Suite 102
Overland Park, KS 66204

Interchange
5350 College Boulevard Suite 205
Overland Park, KS 66211

PARSONS

Labette Center for Mental Health Service, Inc. Alcohol and Drug Abuse Program
1730 Belmont Street
Parsons, KS 67357

PITTSBURG

Bartholomew and Dillon Counseling Service
204 North Smith Street
Pittsburg, KS 66762

Crawford County Mental Health Center
Alcohol and Drug Program
3101 Michigan Street
Pittsburg, KS 66762

Elm Acres Youth Home, Inc.
1002 East Madison Street
Pittsburg, KS 66762

Pesciluna Center
401 West Euclid Street
Pittsburg, KS 66762

PRATT

South Central Kansas Foundation on Chemical Dependency, Inc.
501 South Ninnescah Street
Pratt, KS 67124

SAINT JOHN

New Day, Inc.
308 North Gray Street
Saint John, KS 67576

SALINA

Central Kansas Foundation
1805 South Ohio Street
Salina, KS 67401

Dunn Counseling and Consulting, Inc.
1407 South Santa Fe Street
Salina, KS 67401

Saint Francis at Salina
5907 West Cloud Street
Salina, KS 67401

SHAWNEE

Menninger SUCS at Mill Creek
6301 Pflumm Street, Suite 140
Shawnee, KS 66216

Mirror, Inc.
6221 Richards Road
Shawnee, KS 66216

Total Concept EAP
6301 Pflumm Road, Suite 140
Shawnee, KS 66216

SHAWNEE MISSION

Catholic Community Services
10200 West 75th Street
Building B-274
Shawnee Mission, KS 66204

Charles Stebbins Counseling Services
8000 West 127th Street
Shawnee Mission, KS 66213

Clinical Associates PA
7315 Frontage Road, Suite 110
Shawnee Mission, KS 66204

Columbia Health Systems,Inc.
10114 West 105th Street
Suite 100
Shawnee Mission, KS 66212

Shawnee Mission Medical Center Addiction Recovery Unit
9100 West 74th Street
Shawnee Mission, KS 66201

SYRACUSE

Syracuse Chemical Addiction Treatment of Kansas, Inc. (SCAT)
504 North Johnson Street
Syracuse, KS 67878

TOPEKA

ADAPT/TCF
815 SE Rice Road
Topeka, KS 66607

Carole Dorsch Counseling Services
2914 Plass Court, Suite A
Topeka, KS 66611

Relapse Prevention Counseling, Inc.
1913 SW 29th Terrace
Topeka, KS 66611

Saint Francis Hospital Medical Center
1700 SW 7 Street, 3rd Floor
Topeka, KS 66606

Shawnee Community Mental Health Center Substance Abuse Recovery Program
330 SW Oakley Street
Topeka, KS 66606

Shawnee Regional Prevention Center
2209 Southwest 29th Street
Topeka, KS 66603

Sims/Kemper Clinical Counseling
1709 SW Medford Avenue
Topeka, KS 66604

Veterans' Affairs Medical Center Alcohol/Drug Treatment Unit
2200 Gage Boulevard
Building 15-1C
Topeka, KS 66622

Women's Recovery Center
1324 SW Western Street
Topeka, KS 66604

WAMEGO

L and L Assessment and Counseling Center
5245 North Highway 99
Wamego, KS 66547

WELLINGTON

Sumner County Mental Health Center
Wellington, KS 67152

Sumner Mental Health Drug Addiction Services
1601 West 16th Street
Wellington, KS 67152

WICHITA

Addiction Specialist of Kansas, Inc.
650 Carriage Parkway, Suite 135
Wichita, KS 67208

Adolescent/Adult/Family Recovery Program
3540 West Douglas Street
Wichita, KS 67203

Alcoholism Family Counseling Center
714 South Hillside Street
Wichita, KS 67211

Associated Word of Life Counselors Addiction Treatment
3811 North Meridian Street
Wichita, KS 67204

Bharati, Ralph, MD PA
7701 East Kellogg Street
Suite 610
Wichita, KS 67207

Behavior Consultants
1604 North Market Street
Wichita, KS 67220

Center for Human Development
2601 East Central Street
Wichita, KS 67214

Family Psychological Center
804 South Oliver Street
Wichita, KS 67218

Great Meeting is on for Your Success
1015 East 9th Street
Wichita, KS 67214

Hunter Health Clinic, Inc.
2318 East Central Street
Wichita, KS 67214

Indian Alcoholism Treatment Services
313 North Seneca Street
Suite 109
Wichita, KS 67203

Individual and Family Systems Recovery
2400 North Woodlawn Street
Suite 210
Wichita, KS 67220

Knox Center, Inc.
2400 North Woodlawn Street
Suite 210
Wichita, KS 67220

Life Challenges Consulting
566-A South Oliver Street
Wichita, KS 67218

Lighthouse of Wichita Inc
204 South Osage Street
Wichita, KS 67213

Miracles, Inc.
1250 North Market Street
Wichita, KS 67214

Mirror, Inc.
210 North Saint Francis Street
Wichita, KS 67214

New Attitudes, Inc.
9319 East Harry Street, Suite 110
Wichita, KS 67207

New Beginning
2423 East 13th Street
Wichita, KS 67214

Outpatient Drug Alcohol Treatment and Assessment
8911 East Orme Street, Suite B
Wichita, KS 67207

Parallax Program, Inc.
3401 East Funston Street
Wichita, KS 67218

PMA Addiction Medicine
1725 East Douglas Street
Wichita, KS 67211

Recovery Unlimited
3312 West Douglas Street
Wichita, KS 67203

Relapse Prevention Counseling
1333 North Broadway, Suite D
Wichita, KS 67214

Saint Mark United Methodist Church Counseling and Outreach
1525 North Lorraine Street
Wichita, KS 67214

Sedgwick County Dept of Corrections Adult Facility
209 North Emporia Street
Wichita, KS 67203

Sward, Jon M., Ph.D.
1999 North Amidon Street
Suite 211
Wichita, KS 67203

Therapeutic Alliance
1333 North Broadway, Suite D
Wichita, KS 67214

Tiyospaye, Inc.
1856 Woodland Street
Wichita, KS 67203-2742

Wichita Treatment Center
1044 North Waco Street
Wichita, KS 67202

KENTUCKY

ALBANY

Adanta Behavioral Health Services Albany Clinic
Highway 127 South
Albany, KY 42602

ASHLAND

Our Lady of Bellefonte Hospital Chemical Dependency Careunit
Saint Christopher Drive
Ashland, KY 41101

Pathways, Inc.
Withdrawal Unit
201 22nd Street
Ashland, KY 41101

Boyd County Outpatient Unit
Withdrawal Unit
201 22 Street
Ashland, KY 41101

BARBOURVILLE

Cumberland River Comprehensive Care Center
317 Cumberland Avenue
Barbourville, KY 40906

Decisions, Inc.
Knox County Court House
Fiscal Court Conference Room
Barbourville, KY 40906

BARDSTOWN

Caritas Peace Counseling Center
300 North 2nd Street
Bardstown, KY 40004

Communicare Clinic
331 South 3 Street
Bardstown, KY 40004

Family Institute Project Calm
116 East Flaget Avenue
Bardstown, KY 40004

BARDWELL

Western Kentucky MH/MR Board Carlisle County Services
Highway 51 South
Bardwell, KY 42023

BARLOW

Western Kentucky MH/MR Board Ballard County Services
Highway 60
Barlow, KY 42024

BEATTYVILLE

Kentucky River Community Care, Inc.
Beattyville By Pass
Beattyville, KY 41311

BENHAM

Cumberland River Comprehensive Care Center
Tri-Cities Center
Main Street
Benham, KY 40807

BENTON

Western Kentucky MH/MR Board Benton/Marshall County Services
1304 Main Street
Benton, KY 42025

Kentucky River Community Care, Inc.
North Court Square
Booneville, KY 41314

BOWLING GREEN

Bowling Green Professional Associates
959 Lovers Lane
Bowling Green, KY 42103

Leap, Inc.
1733 Campus Plaza Court
Suite 15
Bowling Green, KY 42101-7901

Lifeskills, Inc.
Bowling Green Center
Park Place
822 Woodway
Bowling Green, KY 42102

Prevention Counseling Services
1045 Elm Street
Bowling Green, KY 42101

BRANDENBURG

East Hill Associates
2025 Bypass Road, Suite 1
Brandenburg, KY 40108

BROOKSVILLE

Comprehend, Inc.
Bracken County Community Care Outpatient Drug Services
134 Grandview Drive
Brooksville, KY 41004

BROWNSVILLE

Lifeskills, Inc.
1120 South Main Street
Brownsville, KY 42210

BURKESVILLE

Adanta Behavioral Health Services Burkesville Clinic
390 Keen Street
Burkesville, KY 42717

CAMPBELLSVILLE

Adanta Behavioral Health Services Campbellsville Clinic
3020 Lebanon Road
Campbellsville, KY 42718

CAMPTON

Kentucky River Community Care,
Inc. Wolfe County Health Department
605 Highway 15 South
Suites 1 and 2
Campton, KY 41301

CARLISLE

Bluegrass West Comprehensive
Care Center Nicholas County Comprehensive Care
Post Office Building, Room 4
Carlisle, KY 40311

CARROLLTON

Comprehensive Care Centers of Northern Kentucky
Carroll County Center
1714 Highland Avenue
Carrollton, KY 41008

CLINTON

Western Kentucky MH/MR Board Clinton/Hickman County Services
South Washington Street
Clinton, KY 42031

COLUMBIA

Adanta Behavioral Health Services Columbia Clinic
808 C Jamestown Street
Columbia KY 42728

Westlake Regional Hospital
100 Westlake Drive
Columbia, KY 42728

CORBIN

Baptist Regional Medical Center Adult Chemical Dependency Unit
1 Trillium Way
Corbin, KY 40701

Corbin Professional Associates
1707 Cumberland Falls Road
Falls Road Plaza LL-4
Corbin, KY 40701

Cumberland River
Comprehensive Care Center
American Greetings Road
Corbin, KY 40701

Independence House
3110 Cumberland Falls Highway
Corbin, KY 40701

Decisions, Inc.
801 Master Street, Suite 4
Corbin, KY 40701

COVINGTON

DUI Defendant Referral Systems, Inc.
808 Scott Street
Covington, KY 41011

Lindemann, David
722 Scott Street
Covington, KY 41012

Transitions, Inc.
Women's Residential Addiction Program (WRAP)
1629 Madison Avenue
Covington, KY 41011

CYNTHIANA

Bluegrass West Comprehensive Care
Harrison County Comprehensive Care
122 East Pleasant Street
Cynthiana, KY 41031

DANVILLE

Bluegrass South Comprehensive Care
Court Referral Services
1000 Lexington Road, Suite 1
Danville, KY 40422

Recovery Center
650 High Street
Danville, KY 40422

DAYTON

Transitions, Inc. Droege House
925 5th Avenue
Dayton, KY 41074

DIXON

Community Methodist Hospital DUI Program
Ambulatory Care Center
1355 U.S. Highway 41 A South
Dixon, KY 42409

EDDYVILLE

Western Kentucky Drug and Alcohol Intervention Services, Inc.
1216 Fairview Avenue
Eddyville, KY 42038

EDGEWOOD

Saint Elizabeth Medical Center Chemical Dependency Units
200 Medical Village Drive
Edgewood, KY 41017

EDMONTON

Prevention Counseling Services
1608 West Stockton Road
Edmonton, KY 42129

ELIZABETHTOWN

Caritas Peace Counseling Center
790 North Dixie Highway Suite 800
Elizabethtown, KY 42701

Communicare Recovery Center
1311 North Dixie Avenue
Elizabethtown, KY 42701

Heartland Counseling Services PSC
29 Public Square
Elizabethtown, KY 42701

Hub City Education Services
30 Public Square
Elizabethtown, KY 42701

FAIRDALE

Shelton Counseling
10601 West Manslick Road
Fairdale, KY 40118

FALMOUTH

Comprehensive Care Centers of Northern Kentucky
Pendelton County Center
318 Mountjoy Street
Falmouth, KY 41040

Saint Luke Hospital Alcohol Drug Treatment Center
512 South Maple Avenue
Falmouth, KY 41040

FLEMINGSBURG

Comprehend, Inc. Fleming
County CMHC Outpatient Alcohol and Drug Offices
610 Elizaville Road
Flemingsburg, KY 41041

FLORENCE

Commonwealth Substance Abuse Specialists
7415 Burlington Pike, Suite A
Florence, KY 41042

Comprehensive Care Centers of Northern Kentucky
Boone County Comprehensive Care Center
7459 Burlington Park
Florence, KY 41042

Modlin and Associates Alcohol and Drug Treatment Center and Education Center
2 Dortha Avenue
Florence, KY 41042

FORT CAMPBELL

Community Counseling Services
21st Street and Indiana Avenue
MCXD/CLC Building 2437
Fort Campbell, KY 42223

FORT MITCHELL

Cincinnati Counseling Services, Inc.
100 Chrysler Avenue
FAA Building/Buttermilk Pike
Fort Mitchell, KY 41017

FRANKFORT

Bluegrass Education and Treatment for Addiction
925 Wash Road
Frankfort, KY 40601

Bluegrass West Comprehensive Care Center
Frankfort Office
191 Doctors Drive
Frankfort, KY 40601

Halfway House
943 Wash Road
Frankfort, KY 40601

Counseling Center, Inc.
309 West Main Street
Frankfort, KY 40601

Decisions, Inc.
101 Saint Clair Street
Frankfort, KY 40601

FRANKLIN

Counseling Services RAP
215-B Bluegrass Road
Franklin, KY 42135

Lifeskills, Inc.
112 South High Street
Franklin, KY 42134

FRENCHBURG

Pathways, Inc. Menifee County Outpatient Unit
HCR 69 US 460 west
Frenchburg, KY 40322

FULTON

Fulton County Mental Health Service
350 Browder Street
Fulton, KY 42041

GEORGETOWN

Bluegrass West Comprehensive Care
Scott County Clinic
1226 Paris Pike
Georgetown, KY 40324

Counseling Center, Inc.
137 East Main Street
Georgetown, KY 40324

GLASGOW

Lifeskills, Inc. Barren County Office
608 Happy Valley Road
Glasgow, KY 42142

Prevention Counseling Services
130 North Race Street
Glasgow, KY 42141

GRAYSON

Pathways, Inc. Carter County Outpatient Unit
515 West Main Street
Grayson, KY 41143

GREENSBURG

Adanta Behavioral Health Services Greensburg Clinic
429 Hodgensville Road
Greensburg, KY 42743

Jane Crawford Hospital Behavioral Center
202 Milby Street
Greensburg, KY 42743-1136

GREENUP

Pathways, Inc. Greenup County Outpatient Unit
1018 Walnut Street
Greenup, KY 41144

GREENVILLE

DUI Defendant Referral Systems, Inc.
117 South Main Street
Harbin Library
Greenville, KY 42345

Pennyroyal Mental Health Services Muhlenberg County MH/MR Center
506 Hopkinsville Street
Greenville, KY 42345

HARLAN

Cumberland River Comprehensive Care Center
134 Comp Drive
Harlan, KY 40831

Decisions, Inc.
Harlan County Courthouse
Fiscal Conference Room
1st and Central Street
Harlan, KY 40831

HARRODSBURG

Bluegrass South Comprehensive Care
Crisis Stabilization Unit
710 Perryville Road
Harrodsburg, KY 40330

Ransdell Community Mental Health Center
352 Mr Kwik Shopping Plaza
Harrodsburg, KY 40330

HAZARD

Kentucky River Community Care, Inc.
115 Rockwood Lane
Daniel Boone Parkway
Hazard, KY 41701

HENDERSON

DUI Defendant Referral Systems, Inc.
128 2nd Street, Suite C
Henderson, KY 42420

Employee Assistance DUI Services, Inc.
Citi Center Building
230 Second Street, Suite 308
Henderson, KY 42420

New Choice Center
435 South Y Street
Henderson, KY 42420

Pathways Counseling Services
323 3rd Street
Henderson, KY 42420

Regional Addiction Resources (RAR)
6347 Highway 60 East
Henderson, KY 42420

HINDMAN

Kentucky River Community Care, Inc.
Highway 80
Hindman, KY 41822

HOPKINSVILLE

Alliance Counseling
110 West 2nd Street
Hopkinsville, KY 42240

FHC Cumberland Hall
210 West 17th Street
Hopkinsville, KY 42240-1912

Pennyroyal Center
Adult Clinic
Children's Services
735 North Drive
Hopkinsville, KY 42240

Adolescent Chemical Dependency Program
676 North Drive
Hopkinsville, KY 42240

Volta Program Substance Abuse Treatment Center
Russellville Road Highway 68
Johnson Building
Hopkinsville, KY 42240

Western Kentucky Drug and Alcohol Intervention Services, Inc.
600 South Main Street
Hopkinsville, KY 42240

HYDEN

Kentucky River Community Care, Inc.
Hurts Creek Shopping Center
Post Office Building
Hyden, KY 41749

INEZ

Gateway Counseling Services, Inc.
Main Street
Inez, KY 41224

Mountain Comprehensive Care Center Martin County Clinic
Rockcastle Street, Route 3
Inez, KY 41224

IRVINE

Bluegrass South Comprehensive Care
Irvine Comprehensive Care Center
Handy Brothers Shopping Center
Irvine, KY 40336

ISOM

Kentucky River Community Care, Inc.
Route 7 and Highway 15
Isom, KY 41824

JACKSON

Kentucky River Community Care, Inc.
Outpatient/Next Step
3775 Highway 15 South
Jackson, KY 41339

Sewell Family Children's Center
3875 Highway 15 South
Jackson, KY 41339

JAMESTOWN

Adanta Behavioral Health Services
Jamestown Clinic
Russell School Program
Highway 127 South
Jamestown, KY 42629

Windows of Discovery
Russell County Courthouse
Jamestown, KY 42629

LANCASTER

Garrard Community Mental Health
67 Public Square
Lancaster, KY 40444

LAWRENCEBURG

Bluegrass West Comprehensive
Care Center Lawrenceburg Comprehensive Care Center
1060 Glensboro Road
Lawrenceburg, KY 40342

LEBANON

Communicare
Route 4/Springfield Road
Lebanon, KY 40033

LEITCHFIELD

Communicare
300 South Clinton Street
Health Department Annex
Leitchfield, KY 42754

LEXINGTON

Alcohol Related Offenders Program
1388 Alexandria Drive
Lexington, KY 40504

Anchor Counseling
106 Dennis Drive
Lexington, KY 40503

Baker Programs
174 North Martin Luther King Boulevard
Lexington, KY 40507

Bluegrass Driver School, Inc.
169 East Reynolds Road
Suite 202-A
Lexington, KY 40517

Bluegrass East Comprehensive Care
Aftercare Program
Narcotics Addiction Program
201 Mechanic Street
Lexington, KY 40507

Drug and Alcohol Program
Teen Primary Outpatient Program
200 West 2 Street
Lexington, KY 40507

Forensic Services
177 North Upper Street
Lexington, KY 40507

Growth Resources
1517 Nicholasville Road
Lexington, KY 40503

Pride Program
1101 South Limestone Avenue
Lexington, KY 40508

Charles I Schwartz Chemical Dependency Treatment Center
627 West 4th Street
Lexington, KY 40508

Charter Ridge Behavioral Health System
3050 Rio Dosa Drive
Lexington, KY 40509

Chrysalis House, Inc.
251 East Maxwell Street
Lexington, KY 40508

Chrysalis Family Program
120 Chrysalis Court
Lexington, KY 40508

Clark and Clark
480 West 2nd Street
Lexington, KY 40507

Counseling Center, Inc.
248 East Short Street
Lexington, KY 40507

DUI Defendant Referral Systems
431 South Broadway
Suite 331
Lexington, KY 40508

Family Preservation
570 East Main Street
Lexington, KY 40502

Hope Center
360 West Loudon Street
Lexington, KY 40508

Kentucky Alcohol Offenders
174 North Martin Luther King Boulevard
Lexington, KY 40507

Leap, Inc.
174 North Martin Luther King Boulevard
Lexington, KY 40507

Lexington Professional Associates
1718 Alexandria Drive, Suite 204
Lexington, KY 40504

Modlin and Rulli Alcohol and Drug Treatment and Education Center
174 North Martin Luther King Boulevard
Lexington, KY 40507

Morton Center, Inc.
535 West 2nd Street
Lexington, KY 40508

Patti Hard Marriage and Family Therapy
1517 Nicholasville Road
Lexington, KY 40503

Saint Joseph Hospital
One Saint Joseph Drive
Lexington, KY 40504

Samaritan Hospital
310 South Limestone Street
Lexington, KY 40508

Shepherds House, Inc.
154 Bonnie Brae Drive
Lexington, KY 40508

University Hospital
800 Rose Street
Lexington, KY 40536-0226

Van Hoose and Associates
501 Darby Creek Road, Suite 3
Lexington, KY 40509

Veterans' Affairs Medical Center Substance Abuse Treatment Program
2250 Leestown Road
Lexington, KY 40511

LIBERTY

Adanta Behavioral Health Services Liberty Clinic
112 Liberty Square
Liberty, KY 42539

LONDON

Decisions, Inc.
c/o Best Western
Highway East 80
London, KY 40741

Windows of Discovery
105 South Broad Street
London, KY 40741-1800

LOUISA

Gateway Counseling Services, Inc.
Jefferson Lawrence County Library
102 West Main Street
Louisa, KY 41230

Pathways, Inc. Lawrence County Outpatient Unit
314 East Madison
Louisa, KY 41230

LOUISVILLE

Alcohol Awareness Counseling
4400 Breckenridge Lane
Breckenridge Business Center
Suite 307
Louisville, KY 40201

Baptist Hospital East Chemical Dependency Program Center for Behavioral Health
4000 Kresge Way
Louisville, KY 40207

Bluegrass Pleasant Grove Counseling Center
5330 South 3rd Street, Suite 114
Louisville, KY 40214

4801 Sherburn Lane, Suite 203
Louisville, KY 40207

Bumpas, Thomas J.
6000 Brownsboro Park Boulevard
Suite G
Louisville, KY 40207

Caritas Peace Counseling Center
2120 Newburg Road, Suite 200
Louisville, KY 40205

Charter Louisville Behavioral Health System
1405 Browns Lane
Louisville, KY 40207

Chemical Dependency Counseling
4342 Taylor Boulevard
Louisville, KY 40215

Counseling Center, Inc.
2210 Goldsmith Lane, Suite 126
Louisville, KY 40205

David Harmon and Associates
4010 Dupont Circle, Suite 226
Louisville, KY 40207

Eastern Star Baptist Church
824 South 24th Street
Louisville, KY 40211

Dismas Charities Drug and Alcohol Treatment Program
1501 Lytle Street
Louisville, KY 40203

Dr. Donald T. Stokes and Associates, Inc.
1941 Bishop Lane
Watterson City West Building
Suite 505
Louisville, KY 40218

Dual Diagnosis Unit Central State Hospital
10510 Lagrange Road
Louisville, KY 40223

Healing Place for Women
1607 West Broadway
Louisville, KY 40203

Interlink Counseling Services, Inc.
8311 A and B Preston Highway
Louisville, KY 40219

Jefferson County Drug Court
2516 West Madison Street
Louisville, KY 40211

John P. Sohan Counseling Services
1169 Eastern Parkway
Medical Arts Building, Suite 3358
Louisville, KY 40217

Kentucky Correctional Institute for Women Alcohol and Drug Abuse Program
600 South Preston Street
Louisville, KY 40202

Leap, Inc.
5201 Dixie Highway
Louisville, KY 40216

310 West Liberty Street
Louisville, KY 40202

Lighthouse Adolescent Recovery Center
1935 Bluegrass Avenue
Louisville, KY 40215

Methadone/Opiate Rehabilitation and Education Center
1448 South 15th Street
Louisville, KY 40210

Morton Center, Inc.
982 Eastern Parkway
Kosair Charities Center
Louisville, KY 40217

Norton Psychiatric Clinic
200 East Chestnut Street
Louisville, KY 40232

Rehabilitation and Recovery, Inc.
1169 Eastern Parkway, Suite 1138
Louisville, KY 40217

Seven Counties Services/ Jefferson Alcohol and Drug Abuse Center
600 South Preston Street
Louisville, KY 40202

Tabler/Dawson and Associates
2520 Bardstown Road
Louisville, KY 40201

Talbot House
520 West Saint Catherine Street
Louisville, KY 40203

Ten Broeck Hospital Substance Abuse Services
8521 La Grange Road
Louisville, KY 40242

Triad Recovery Center
214 South 8th Street
Louisville, KY 40202

Volunteers of America
Freedom House
1432 South Shelby Street
Louisville, KY 40217

Third Step Program
1436 South Shelby Street
Louisville, KY 40217

Wellness Institute
332 West Broadway
Suite 1707
Louisville, KY 40202

Whelan, Patrick
1238 East Broadway
Louisville, KY 40204

MADISONVILLE

DUI Defendant Referral System, Inc.
333 1/2 Union Street
Madisonville, KY 42431

Madisonville Regional Medical Center Addiction Recovery Center (ARC)
Hospital Drive
Madisonville, KY 42431

Pennyroyal Mental Health Services
1303 West Noel Street
Madisonville, KY 42431

MANCHESTER

Cumberland River Comprehensive Care Center
Route 9, Box 940
Manchester, KY 40962

DUI Defendant Referral Systems, Inc.
224 White Street
Manchester, KY 40962

MARION

Community Methodist Hospital
212 West Depot Street
Marion, KY 42064

MAYFIELD

Western Kentucky Drug and Alcohol Intervention Services, Inc.
1301 Princeton Drive
Mayfield, KY 42066

William H. Fuller Memorial Substance Abuse Center
1525 Cuba Road
Mayfield, KY 42066

MAYSVILLE

Comprehend, Inc. Mason County CMHC
611 Forest Avenue
Maysville, KY 41056

MCKEE

Cumberland River Comprehensive Care Center
McKee, KY 40447

MIDDLESBORO

Cumberland River Comprehensive Care Center
324 1/2 North 19th Street
Middlesboro, KY 40965

Decisions, Inc.
Days Inn Conference Room B
1252 North 12th Street
Middlesboro, KY 40965

MONTICELLO

Adanta Behavioral Health Services Wayne County Clinic
1994 North Main Street
Monticello, KY 42633

Windows of Discovery
Wayne County Courthouse
Monticello, KY 42633

MOREHEAD

Pathways, Inc. Rowan County Outpatient Unit
321 East Main Street
Morehead, KY 40351

MORGANTOWN

Lifeskills, Inc.
211 East Logan Street
Morgantown, KY 42261

MOUNT STERLING

Pathways, Inc.
Hillcrest Hall
2479 Grassy Lick Road
Mount Sterling, KY 40353

Montgomery County Outpatient Unit
300 Foxglove Drive
Mount Sterling, KY 40353

MOUNT VERNON

Cumberland River Comprehensive Care Center
Mount Vernon, KY 40456

Decisions, Inc.
Rock Castle County Courthouse
Circuit Court Room
Main Street
Mount Vernon, KY 40456

MUNFORDVILLE

Lifeskills, Inc. Hart County Office
118 West 3 Street
Munfordville, KY 42765

MURRAY

Behavioral Medicine, Inc.
100 North 6th Street
Murray, KY 42071-2000

Western Kentucky MH/MR Board
Murray/Calloway County MH/ MR Services
903 Sycamore Street
Murray, KY 42071

NEWPORT

Comprehensive Care Centers of Northern Kentucky
Campbell County Center
10th and Monmouth Streets
Newport, KY 41071

Modlin and Associates
1699 Monmouth Street
Newport, KY 41071

Transitions, Inc.
York Street House
601 York Street
Newport, KY 41071

NICHOLASVILLE

Bluegrass East Comprehensive Care
Jessamine County Center
324 Southview Drive
Nicholasville, KY 40356

OWENSBORO

Employee Assistance DUI Services, Inc.
5000 Backsquare Drive
Building C
Owensboro, KY 42301

Owensboro Area Shelter and Information Services (OASIS)
Owensboro, KY 42302

Saradon Center
920 Frederica Street
Midtown Office Complex
Suite 410
Owensboro, KY 42301

OWENTON

Comprehensive Care Centers of Northern Kentucky
114 West Brown Street
Owenton, KY 40359

OWINGSVILLE

Pathways, Inc.
Bath County Outpatient
Route 36
Owingsville, KY 40360

PADUCAH

Behavioral Medicine, Inc.
102 South 31 Street
Paducah, KY 42001

Charter Hospital of Paducah Substance Abuse Program
435 Berger Road
Paducah, KY 42001

Western Kentucky Drug and Alcohol Intervention Services, Inc.
6th Street
Irvin Cobb Hotel
Paducah, KY 42001

Joseph L. Friedman Substance Abuse Center
1405 South 3 Street
Paducah, KY 42003

PAINTSVILLE

Gateway Counseling Services, Inc.
U.S. 23 North
Wiley Complex
Paintsville, KY 41240

Mountain Comprehensive Care Center Johnson County Clinic
1024 Broadway
Paintsville, KY 41240

PARIS

Bluegrass West Comprehensive Care Center Bourbon County
269 East Main Street
Paris, KY 40361

Stoner Creek Psychiatric Center Burbon Community Hospital
9 Linville Drive
Paris, KY 40361

PIKEVILLE

DUI Defendant Referral Systems, Inc.
419 3rd Street
Pikeville, KY 41501

Gateway Counseling Services, Inc.
89 Division Street
Pikeville, KY 41501

Mountain Comprehensive Care Center Pike County Outpatient Clinic
804 Hambley Boulevard, Suite 4
Pikeville, KY 41501

PINEVILLE

Cumberland River Comprehensive Care Center
110 Kentucky Avenue
Pineville, KY 40977

PRESTONSBURG

Gateway Counseling Services, Inc.
Highway 1428 South
Prestonsburg, KY 41653

Mountain Comprehensive Care Center
Layne House
965 South Lake Drive
Prestonsburg, KY 41653

Outpatient Services
18 South Front Avenue
Prestonsburg, KY 41653

PRINCETON

Pennyroyal Mental Health
Services Caldwell County Mental Health Center
115 McGoodwin Street
Princeton, KY 42445

Western Kentucky Drug and Alcohol Intervention Services, Inc.
108 West Main Street
Princeton, KY 42445

RADCLIFF

Lincoln Trail Hospital United Health Care
3909 South Wilson Road
Radcliff, KY 40160

RICHMOND

Comprehensive Care Center
415 Gibson Lane
Richmond, KY 40475

RUSSELLVILLE

Lifeskills, Inc.
237 East 6 Street
Russellville, KY 42276

SALYERSVILLE

Lifestyle Counseling
Old Fire Station Jockey Lot
Salyersville, KY 41465

Mountain Comprehensive Care Center Magoffin County Clinic
145 Allen Drive
Highway 114
Salyersville, KY 41465

SANDY HOOK

Pathways, Inc. Elliott County Outpatient Unit
Route 17 and Route 132
Sandy Hook, KY 41171

SCOTTSVILLE

Lifeskills, Inc. Scottsville Counseling Center
512 Bowling Green Road
Scottsville, KY 42164

SHELBYVILLE

Creative Spirits
615 Washington Street
Shelbyville, KY 40065-1131

Family Institute Project Calm
702 Washington Street
Shelbyville, KY 40065

Insight Outfitters
935 Trout Lane
Shelbyville, KY 40065

SHEPHERDSVILLE

Trummell and Associates Counseling Center
1729-A Highway 44 East
Shepherdsville, KY 40165

SMITHLAND

Livingston County MH/MR Services
Highway 60
McKinney Building
Smithland, KY 42081

SOMERSET

Adanta Behavioral Health Services
Somerset Clinic
101 Hardin Lane
Somerset, KY 42501

Lake Cumberland Regional Hospital
305 Langdon Street
Somerset, KY 42501

Pulaski Child and Adolescent Services
104 Hardin Lane
Somerset, KY 42501

Windows of Discovery
107 West Mount Vernon Street
Somerset, KY 42501

SOUTHGATE

Commonwealth Substance Abuse Specialists
525 Alexandria Pike
South Hills Medical Center
Suite 100
Southgate, KY 41071

SOUTH WILLIAMSON

Mountain Comprehensive Care Center
2000 Central Avenue
South Williamson, KY 41503

SPRINGFIELD

Springfield Counseling Services
208-C West Main Street
Springfield, KY 40069

STANFORD

Bluegrass South Comprehensive Care Court Referral
410 Anderson Heights
Stanford, KY 40484

Fort Logan Comprehensive Care Center
110 Somerset Street
Stanford, KY 40484

STANTON

Bluegrass East Comprehensive Care Center Stanton Unit
354 West College Street
Stanton, KY 40380

TOMPKINSVILLE

Lifeskills, Inc.
200 East 4 Street
Tompkinsville, KY 42167

VANCEBURG

Comprehend, Inc. Lewis County CMHC Outpatient Alcohol and Drug Office
502 2nd Avenue
Vanceburg, KY 41179

VERSAILLES

Bluegrass West Comprehensive Care
Woodford County Center
125b Big Sink Pike
Versailles, KY 40383

WARSAW

Commonwealth Substance Abuse Specialists
100 West High Street
Warsaw, KY 41095

Comprehensive Care Centers of Northern Kentucky
Gallatin City Center
203 West Martin Street
Warsaw, KY 41095

WEST LIBERTY

Pathways, Inc.
Morgan County Outpatient Unit
280 Prestonsburg Street
Morgan County Office Building
West Liberty, KY 41472

WHITESBURG

Whitesburg DUI Service Agency
117 Hayes Street, Suite 203
Whitesburg, KY 41858

WHITLEY CITY

Adanta Behavioral Health Services
Whitley City Clinic
South Fork Centera
Highway 27
Whitley City, KY 42653

WILLIAMSBURG

Cumberland River Comprehensive Care Center
Cemetary Road
Williamsburg, KY 40769

WILLIAMSTOWN

Comprehensive Care Centers of Northern Kentucky
Grant County Center
308 Barnes Road
Williamstown, KY 41097

Modlin and Rulli Alcohol and Drug Treatment and Education Center
214-C South Main Street
Williamstown, KY 41097

WINCHESTER

Bluegrass East Comprehensive Care
26 North Highland Street
Winchester, KY 40391

Counseling Center, Inc.
52 North Maple Street
Winchester, KY 40391

LOUISIANA

ALEXANDRIA

Crossroads Regional Hospital Substance Abuse Services
110 John Eskew Drive
Alexandria, LA 71315

Louisiana Black Alcoholism Council, Inc.
2403 Harris Street
Alexandria, LA 71307

Veterans' Affairs Medical Center
Chemical Dependency Clinic
Building 6, 116E
Alexandria, LA 71301

BASTROP

Bastrop Alcohol and Drug Abuse Clinic
218 North Franklin Street
Bastrop, LA 71220

BATON ROUGE

Alcohol and Drug Abuse Council of Greater Baton Rouge
1801 Florida Boulevard
Baton Rouge, LA 70802

BHC Meadow Wood Hospital Center for Addictive Disorders
9032 Perkins Road
Baton Rouge, LA 70810

Baton Rouge Area Alcohol and Drug Center, Inc.
1819 Florida Boulevard
Baton Rouge, LA 70802

Baton Rouge Substance Abuse Clinic
4615 Government Street
Building A
Baton Rouge, LA 70806

Behavioral Health Center
3601 North Boulevard
Baton Rouge, LA 70806

Community Counseling
2356 Drusilla Lane
Baton Rouge, LA 70809

Louisiana Health and Rehabilitation Options
2744 Florida Boulevard
Baton Rouge, LA 70802

O'Brien House
1231 Laurel Street
Baton Rouge, LA 70802

Our Lady of the Lake Hospital Tau Chemical Dependency Center
8080 Margaret Ann Drive
Baton Rouge, LA 70809

Salvation Army
7361 Airline Highway
Baton Rouge, LA 70805

Serenity House, Inc.
3370 Victoria Drive
Baton Rouge, LA 70805

BELLE CHASSE

Plaquemines Alcohol and Drug Abuse Clinic
3708 Main Street
Belle Chasse, LA 70037

BOGALUSA

Washington Parish Alcohol and Drug Abuse Clinic
2601 Avenue F
Bogalusa, LA 70427

CHALMETTE

Saint Bernard Alcohol and Drug Abuse Clinic
2712 Palmisano Boulevard
Building A
Chalmette, LA 70043

CHARENTON

Chitimacha Human Services Department
3287 Chitimacha Trail
Charenton, LA 70523

CROWLEY

Crowley and Ville Platte Alcohol and Drug Abuse Clinic
703 East 8 Street
Crowley, LA 70526

ELTON

Coushatta Health Department
2003 CC Bel Road
Elton, LA 70532

FRANKLINTON

Seven Acres Substance Abuse Center
23046 Yacc Road
Franklinton, LA 70438

GONZALES

Parish of Ascension Substance Abuse Center
1112 SE Ascension Complex Avenue
Gonzales, LA 70737

Power House Services, Inc.
715 West Worthey Road
Gonzales, LA 70737

GREENSBURG

Saint Helena Alcohol and Drug Abuse Clinic
102 North 2nd Street
Greensburg, LA 70441

HAMMOND

Hammond Alcohol and Drug Abuse Clinic
202 East Robert Street
Hammond, LA 70401

HARVEY

Family House/Louisiana
1125-B Inca Court
Harvey, LA 70058

HOUMA

Terrebonne Alcohol and Drug Abuse Clinic
521 Legion Avenue
Houma, LA 70364

Detox Center
1116 Church Street
Houma, LA 70364

JENNINGS

Jefferson Davis Chemical Health, Inc.
203 North Cutting Street
Jennings, LA 70546

KENNER

Kenner Substance Abuse Clinic
1919 Veterans Boulevard
Kenner, LA 70062

KINDER

Allen Parish Hospital
108 6th Avenue
Kinder, LA 70648

LAFAYETTE

Charter Cypress Behavioral Health Service
302 Dulles Drive
Lafayette, LA 70506

Gatehouse Foundation
206 South Magnolia Street
Lafayette, LA 70501

Lafayette Alcohol and Drug Abuse Clinic
400 Saint Julien Street
Suite 1
Lafayette, LA 70506

Saint Francis Foundation
1610 West University Street
Lafayette, LA 70506

Vermilion Hospital for Psychiatric and Addictive Medicine
2520 North University Avenue
Lafayette, LA 70507

LAKE CHARLES

Joseph R. Briscoe Alcohol and Drug Abuse Center
4012 Avenue H
Lake Charles, LA 70601

Lake Charles Substance Abuse Clinic, Inc.
711 North Prater Street
Lake Charles, LA 70601

Lake Charles Memorial Hospital Recovery Center
1701 Oak Park Boulevard
Lake Charles, LA 70601

LA PLACE

River Parishes Alcohol and Drug Abuse Clinic
421 West Airline Highway
Suite L
La Place, LA 70068

LEESVILLE

Vernon Alcohol and Drug Abuse
300 South 1st Street
Leesville, LA 71446

LULING

Saint Charles Parish Hospital Psychiatric Unit
1057 Paul Maillard Road
Luling, LA 70070

MAMOU

Savoy Medical Center New Horizons
120 Country Club Lane
Mamou, LA 70554

MANDEVILLE

Alcohol and Drug Treatment Unit
SE Hospital
Highway 190
Mandeville, LA 70470

Fontainebleau Treatment Center
Highway 190 West
Mandeville, LA 70448

Northlake Alcohol and Drug Abuse Clinic
101 Brookside Drive
Mandeville, LA 70448

MARKSVILLE

Hamilton House
103 South Main Street
Marksville, LA 71351

Tunica/Biloxi Indians of Louisiana Substance Abuse Prevention Program
Highway 1
Marksville, LA 71351

Washington Street Hope Center
106 South Washington Street
Marksville, LA 71351

MARRERO

West Bank Alcohol and Drug Abuse Clinic
5001 Westbank Expressway
Marrero, LA 70072

METAIRIE

Jefferson Substance Abuse Clinic
3101 West Napoleon Avenue
Suite 2000
Metairie, LA 70001

New Freedom, Inc.
401 Veteran's Memorial Boulevard
Suite 102
Metairie, LA 70005

MINDEN

Minden Mental Health and Substance Abuse Clinic
421 Meadowview Drive
Minden, LA 71055

MONROE

Four Runners Community Action Program Serenity House
2502 Georgia Street
Monroe, LA 71211

Monroe Alcohol and Drug Abuse Clinic
3208 Concordia Street
Monroe, LA 71201

Southern Oaks Addiction Recovery Center
4781 South Grand Street
Monroe, LA 71202

MORGAN CITY

Fairview Treatment Center
1101 Southeast Boulevard
Morgan City, LA 70380

Saint Mary Addictive Disorders Clinic
521 Roderick Street
Morgan City, LA 70380

NATCHITOCHES

Natchitoches Alcohol and Drug Abuse Clinic
212 Medical Drive
Natchitoches, LA 71457

NEW IBERIA

New Iberia Alcohol and Drug Abuse Clinic
611 West Admiral Doyle Drive
New Iberia, LA 70560

NEW ORLEANS

Basic of Louisiana, Inc.
1452 Broad Street
New Orleans, LA 70119

BHC East Lake Hospital
3600 Chestnut Street
New Orleans, LA 70115

Bridge House, Inc.
1160 Camp Street
New Orleans, LA 70130

CCYAD Foundation Youth Against Drugs
1528 Louisa Street
New Orleans, LA 70117

Covenant House New Orleans
611 North Rampart Street
New Orleans, LA 70112

Desire Narcotic Rehab Center, Inc.
3307 Desire Parkway
New Orleans, LA 70126

4116 Old Gentilly Road
New Orleans, LA 70126

Division of Addictive Disorders
LSU Medical School
1542 Tulane Avenue
New Orleans, LA 70112

Oscar Carter Rehabilitation Center
5500 North Johnson Street
New Orleans, LA 70117

DRD New Orleans Medical Clinic
530 South Galvez Street
New Orleans, LA 70119

Family Service of Greater New Orleans Community Care
2515 Canal Street
Suite 201
New Orleans, LA 70119

Foundation House/New Orleans
3942 Laurel Street
New Orleans, LA 70115

Grace House of New Orleans, Inc.
1401 Delachaise Street
New Orleans, LA 70115

Guillaume Center, Inc.
210 State Street
New Orleans, LA 70118

Methodist Psychiatric Pavilion
5610 Read Boulevard
New Orleans, LA 70127

Metropolitan Treatment Center, Inc.
3604 Tulane Avenue
New Orleans, LA 70119

New Orleans Substance Abuse Clinic
2025 Canal Street
Suite 300
New Orleans, LA 70112

Ochsner Addictive Behavior
1516 Jefferson Highway, Floor 4
New Orleans, LA 70121

Odyssey House Louisiana, Inc.
1125 North Tonti Street
New Orleans, LA 70119

Velocity Foundation, Inc.
4730 Washington Avenue
New Orleans, LA 70113

Veterans Administration Hospital
1601 Perdido Street
Unit 116A
New Orleans, LA 70146

NEW ROADS

Bonne Sante Chemical Health and Wellness Center
282-A Hospital Road
New Roads, LA 70760

OPELOUSAS

New Beginnings of Opelousas
1692 Linwood Loop
Opelousas, LA 70570

Opelousas Alcohol/Drug Abuse Clinic
532 North Court Street
Opelousas, LA 70570

PINEVILLE

Alexandria/Pineville Alcohol and Drug Abuse Clinic
401 Rainbow Drive
Pineville, LA 71361

Cenla Chemical Dependency Council
Bridge House/Phase II
401 Rainbow Drive
Pineville, LA 71361

Gateway Adolescent Unit
Pineville, LA 71360

Rainbow House Detox
Rainbow Drive
Pineville, LA 71361

Red River Treatment Center Central Louisiana State Hospital
Unit 6-D
Pineville, LA 71360

PORT ALLEN

People Rehabilitation and Recovery Services Corporation
710 Louisiana Avenue
Port Allen, LA 70767

RAYNE

American Legion Hospital Pauline Faulk Center
301 South Chevis Street
Rayne, LA 70578

RAYVILLE

Palmetto Addiction Recovery Center
86 Palmetto Road
Rayville, LA 71269

RUSTON

Louisiana Tech University Teen Institute
Ruston, LA 71272

Professional Counseling Services of Ruston
101 Reynolds Drive
Ruston, LA 71270

Ruston Alcohol and Drug Abuse Clinic
206 Reynolds Drive
Suite B-3
Ruston, LA 71270

SCHRIEVER

Assisi Bridge House
600 Bull Run Road
Schriever, LA 70395

SCOTT

Opportunities, Inc.
808 Pitt Road
Scott, LA 70583

SHREVEPORT

Buckhalter Recovery Center
527 Crockett Street
Shreveport, LA 71101

Caddo and Bossier Center
6220 Greenwood Road
Shreveport, LA 71119

Center for Families, Inc. Center for Addictive Disorders
864 Olive Street
Shreveport, LA 71104

Council on Alcohol/Drug Abuse of NW Louisiana
2000 Fairfield Avenue
Shreveport, LA 71104

The Adolescent Center
431 Jordan Street
Shreveport, LA 71101

CPC Brentwood Hospital Chemical Dependency Unit
1800 Irving Place
Shreveport, LA 71101

DDTP
510 East Stoner Avenue
Shreveport, LA 71101-4295

Doctors Hospital Addictive Disease Unit
1130 Louisiana Avenue
Shreveport, LA 71101

First Step Services, Inc.
2004 Creswell Street
Shreveport, LA 71104

Northwest Regional Alcohol and Drug Abuse Clinic
6244 Greenwood Road
Shreveport, LA 71119

Pines Treatment Center
6240 Greenwood Road
Shreveport, LA 71119

Sharing Through Examples of Personal Sobriety (STEPS)
525 Crockett Street
Shreveport, LA 71101

Volunteers of America Madre Program
345 Jordan Street
Shreveport, LA 71101

SLIDELL

Slidell Alcohol and Drug Abuse Clinic
2335 Carey Street
Slidell, LA 70458

TALLULAH

Delta Community Action Association Delta Recovery Center
404 East Craig Street
Tallulah, LA 71282

THIBODAUX

South Louisiana Rehabilitation Center Power House
614 Jackson Street
Thibodaux, LA 70301

Thibodaux Alcohol and Drug Abuse Clinic
303 Hickory Street
Thibodaux, LA 70301

WINNFIELD

Winnfield Alcoholism and Drug Abuse Clinic
308 Main Street, Suite 208-B
Winnfield, LA 71483

WINNSBORO

Northeast Louisiana Substance Abuse, Inc.
210 Main Street
Winnsboro, LA 71295

MAINE

ALBION

Health Reach Network
New Directions/Albion
School Street
Albion, ME 04910-1568

ALFRED

York County Shelters Inc
Shaker Hill Road
Alfred, ME 04002

ASHLAND

Aroostook Mental Health Center Outpatient Services
Walker Street
Ashland, ME 04732

AUBURN

Catholic Charities Maine Saint Francis House
88 3rd Street
Auburn, ME 04210

Community Concepts, Inc.
2 Court Street
Auburn, ME 04210

Family Intervention Services
233 Main Street
Auburn, ME 04210

Hayden, William, LSAC
81 Main Street, Box 3
Auburn, ME 04210

AUGUSTA

Bachand, Robert P.
33 Water Street
Augusta, ME 04330

Crisis and Counseling Center
99 Western Avenue
Augusta, ME 04330

Health Reach Network
Hearthside
Belgrade Road
Route 27
Augusta, ME 04330

New Directions
1 Weston Court
Augusta, ME 04330

Kassal, Jeannette, LCPC, LADC
74 Winthrop Street
Augusta, ME 04330

Maine General Medical Center Spruce Street Residence
9 Spruce Street
Augusta, ME 04330

Veterans' Affairs Medical Center Chemical Dependence Recovery Program
Route 17 East, 116-A2
Augusta, ME 04330

Wellness Health Association, Inc.
283 Water Street
Augusta, ME 04330

BANGOR

ABBAK Counseling Services
Bangor, ME 04402

Acadia Healthcare, Inc.
268 Stillwater Avenue
Bangor, ME 04401

Alternative Counseling Services
27 State Street
Suite 20-24
Bangor, ME 04401

BMHI/Acadia Recovery Community Substance Abuse Services
Bangor, ME 04401

Columbia Psychology Associate
82 Columbia Street
Bangor, ME 04401-6357

Community Health and Counseling Services Substance Abuse Services
900 Hammond Street, Suite 915
Bangor, ME 04401

Dunning, Frances
13-A North High Street
Bangor, ME 04401

Levenson, Laura
73 Pine Street
Bangor, ME 04401

Northeast Care Foundation
268 Center Street
Bangor, ME 04401

Outpatient Chemical Dependency Agency
185 Harlow Street
Bangor, ME 04401

Project Atrium, Inc. Janus House
51 Forth Street
Bangor, ME 04401

Sign of Hope Counseling Association
115 Franklin Street, Suite GA
Bangor, ME 04401

Tingley, Charles
248 Center Street
Bangor, ME 04401

Veterans Administration Bangor Clinic
304 Hancock Street, Suite 3-B
Bangor, ME 04401

Wabanaki Mental Health Association
277 State Street, Suite 3-B
Bangor, ME 04401

Wellspring, Inc.
Men's Program
98 Cumberland Street
Bangor, ME 04401

Outpatient Services
136 Union Street
Bangor, ME 04401

Women's Program
319 State Street
Bangor, ME 04401

BAR MILLS

Drug Rehabilitation, Inc. Day One Residence
James C Harrod Center
Bar Mills, ME 04004

BATH

Midcoast Hospital Addiction Resource Center
1356 Washington Street
Bath, ME 04530

BELFAST

Kelley, Karen, LSAC
143 High Street
Belfast, ME 04915

Waldo County General Hospital Counseling Service
118 Northpoint Avenue
Belfast, ME 04915

Westbay Counseling Services, Inc.
22 Spring Street
Belfast, ME 04915

BINGHAM

Health Reach Network
New Directions/Bingham
Upper Main Street
Bingham, ME 04920

BOOTHBAY HARBOR

Helmstadter, John
54 Oak Street
Boothbay Harbor, ME 04538

BRIDGTON

Danley, Colleen
Route 302
Roosevelt Trail Professional Building
Bridgton, ME 04009

Lake Region Counseling Center
Chase Street
Bridgton, ME 04009

Tri-County Mental Health Services Substance Abuse Services
41 North High Street
Bridgton, ME 04009

BRUNSWICK

Bellville Counseling Association
8 Stanwood Street
Brunswick, ME 04011

Connor, Pat
153-B Park Row
Brunswick, ME 04011-2005

Counseling and Assistance Center Naval Air Station
Building 12
Brunswick, ME 04011

BUCKSPORT

Lawrence, Suzanne, BS LSAC
505 Main Street
Bucksport, ME 04416

CALAIS

Calais Regional Hospital Substance Abuse Treatment Facility
50 Franklin Street
Calais, ME 04619

CAMDEN

Midcoast Substance Abuse Council
89 Elm Street
Camden, ME 04843

New Dawn Associates
88 Elm Street
Camden, ME 04843

CARIBOU

Aroostook Mental Health Center Outpatient Substance Abuse Services
Downtown Mall
Saint Peter Building
Caribou, ME 04736

COOPERS MILLS

Health Reach Network
New Directions/Coopers Mill
Main Street
Coopers Mills, ME 04341

DAMARISCOTTA

Addiction Resource Center of Lincoln County
Rural Route 2, Box 3-A
Damariscotta, ME 04543

DANFORTH

Aroostook Mental Health Center East Grand Rural Health Center
Houlton Road
Danforth, ME 04424

DOVER FOXCROFT

Mayo Regional Hospital Substance Abuse Services
75 West Main Street
Dover Foxcroft, ME 04426

EAST MILLINOCKET

Denney, Elizabeth
103 Main Street
East Millinocket, ME 04430

ELLSWORTH

Open Door Recovery Center
10 High Street
Ellsworth, ME 04605

Substance Abuse Services of Ellsworth
53 Church Street
Ellsworth, ME 04605

FARMINGTON

Evergreen Behavioral Services Mount Blue Health Center
Rural Route 4
Farmington, ME 04938

Health Reach Network
New Directions/Farmington
Farmington, ME 04938-1568

Tri-County SACS
28 High Street
Farmington, ME 04938

FORT KENT

Aroostook Mental Health Center Outpatient Substance Abuse Services
96 Market Street
Fort Kent, ME 04743

FREEPORT

Thacher, Sarah A.
102 South Freeport Road
Freeport, ME 04032

GORHAM

Southwestern Maine Associates PA
510 Main Street
Gorham, ME 04038

HALLOWELL

True, Robert A., LCSW
402 Water Street
Hallowell, ME 04347

Your Choice, Inc.
24 Wilder Street
Hallowell, ME 04347

HARTLAND

Health Reach Network
New Directions/Scott Webb Health Center
1 Great Moose Drive
Hartland, ME 04943

HINCKLEY

George Walter Associates
Route 201
Hinckley, ME 04944

HOULTON

Aroostook Mental Health Center Outpatient Substance Abuse Services
11 Riverside Street
Houlton, ME 04730

Paul, William
4 Charles Street
Houlton, ME 04730

KENNEBUNK

Ristine, Susannah
7 Blue Wave Professional Center
Kennebunk, ME 04043

KEZAR FALLS

Counseling Services, Inc. Sacopee Valley Unit
Kezar Falls, ME 04047

KINGFIELD

Health Reach Network
New Directions/Kingfield
2 Stanley Avenue
Kingfield, ME 04947

LEEDS

Health Reach Network
New Directions/Leeds
Church Hill Road
Leeds, ME 04263

LEWISTON

Catholic Charities Maine Fellowship House
95 Blake Street
Lewiston, ME 04240

Central Maine Counseling Services, Inc.
55 Lisbon Street
Lewiston, ME 04240

Iannotti, Dominick J., Addiction and Behavior Counseling
145 Lisbon Street Suite 208
Lewiston, ME 04240

Facing Change PA
4 Park Street Suite 1
Lewiston, ME 04240

Saint Mary's Regional Medical Center Chemical Dependency Service
100 Campus Avenue
Lewiston, ME 04243

Transitions Counseling, Inc.
105 Middle Street
Lewiston, ME 04240

Tri-County Substance Abuse Counseling Services
1155 Lisbon Street
Lewiston, ME 04240

LIMESTONE

Aroostook Mental Health Center Residential Treatment Facility
Route 1A
Limestone, ME 04750

LINCOLN

Riverside Community Center
43 Fleming Street
Lincoln, ME 04457

LISBON FALLS

Right Direction
679 Lisbon Road
Lisbon Falls, ME 04252

LIVERMORE FALLS

Evergreen Behavioral Services Mount Blue Health Center
80 Main Street
Livermore Falls, ME 04254

Health Reach Network
New Directions/Livermore Falls
80 Main Street
Livermore Falls, ME 04254

LUBEC

Regional Medical Center
Eastport Health Care
Substance Abuse Services
South Lubec Road
Lubec, ME 04652

MACHIAS

Cornerstone
2 Lower Main Street
Machias, ME 04654

MADAWASKA

Aroostook Mental Health Center Outpatient Substance Abuse Services
66 Fox Street
Madawaska, ME 04756

MADISON

Health Reach Network
New Directions/Madison
South Main Street
Madison, ME 04950

MEXICO

New England Counseling Services, Inc.
3 Brown Street
Mexico, ME 04257

Saint Mary's Counseling Center
6 Porters Bridge Road
Mexico, ME 04257

NEWPORT

Northeast Occupational Exchange
18 Main Street
Newport, ME 04953

OLD ORCHARD BEACH

Milestone Extended Care
28 Portland Avenue
Old Orchard Beach, ME 04064

PERRY

Pleasant Point Health Center Substance Abuse Program
Pleasant Point Indian Health Center
Perry, ME 04667

PITTSFIELD

Acadia Recovery Community Sebasticook Valley Hospital
169 South Street
Pittsfield, ME 04967

PORTLAND

Access Team
82 Elm Street
Portland, ME 04101

Catholic Charities of Maine
Counseling Services
562 Congress Street
Portland, ME 04101

Evodia House
79 Allen Avenue
Portland, ME 04103

Chemical Dependency Recovery Program (CDRP)
980 Forest Avenue, Suite 204
Portland, ME 04103

Coose, Chris
Top of the Hill Counseling
87 Saint Lawrence Street
Portland, ME 04101

Crossroads for Women
66 Pearl Street
Portland, ME 04101

Day One Outpatient Office
23 Ocean Avenue
Portland, ME 04103

Family Institute of Maine
38 Deering Street
Portland, ME 04101

Food Addiction and Chemical Dependency Consultant
219 Vaughn Street, Apartment 5
Portland, ME 04102

Hood, Betsy, PA
95 High Street
Portland, ME 04101

Ingraham, Inc.
Bridge Program
54 Maple Street
Portland, ME 04101

Mainstay Program
165 Cumberland Avenue
Portland, ME 04101

Randall Place
12 Randall Street
Portland, ME 04103

McKenney Counseling Service
175 Lancaster Street
Suite 714-F
Portland, ME 04101

Milestone Foundation
65 India Street
Portland, ME 04101

Portland Public Health
389 Congress Street
Portland, ME 04101

Recovery Center at Mercy Hospital
144 State Street
Portland, ME 04101

Serenity House
30 Mellen Street
Portland, ME 04101

Transitions Counseling Associates
222 Saint Johns Street
Portland, ME 04102

491 Stevens Avenue
Portland, ME 04103

158 Danforth Street
Portland, ME 04101

Wellness Health Association Inc
650 Brighton Avenue
Portland, ME 04102

PRESQUE ISLE

Aroostook Mental Health Center Outpatient Substance Abuse Services
1 Edgemont Drive
Presque Isle, ME 04769

PRINCETON

Indian Township Health Center Human Services Division
Passamaquoddy Tribe
Peter Dana Point
Indian Township
Princeton, ME 04668

RICHMOND

Health Reach Network
New Directions/Richmond
24 Gardiner Street
Richmond, ME 04357

ROCKLAND

Alternate Choices
81 Park Street
Rockland, ME 04841

Barnett, Amy
336 Main Street
Rockland, ME 04841

Midcoast Mental Health Center
12 Union Street
Rockland, ME 04841

Penobscot Bay Medical Center Choice Skyward
22 White Street
Rockland, ME 04841-2931

ROCKPORT

Pyschiatric and Addiction Recovery Center
Pen Bay Medical Center
6 Glen Cove Drive
Rockport, ME 04856-4240

RUMFORD

Rumford Community Hospital Substance Abuse Services
420 Franklin Street
Rumford, ME 04276

SACO

Counseling Services, Inc.
333 Lincoln Street
Saco, ME 04072

Dayowl Counseling
23 Water Street
Saco, ME 04072

Transitions Counseling Associates
5 Horton Avenue
Saco, ME 04005

SANFORD

Counseling Services, Inc.
1 High Street
Sanford, ME 04073

SCARBOROUGH

Jackson Brook Institute (JBI)
600 Roundwood Drive Box 10
Scarborough, ME 04074

SEARSPORT

Searsport Counseling Associates
7 Knox Brothers Avenue
Searsport, ME 04974

SKOWHEGAN

Corson, Donna Dearborn
Oxbow Road
Skowhegan, ME 04976

Health Reach Network
New Directions/Skowhegan
251 North Avenue
Skowhegan, ME 04976

Youth and Family Services, Inc. Substance Abuse Program
Route 201
Skowhegan, ME 04976

SOUTH PARIS

Community Concepts, Inc. Supported Journey
Oxford Hills High School
250 Main Street
South Paris, ME 04281

Tri-County Mental Health Services
Oxford Hills Unit
28 East Main Street
South Paris, ME 04281

SOUTH PORTLAND

Day One
Maine Youth Center
675 West Brook Street
South Portland, ME 04106

Discovery House
400 Western Avenue
South Portland, ME 04106

Rice, Ted, Counseling and Consultation Services
182 Highland Avenue
South Portland, ME 04106

SOUTHWEST HARBOR

Acadia Family Center
Clark Point Road
Southwest Harbor, ME 04679

STRONG

Health Reach Network
New Directions/Strong
Strong, ME 04983

THORNDIKE

Steppingstone
Rural Route 1
Thorndike, ME 04986

VAN BUREN

Aroostook Mental Health Center Outpatient Alcoholism Services
2 Main Street
Van Buren, ME 04785

WALDOBORO

Alternate Choices Counseling Services
32 Friendship Street
Waldoboro, ME 04572

WATERFORD

Kimball, Elaine
Brownhill Road
Waterford, ME 04088

WATERVILLE

Health Reach Network
New Directions
8 Highwood Street
Waterville, ME 04901

WINDHAM

Crossroads for Women
114 Main Street
Windham, ME 04062

WINSLOW

Discovery House of Central Maine
13 Bay Street
Winslow, ME 04901

YARMOUTH

World Tree Psychotherapy
261 Main Street
Yarmouth, ME 04096

YORK

Family Resource Services
15 Hospital Drive
York, ME 03909

MARYLAND

ABERDEEN

Ashley, Inc. Outpatient Program
10 Howard Street
Aberdeen, MD 21001

Abingdon

Emmorton Psych
3105 Emmorton Road
Abingdon, MD 21009

ANNAPOLIS

Addictions Services Methadone Program
2200 Somerville Road
Annapolis, MD 21401

Alcohol and Drug Programs Management, Inc.
107 Ridgely Avenue
Suite 13B
Annapolis, MD 21401

Cornerstone Care
2525 Riva Road Suite C
Annapolis, MD 21401

Pathfinder Health Services
2448 Holly Avenue Suite 200
Annapolis, MD 21401

Pathways
2620 Riva Road
Annapolis, MD 21401

Samaritan House
2610 Greenbrier Lane
Annapolis, MD 21401

Sheppard Pratt at Annapolis
147 Old Solomon Island Road
Suite 206
Annapolis, MD 21401

BALTIMORE

Adapt Cares/Primary
3101 Towanda Avenue
Baltimore, MD 21215

Addict Referral and Counseling Center, Inc. (ARCC)
21 West 25 Street
Baltimore, MD 21218

Alcohol and Drug Abuse Program
630 West Fayette Street
Room 1-106
Baltimore, MD 21202

All Addictions Treatment Center
3655-A Old Court Road, Suite 12
Baltimore, MD 21208

Alliance, Inc. SPMI/SA Day Program
9201 Philadelphia Road
Baltimore, MD 21237

Alternatives to Dependencies
518 Eastern Boulevard
Baltimore, MD 21212

40 West Chesapeake Avenue
Suite 205
Baltimore, MD 21204

Atlantic Coast Evaluation and Recovery Services
98 North Broadway Street
Suite 205
Baltimore, MD 21231

Aware
6229 North Charles Street
Building A
Baltimore, MD 21212

Awele Treatment and Rehab Clinic
2300 North Calvert Street
Suite 102
Baltimore, MD 21218

Baltimore American Indian Center, Inc. Substance Abuse and Prevention Program
113 South Broadway
Baltimore, MD 21231

Baltimore Behavioral Health, Inc.
200 South Arlington Avenue
Baltimore, MD 21223

Baltimore City Health Department
Daybreak Rehabilitation Program
Gateway Adolescent Program
2490 Giles Road
Baltimore, MD 21225

Baltimore County Office of Substance Abuse
Comprehensive Treatment Program
401 Washington Avenue
Suite 300
Baltimore, MD 21204

Baltimore County Outpatient Cocaine Abuse Treatment Program
208 Washington Avenue
Baltimore, MD 21204

Baltimore Health System/Next Passage Drug Free Substance Abuse Counseling Services
2901 Druid Park Drive
Suite A-103
Baltimore, MD 21215

Baltimore Recovery Center Continuing Care/Aftercare
100 South Arlington Street
Baltimore, MD 21201

Baltimore Rescue Mission, Inc.
4 North Central Avenue
Baltimore, MD 21202

Bay Life Counseling Services Franklin Square at White Marsh
8114 Sandpiper Circle, Suite 116
Baltimore, MD 21236

Bright Hope House, Inc.
1611 Baker Street
Baltimore, MD 21217

Charles H Hickey Jr School Adolescent Drug Treatment Unit
2400 Cub Hill Road
Baltimore, MD 21234

Chesapeake Counseling
825 Eastern Boulevard
Baltimore, MD 21221

Comprehensive Psycho/Social Services
1401 Reisterstown Road
Suite L1
Baltimore, MD 21208

Counseling Resource Associates
6423 Frederick Avenue, Suite 3
Baltimore, MD 21228

Crossroads Centers
2 West Madison Street
Baltimore, MD 21201

Damascus House
4203 Ritchie Highway
Baltimore, MD 21225

Deaf Substance Abuse Treatment Services Family Services Foundation, Inc.
2310 North Charles Street
Baltimore, MD 21218

Dependency Recovery
26 West Pennsylvania Avenue
Baltimore, MD 21204

Echo House Multi Service Center Seekers After a New Direction (SAND)
1705 West Fayette Street
Baltimore, MD 21223

EPOCH Counseling Center
Dundalk
1107 North Point Boulevard
East Point Office Park, Suite 205
Baltimore, MD 21224

Counseling Center/East
621 East Stemmers Run Road
Baltimore, MD 21221

Counseling Center
3902 Annapolis Road
Baltimore, MD 21228

Evelyn Jordan Drug Treatment Program Walter P Carter Center
630 West Fayette Street
Room 1-135
Baltimore, MD 21201

Family Service Foundation, Inc. Substance Abuse Program
4806 Seton Drive, Suite 204
Baltimore, MD 21215

Fayette House
1319 South Fulton
Baltimore, MD 21223

First Step Inc.
8303 Liberty Road
Baltimore, MD 21244

Glass Counseling Center Intensive Outpatient Program
405 Frederick Road
Catonsville Professional Building
Baltimore, MD 21228

Glass Substance Abuse Program, Inc.
Methadone Program
821 North Eutaw Street
Suite 101
Baltimore, MD 21201

Glenwood Life Drug Abuse Treatment Program
516 Glenwood Avenue
Baltimore, MD 21212

Greater Baltimore Medical Center
Weinberg Community Health Center
1200 East Fayette Street
Baltimore, MD 21204

Harbel Prevention and Recovery Center
5807 Harford Road
Baltimore, MD 21214

Harbor Clinical Services
1055 Taylor Avenue, Suite 300
Baltimore, MD 21214

Harbour Center
924 East Baltimore Street
Baltimore, MD 21202-4739

Health Care for the Homeless, Inc.
111 Park Avenue
Baltimore, MD 21201

Help and Recovery Today, Inc. (HART, Inc.)
8200 Harford Road
Suite 200
Baltimore, MD 21234

Helping Up Mission
1029 East Baltimore Street
Baltimore, MD 21202

Institutes for Behavior Resources, Inc. (IBR) Mobile Health Services/Primary
2457 Maryland Avenue
Baltimore, MD 21224

JAI Medical Center
5010 York Road
Baltimore, MD 21212

Jewish Addiction Services Drug Abuse Services
1515 Reisterstown Road
Suite 300
Baltimore, MD 21208

Johns Hopkins Bayview Medical Center
Behavioral Pharmaceutical Research Unit
5510 Nathan Shock Drive
Baltimore, MD 21224

Center for Addiction and Pregnancy
Community Psychiatry Program
4940 Eastern Avenue
M F Lord Building, Suite D-2 East
Baltimore, MD 21224

Johns Hopkins Hospital
Comprehensive Women's Center
Outpatient Program
Program for Alcohol and Other Drug Dependency
Stop Program
911 North Broadway
Baltimore, MD 21205

Judith P. Ritchey Youth Services Center
8840 Belair Road
Baltimore, MD 21236

Liberty Medical Center Substance Abuse Program Overcome
3101 Towanda Avenue
Baltimore, MD 21215

Loyola College Alcohol and Drug Education and Treatment Program
4501 North Charles Street
Charleston 02-B
Baltimore, MD 21210

Man Alive Research, Inc.
2100 North Charles Street
Baltimore, MD 21218

Methadone for Business Achievers
821 North Eutaw Street Suite 201
Baltimore, MD 21201

Mountain Manor Treatment Center
Outpatient/Residential
3800 Frederick Avenue
Baltimore, MD 21229

New Outlook
821 North Eutaw Street
Suite 201
Baltimore, MD 21201

Nilsson House
5665 Purdue Avenue
Baltimore, MD 21239

Operation Recovery
301 Saint Paul Place Suite 812
Baltimore, MD 21202

Peoples Community Health Center
Addiction Program
3028 Greenmount Avenue
Baltimore, MD 21218

Powell Recovery Center
14 South Broadway
Baltimore, MD 21231

Quarterway Outpatient Clinic
730 Ashburton Street
Baltimore, MD 21216

Raphael, Ralph D., Ph.D., PA
21 West Road Suite 150
Baltimore, MD 21204

Re-Entry Aftercare Center
319 West Monument Street
Baltimore, MD 21201

2100 Guilford Avenue
Baltimore, MD 21218

428 East Preston Street
Baltimore, MD 21202

Reflective Treatment Center
301 North Gay Street
Baltimore, MD 21202

707 Constitution Street
Baltimore, MD 21202

Residential Substance Abuse Treatment for Women
301 North Calverton Road
Baltimore, MD 21223

Resource Group Counseling and Education Center
7801 York Road, Suite 215
Baltimore, MD 21204

S and S Counseling Service
429 Eastern Boulevard
Baltimore, MD 21221

Safe House
7 West Randall Street
Baltimore, MD 21230

Saint Agnes Hospital Mental Health Clinic
900 South Caton Avenue
Baltimore, MD

Sinai Hospital Addiction Recovery Program
2401 West Belvedere Avenue
Baltimore, MD 21215

South Baltimore Family Health Center, Inc.
631 Cherry Hill Road
Baltimore, MD 21225

Total Health Care, Inc. Substance Abuse Services
1800 North Charles Street
8th Floor
Baltimore, MD 21217

Towson Addiction Center
22 West Pennsylvania Avenue
Suite 402
Baltimore, MD 21204

Treatment Resources for Youth (TRY)
2517 North Charles Street
Baltimore, MD 21218

Tuerk House Alcohol and Drug Program
730 Ashburton Street
Baltimore, MD 21216

Turning Corners, Inc.
5200 Moravia Road
Baltimore, MD 21206

Universal Counseling Services, Inc.
101 West Read Street
Suite 422
Baltimore, MD 21201

University of Maryland
Federal Aftercare Clinic
Methadone Treatment Program
Needle Exchange Program
630 West Fayette Street
First Floor
Baltimore, MD 21201

Harambee Treatment Center
3939 Reistertown Road
Baltimore, MD 21215

Valley House
28 South Broadway
Baltimore, MD 21231

Veterans' Affairs Medical Center Substance Abuse Treatment Unit
10 North Green Street
Baltimore, MD 21201

Weisman/Kaplan Houses
2521–2523 Maryland Avenue
Baltimore, MD 21218

William Donald Schaefer House
907 Druid Lake Drive
Baltimore, MD 21217

BARSTOW

Calvert Substance Abuse Services
315 Stafford Road
Barstow, MD 20610

DWI Services, Inc. Calvert County Treatment Facility
315 Stafford Road
Barstow, MD 20610-0730

BEL AIR

Harford County Adolescent Substance Abuse Program
715 Shamrock Road
Bel Air-Lee Professional Center
Bel Air, MD 21014

Mann House, Inc.
14 Williams Street
Bel Air, MD 21014

TRW Associates
728 Bel Air Road
Suite 137
Bel Air, MD 21014

BEL ALTON

Jude House, Inc.
9505 Crain Highway South
Bel Alton, MD 20611

BOWIE

Counseling Services, Inc.
2905 Mitchellville Road
Bowie, MD 20716

BURTONSVILLE

New Horizon Health Services
4140 Sandy Spring Road
Burtonsville, MD 20866

CALIFORNIA

Walden Counseling Center
Saint Andrew's Church Road
California, MD 20619

CAMBRIDGE

Dorchester County Health Department
Addictions Program
310 Gay Street
Cambridge, MD 21613

CAPITOL HEIGHTS

Renaissance Treatment Center
601 60th Place
Capitol Heights, MD 20743

CATONSVILLE

EPOCH Counseling Center/West
800 Ingleside Avenue
Catonsville, MD 21228

CENTREVILLE

Queen Anne's County Health Department Alcohol and Drug Services
205 North Liberty Street
Centreville, MD 21617

CHELTENHAM

Cheltenham Young Women's Residential Treatment Program
11001 Frank Tippett Road
Cheltenham, MD 20623

CHESAPEAKE BEACH

Calvert County Substance Abuse Program
3819 Harbor Road
Chesapeake Beach, MD 20732

CHESTERTOWN

A. F. Whitsitt Center/ Quarterway
Sheeler Road
Chestertown, MD 21620

Publick House
114 A South Lynchburg Street
Chestertown, MD 21620

CHEVERLY

Prince George's County Health Deptartment Addictions/ Northern Region
3003 Hospital Drive
Cheverly, MD 20785

CLINTON

Counseling Services Alternatives, Inc.
7900 Old Branch Avenue
Suite 202
Clinton, MD 20735

Prince George's County Health Dept. Addictions/Southern Region
9314 Piscataway Road
Clinton, MD 20735

COCKEYSVILLE

Community Counseling and Resource Center Alcohol and Drug Treatment
10400 Ridgland Road
Cockeysville, MD 21030

COLLEGE PARK

Ethos Foundation
7309 Baltimore Avenue
Suite 217
College Park, MD 20740

Insight, Inc.
4907 Niagra Road
College Park, MD 20740

Recovery Network
6201 Greenbelt Road, Suite U-18
College Park, MD 20740

University of Maryland Health Center Alcohol and Drug Treatment
University Health Center
Suite 2106
College Park, MD 20742

COLUMBIA

Columbia Addiction Center
10774 Hickory Ridge Road
Hawthorne Industrial Park
Columbia, MD 21044

Howard County Addictions Services Center
7101 Riverwood Drive
Columbia, MD 21046

Pathfinder Health Services Substance Abuse Services
10840 Little Patuxent Parkway
Suite 203
Columbia, MD 21044

CROFTON

DWI Assessment and Counseling
1520 Birdwood Court
Crofton, MD 21114

New Way Clinic
2135 Espey Court, Suite 2
Crofton, MD 21114

CROWNSVILLE

Chrysalis House
1570 Crownsville Road
Crownsville, MD 21032

Hope House
26 Marbury Drive
Crownsville, MD 21032

Second Genesis, Inc.
107 Circle Drive
Phillips Building
Crownsville, MD 21032

CUMBERLAND

Allegany County Addictions Services
Alcohol and Drug Outpatient
12500 Willowbrook Road SE
Cumberland MD 21502

Joseph S. Massie Unit
Country Club Road
Thomas B. Finan Center Cottage Four
Cumberland, MD 21502

Lois E. Jackson Unit
10102 SE Country Club Road
Thomas B. Finan Center Cottage Three
Cumberland, MD 21502

Family Therapy Services
621 Crest Drive
Cumberland, MD 21502

DELMAR

Delmarva Counseling Center
28 East State Street
Delmar, MD 21875

DENTON

Caroline County Health Department Caroline Counseling Center
104 Franklin Street
Denton, MD 21629

DERWOOD

Metro Alcohol and Drug Abuse Services, Inc.
15719 Crabbs Branch Way
Derwood, MD 20855

DUNDALK

EPOCH Counseling Center/ Southeast
7701 Dunman Way
Dundalk, MD 21222

EAST NEW MARKET

Charter Behavioral Health Systems
3680 Warwick Road
East New Market, MD 21631

EASTON

Shore Behavioral Health Services
29515 Canvas Back Drive, Suite A
Easton, MD 21601

Talbot County Addictions Program
100 South Hanson Street
Easton, MD 21601

ELDERSBURG

Metwork Health Service, Inc.
2120-A Liberty Road
Eldersburg, MD 21784

ELKTON

Cecil County Health Department Alcohol and Drug Center
401 Bow Street
Elkton, MD 21921

Haven House, Inc.
Outpatient Unit
111 East Main Street, Suite A
Elkton, MD 21921

1195 Augustine Herman Highway
Elkton, MD 21921

ELLICOTT CITY

Charter Behavioral Health System Warwick Manor at Columbia
4785 Dorsey Hall Road, Suite 118
Ellicott City, MD 21042

Counseling Resources
8388 Court Avenue Wall Building
Ellicott City, MD 21043

Jael Health Services
10176 Baltimore National Pike
Suite 115
Ellicott City, MD 21042

Montgomery General Hospital Outpatient Addiction Treatment Services
2850 North Ridge Road, Suite 207
Ellicott City, MD 21043

Taylor Manor Hospital Dual Diagnosis Program
4100 College Avenue
Ellicott City, MD 21041

EMMITSBURG

Mountain Manor Safe Harbor Project Potomac Health Services for Pregnant Clients
Route 15 and Keysville Road
Emmitsburg, MD 21727

Mountain Manor Treatment Center Emmitsburg Rehabilitation/Outpatient
Route 15
Emmitsburg, MD 21727

FORESTVILLE

Children and Parent Programs
5408 Silver Hill Road, 5th Floor
Forestville, MD 20747

Comprehensive Alcohol/Drug Counseling Service, Inc.
2810 Walters Lane
Room 10
Forestville, MD 20747

Prince Georges County Health Department Addictions/ Central Region
5408 Silver Hill Road, First Floor
Forestville, MD 20747

FORT GEORGE G. MEADE

Kimbroush Ambulatory Care Center Substance Abuse Rehab Clinic
85th Medical Battalion Avenue
Fort George G Meade, MD 20755

FORT HOWARD

Veterans' Affairs Maryland Health Care System
9600 North Point Road
Building 51
Fort Howard, MD 21052

FREDERICK

Allied Counseling Group Drug and Alcohol Treatment
306 West Patrick Street
Frederick, MD 21701

Catoctin Counseling Center
250 West Patrick Street
Frederick, MD 21701

Crossroad Center
176 Thomas Johnson Drive
Suite 104
Frederick, MD 21702

Frederick County Substance Abuse Services
300 B Scholls Lane
Frederick, MD 21702

Gale House, Inc.
Gale House
336 North Market Street
Frederick, MD 21701

Olson House
608 East Patrick Street
Frederick, MD 21701

Guidelines Counseling Program, Inc.
309 West Patrick Street
Frederick, MD 21701

Mountain Manor Treatment Center Outpatient Services
137 North Market Street
Suite 2A
Frederick, MD 21701

FROSTBURG

Frostburg State University
Substance Abuse Facts and Education Program (SAFE)
Compton 017
Frostburg, MD 21532

GAITHERSBURG

Circle Treatment Center
424 North Frederick Avenue
Suite 8A
Gaithersburg, MD 20877

Ethos Foundation
19638 Clubhouse Road
Suite 215
Gaithersburg, MD 20878

Guide Program, Inc.
Adolescent Treatment Program
1 West Deer Park Drive
Room 101
Gaithersburg, MD 20877

Adult Program
1 West Deer Park Drive
Room 401
Gaithersburg, MD 20877

GERMANTOWN

Alcohol/Drug Education Counseling Center
20120 Timber Oak Lane
Germantown, MD 20874

GLEN BURNIE

Alcohol and Drug Programs Management, Inc.
7495 Baltimore-Annapolis Boulevard
Glen Burnie, MD 21061

Anne Arundel County Health Department
Addictions Services/Adolescent and Family
407 South Crain Highway
2nd Floor
Glen Burnie, MD 21060

Addictions Services/Drug Intervention
7495 Baltimore Annapolis Boulevard
Suite 200
Glen Burnie, MD 21060

Ejal Health Services, Inc.
550 Crain Highway Unit 8
Glen Burnie, MD 21061

Recovery Resources Group, Inc.
2-B Crain Highway SW
Glen Burnie, MD 21061

Transformation
407 South Crain Highway
Suite 101
Glen Burnie, MD 21061

We Care Arundel Health Service, Inc.
13 Aquahart Road, Suite A
Glen Burnie, MD 21061

GRANTSVILLE

Meadow Mountain Drug Treatment Program
234 Recovery Road
Grantsville, MD 21536

HAGERSTOWN

Behavioral Health Services of Washington County Health Systems
1198 Kenly Avenue Suite 101
Hagerstown, MD 21740

Catoctin Counseling Center
162 West Washington Street
Hagerstown, MD 21740

Functional Social Work, Inc. Drug and Alcohol Treatment Unit
10401 Sharpsburg Pike
Hagerstown, MD 21740

Jail Substance Abuse Program (JSAP) Aftercare
13126 Pennsylvania Avenue
Hagerstown, MD 21742

W House, Inc.
37 East Antietam Street
Hagerstown, MD 21740

Washington County Health Department
Comprehensive Addiction Program
1302 Pennsylvania Avenue
Hagerstown, MD 21742

Jail Substance Abuse Program
500 Western Maryland Parkway
Hagerstown, MD 21740

Intensive Substance Abuse Program
13126 Pennsylvania Avenue
Hagerstown, MD 21742

Wells House Residential Facility
324 North Locust Street
Hagerstown, MD 21740

HAVRE DE GRACE

Ashley, Inc. Quarterway Unit
800 Tydings Lane
Havre de Grace, MD 21078

SAFE Associates Inc
420 South Stokes Street
Havre De Grace, MD 21078

HUNTINGTOWN

Courage to Change Counseling Program
4020 Hidden Hill Drive
Huntingtown, MD 20639

HYATTSVILLE

Prince George's County Health Department Center for Addiction and Pregnancy
3003 Hospital Drive
Hyattsville, MD 20781

JESSUP

Clifton T. Perkins Hospital Center Alcohol and Drug Abuse Services
8450 Dorsey Run Road
Jessup, MD 20794

Regimented Offender Treatment Center for Men
Jessup, MD 20794

JOPPA

Joppa Health Services, Inc.
623-A Pulaski Highway
Joppa, MD 21085

LANHAM

Kolmac Clinic
7726 Finns Lane, Suite 101
Lanham, MD 20706

LAUREL

Act II Counseling Services, Inc.
379 Main Street, Suite 4
Laurel, MD 20707

Counseling Services, Inc.
150 Washington Boulevard
Suite 200
Laurel, MD 20707

Flynn/Lang Counseling Center
13 C Street, Suite H
Laurel, MD 20707

Mental Health and Addiction Services Laurel Regional Hospital
7300 Van Dusen Road
Laurel, MD 20707

Reality, Inc.
Aftercare
Quarterway House
419 Main Street
Laurel, MD 20707

Continuing Care Facility
429 Main Street
Laurel, MD 20707

We Care Health Services Inc
8730-1 Cherry Lane
Laurel, MD 20707

LEONARDTOWN

Marcey Halfway House
Leonardtown, MD 20650

LUSBY

Calvert County Substance Abuse Program South Maryland Community Center
20 Appeal Lane
Lusby, MD 20657

LUTHERVILLE TIMONIUM

Awakenings Counseling Program
2 West Aylesbury Road
Lutherville Timonium, MD 21093

MILLERSVILLE

Comprehensive Treatment Center of Maryland
1110 Benfield Boulevard I-97
Business Park, Suite H Front
Millersville, MD 21108

MOUNT RAINIER

C. A. Mayo and Associates, Inc.
3403 Perry Street
Mount Rainier, MD 20712

NEW CARROLTON

Awele Social Health Clinic, Inc.
7515 Annapolis Road, Suite 406
New Carrollton, MD 20784

OAKLAND

Garrett County Health Department Addictions Service
221 South 3 Street
Oakland, MD 21550

ODENTON

Ferry Point, Inc. Treatment Center
8379 Piney Orchard Parkway
Odenton, MD 21113

OWINGS MILLS

Phoenix Counseling and Consulting Services, Inc.
10806 Reisterstown Road
Suite 1-B
Owings Mills, MD 21117

Right Turn of Maryland, LLC
10225 Jensen Lane
Owings Mills, MD 21117

PASADENA

New Life Addiction Counseling Services
2528 Mountain Road
Pasadena, MD 21122

PATUXENT RIVER

Counseling and Assistance Center
47096 Liljencrantz Road
Building 438
Patuxent River, MD 20670

PERRY POINT

VA Medical Center Substance Abuse Treatment Program
Building 22
Perry Point, MD 21902

PRINCE FREDERICK

Calvert Substance Abuse Services New Leaf Counseling Center
Route 4 and Stokely Road
Prince Frederick, MD 20678

RANDALLSTOWN

First Step Inc/Northwest Area Program Family Resource Center
3525 Resource Drive
Randallstown, MD 21133

ROCKVILLE

Avery House Halfway House for Women and Children
14705 Avery Road
Rockville, MD 20853

Avery Road Treatment Center
Detoxification Program
Intermediate Care Facility
14703 Avery Road
Rockville, MD 20853

Charter Behavioral Health System at Potomac Ridge
14901 Broschart Road
Rockville, MD 20850

Jail Addictions Services
1307 Seven Locks Road
Rockville, MD 20850

Montgomery County Department of Health and Human Services
The Other Way Day Treatment Program
401 Fleet Street
Rockville, MD 20850

Outpatient Addiction Services
751 Twinbrook Parkway
Rockville, MD 20851

Lawrence Court Halfway House
1 Lawrence Court
Rockville, MD 20850

Montgomery Recovery Services, Inc.
14636 Rothgeb Drive
Rockville, MD 20850

OACES Corporation
330A Hungerford Drive
Rockville, MD 20850

Second Genesis, Inc. Montgomery County
14701 Avery Road
Rockville, MD 20853

Suburban Hospital Addiction Treatment Center
6001 Montrose Road, Suite 205
Rockville, MD 20850

White Flint Recovery, Inc.
1335 Rockville Pike
Suite 106
Rockville, MD 20852

SABILLASVILLE

Catoctin Summit Adolescent Program
5980 Cullen Drive
Sabillasville, MD 21780

SALISBURY

Hudson Health Services, Inc.
Willis Hudson Alcohol and Drug Treatment Center
1506 Harting Drive
Salisbury, MD 21802

Peninsula Addiction Services
104 West Market Street
Salisbury, MD 21801

Peninsula Regional Medical Center
100 East Carroll Street
Salisbury, MD 21801-5493

Second Wind, Inc.
309 Newton Street
Salisbury, MD 21801

Wicomico Behavioral Health
108 East Main Street
Salisbury, MD 21801

SEVERNA PARK

Stress and Health Management Center Inc.
540 Ritchie Highway
Suite 101
Severna Park, MD 21146

SILVER SPRING

Another Way, Inc.
11308 Grandview Avenue
2nd Floor
Silver Spring, MD 20902-4634

Bilingual Counseling Center
2419 Reedie Drive Suite 201
Silver Spring, MD 20902

D. A. Wynne and Associates Inc.
1709 Elton Road
Silver Spring, MD 23903

Guide Program, Inc. Adult Treatment Services
11141 Georgia Avenue, Suite 420
Silver Spring, MD 20902

Kolmac Clinic
1003 Spring Street
Silver Spring, MD 20910

Saint Luke Institute
8901 New Hampshire Avenue
Silver Spring, MD 20903

Second Genesis, Inc. Outpatient Adolescent Family Services
1721 Elton Road
Silver Spring, MD 20901

Thomas Comp. Counseling Services, Inc.
800 Pershing Drive
Suite 105A
Silver Spring, MD 20910

SNOW HILL

Worcester County Center for a Clean Start
Snow Hill, MD 21863

Worcester County Health Department Alcohol and Other Drug Services
6040 Public Landing Road
Snow Hill, MD 21863

SUITLAND

Prince Georges County Health Department Addictions/ Northern Region
5408 Silver Hill Road, Room 213
Suitland, MD 20747

SYKESVILLE

Adapt Counseling Incorporated
1643 Liberty Road Suite 204
Sykesville, MD 21784

Clinical Services Program Residential Substance Abuse Treatment
Central Laundry Facility
Sykesville, MD 21784

Shoemaker Center
6655 Buttercup Road
Sykesville, MD 21784

Women's Project
6655 Buttercup Road
Sykesville, MD 21784

TAKOMA PARK

Washington Adventist Hospital
7600 Carroll Avenue
Takoma Park, MD 20912

THURMONT

Catoctin Counseling Center
18 North Church Street
Thurmont, MD 21788

TOWSON

Pathfinder Health Services
300 East Joppa Road, Suite 303
Towson, MD 21286

Towson University
8000 York Road
Towson, MD 21252-0001

UPPER MARLBORO

Another Spring Counseling Services
5302 Water Street, Suite 204
Upper Marlboro, MD 20772

Drinking Driver Monitor Program
14735 Main Street
PG County Courthouse
Room 068-B
Upper Marlboro, MD 20772

Institute of Life and Health Alcohol andDrug Assessment and Therapy Program
5311 Water Street, Suite D
Upper Marlboro, MD 20772

Second Genesis, Inc.
Mellwood House
4620 Mellwood Road
Upper Marlboro, MD 20772

VALLEY LEE

Seafarers Addiction Rehabilitation Center
45705 Locust Grove Drive
Valley Lee, MD 20692

WALDORF

Charles County Health Department Substance Abuse Program
2670 Crain Highway, Site 300
Waldorf, MD 20604

Mid Atlantic Mental Health Center, Inc. QUIT Program
2 Industrial Park Drive, Suite B
Waldorf, MD 20602

Open Arms, Inc.
2590 Business Park Court
Waldorf, MD 20601-2904

WESTMINSTER

Carroll County Health Department
Bureau of Addiction Outpatient Treatment Services
290 South Center Street
Westminster, MD 21157

Junction, Inc. Drug and Alcohol Abuse Treatment Program
98 North Court Street
Westminster, MD 21157

Mountain Manor Treatment Center
Carroll Plaza, Suite 2
Westminster, MD 21158

Reentry Mental Health Services Addiction Services
40 South Church Street
Suite 105
Westminster, MD 21157

Westminster Rescue Mission
685 Lucabaugh Mill Road
Westminster, MD 21157

WESTOVER

Somerset County Health Department Behavioral Health Services
7920 Crisfield Highway
Westover, MD 21871

WHEATON

Counseling Plus, Inc.
11141 Georgia Avenue, Suite A-24
Wheaton, MD 20902

MASSACHUSETTS

ALLSTON

Granada House, Inc
70 Adamson Street
Allston, MA 02134

ATTLEBORO

The Road Back
7 Forest Street
Attleboro, MA 02703

BEDFORD

Veterans' Affairs Addiction Treatment Center
200 Springs Road
Room 116 A
Bedford, MA 01730

BELMONT

McLean Hospital Alcohol and Drug Abuse Treatment Center
115 Mill Street
Appleton Building
Belmont, MA 02178

BEVERLY

Leland Unit Beverly Hospital
85 Herrick Street
Beverly, MA 01915

BOSTON

Bay Cove Human Services
66 Canal Street
Boston, MA 02111

Boston Alcohol and Substance Abuse Program
30 Winter Street
Boston, MA 02108

Boston Childrens Services Alcohol/Drug Use Assessment and Treatment Program
271 Huntington Avenue
Boston, MA 02116

Boston Public Health Commission
Acupuncture Clinic
Addiction Services
Outpatient Counseling
723 Massachusetts Avenue
Boston, MA 02118

Bridge Over Troubled Waters, Inc.
Youth Intervention Program
47 West Street
Boston, MA 02111

Entre Familia
1010 Massachusetts Avenue
Boston, MA 02118

Fenway Community Health Center
Acupuncture Detoxification Clinic
Outpatient Substance Abuse Services
7 Haviland Street
Boston, MA 02115

Harvard Vanguard Medical Associates
23 Miner Street
Boston, MA 02215

Justice Resource Institute Health Division
130 Boylston Street
Boston, MA 02116

Latino Health Institute Substance Abuse Clinic
95 Berkeley Street
Boston, MA 02116

Marathon Acute Treatment Services
Administration Building, 2nd Floor
Long Island Health Campus
Boston, MA 02122

Massachusetts General Hospital Addiction Services/Outpatient
15 Blossom Street
Boston, MA 02114

Span, Inc.
110 Arlington Street
Boston, MA 02116

Spaulding Rehabilitation Hospital
125 Nashua Street
Boston, MA 02114

Veterans' Affairs Medical Center Substance Abuse Treatment Program
251 Causeway Street
Boston, MA 02130

BRAINTREE

Family Counseling and Guidance Center
40 Independence Avenue
Braintree, MA 02184

BRIGHTON

Addiction Treatment Center of New England, Inc. Methadone Services
77 Warren Street
Brighton, MA 02135

Saint Elizabeth's Hospital Comprehensive Alcohol and Addiction Program
736 Cambridge Street
Cardinal Cushing Building
Brighton, MA 02135

BROCKTON

Brockton Hospital Substance Abuse Services
680 Center Street
Brockton, MA 02302

Catholic Charities
Edwina Martin Recovery House
678 North Main Street
Brockton, MA 02401

Alcohol Detox
Outpatient Services
Substance Abuse Services
Resurrection House
686 North Main Street
Brockton, MA 02401

MSPCC Family Counseling Center Outpatient Substance Abuse Services
231 Main Street
Brockton, MA 02401

Old Colony Services Corporation Mental Health Clinic
15-A Bolton Place
Brockton, MA 02401

South Bay Mental Health Center
37 Belmont Street
Brockton, MA 02401

Veterans' Affairs Medical Center Alcohol and Drug Dependence Program
940 Belmont Street
Brockton, MA 02401

BROOKLINE

Bournewood Health Systems
300 South Street
Brookline, MA 02467-3694

CAMBRIDGE

Caspar, Inc.
Outpatient Program
126 Prospect Street
Cambridge, MA 02139

Womanplace Halfway House for Women
11 Russell Street
Cambridge, MA 02140

CHARLESTOWN

John F. Kennedy Family Service Center, Inc. Outpatient Substance Abuse Services
27 Winthrop Street
Boston (Charlestown), MA 02129

CHELSEA

Bay Cove Human Services
Chelsea Substance Abuse Clinic
100 Everett Avenue
Unit 4
Chelsea, MA 02150

CHICOPEE

Community Health Care, Inc. Community Substance Abuse Centers
628 Center Street
Chicopee, MA 01013

CLINTON

Clinton Hospital
201 Highland Street
Clinton, MA 01510

CONCORD

Assabet Human Services, Inc. Outpatient Substance Abuse Services
Damonmill Square
Suite 2A
Concord, MA 01742

Emerson Hospital Aftercare Addiction Services/Outpatient
133 Old Road to Nine Acre Corner
Concord, MA 01742

DANVERS

Cab Health and Recovery Services, Inc.
Inpatient Detox Unit
Opiate Addiction Treatment Services
Residential Intermediate Care Facility
111 Middleton Road
Danvers, MA 01923

DORCHESTER

Boston Hamilton House, Inc. Hamilton Recovery Home
25 Mount Ida Road
Dorchester, MA 02122

Carney Hospital Drug and Alcohol Program/Outpatient Psychiatry
2100 Dorchester Avenue
Dorchester, MA 02124

Codman Square Health Center Outpatient Substance Abuse Services
637 Washington Street
Dorchester, MA 02124

Dorchester House
1353 Dorchester Avenue
Dorchester, MA 02122

Federal Dorchester Neighborhood Houses
Little House/Outpatient
Youth Assistance
275 East Cottage Street
Dorchester, MA 02125

First Hispanic Academy
632 Blue Hill Avenue
Dorchester, MA 02121-3213

First, Inc.
First Step
Outpatient Services
321 Blue Hill Avenue
Dorchester, MA 02121

Interim House, Inc. Recovery Home
62 Waldeck Street
Dorchester, MA 02124

Victory Programs, Inc.
New Victories/Recovery Home
9 Virginia Street
Dorchester, MA 02125

Shepherd House
22 and 24 Windermere Road
Dorchester, MA 02125

Womens Hope
10 Chamblet Street
Dorchester, MA 02125

EAST BOSTON

North Suffolk Mental Health
408 Meridian Street
East Boston, MA 02128

Rehabilitation and Health, Inc. Recovery Home
52 White Street
East Boston, MA 02128

EVERETT

Tri City Mental Health and Retardation Center, Inc.
173 Chelsea Street
Everett, MA 02149

FALL RIVER

Family Service Assoc. of Greater Fall River Outpatient Substance Abuse Services
151 Rock Street
Fall River, MA 02720

Portuguese Youth Cultural Organization
Outpatient Substance Abuse Services
186 South Main Street
Fall River, MA 02721

Stanley Street Treatment and Resources
Alcoholism/Drug Detox Program
Chemical Dependency Services/ Outpatient
Women's Rehab Program
386 Stanley Street
Fall River, MA 02720

Steppingstone, Inc.
Halfway House
466 North Main Street
Fall River, MA 02720

Outpatient Substance Abuse Services
101 Rock Street
Fall River, MA 02720

Therapeutic Community
522 North Main Street
Fall River, MA 02720

FALMOUTH

CCAIRU Gosnold Counseling Center
Outpatient Substance Abuse Services
196 Ter Heun Drive
Falmouth, MA 02540

Cape Cod Detoxification Center
200 Ter Heun Drive
Falmouth, MA 02540

Stephen Miller House Recovery Home
165 Woods Hole Road
Falmouth, MA 02540

FITCHBURG

Luk Crisis Center, Inc. Youth Assistance Program
99 Day Street
Fitchburg, MA 01420

FLORENCE

Cooley Dickinson Hospital Outpatient Behavioral Health Services
10 Main Street
Florence, MA 01062

FRAMINGHAM

Farmingham Detox Program
3 Merchant Road
Framingham, MA 01704-0606

Genesis Counseling Services, Inc.
24 Union Avenue, Suite 11
Framingham, MA 01702

New England Aftercare Ministries, Inc. The Bridge House/Halfway House
18–20 Summit Street
Framingham, MA 01701

South Middlesex Opportunity Council Behavioral Health Services
1100 Wooster Road, 4th Floor
Framingham, MA 01701

Victory Programs, Inc. Women's Hope Center
Loring Avenue
Framingham, MA 01701

Wayside Metrowest Counseling Center
88 Lincoln Street
Framingham, MA 01701

GARDNER

Gardner Athol Area Mental Health Association, Inc. Pathway House
34 Catherine Street
Gardner, MA 01440

North Central Human Services
31 Lake Street
Gardner, MA 01440

GEORGETOWN

Baldpate Hospital Outpatient Services
Baldpate Road
Georgetown, MA 01833

GLOUCESTER

Health and Education Services
298 Washington Street
Gloucester, MA 01930

GREENFIELD

Franklin Medical Center
Beacon Clinic
60 Wells Street
Greenfield, MA 01301

Beacon Recovery Center
164 High Street
Greenfield, MA 01301

Beacon House for Men/Recovery House
57 Beacon Street
Greenfield, MA 01301

Beacon House for Women/ Recovery House
153 High Street
Greenfield, MA 01301

HANSCOM AFB

Hanscom Air Force Base Substance Abuse Program
66 MDOS/SGOFH 90 Vandenberg Drive
Hanscom AFB, MA 01731

HAVERHILL

Team Coordinating Agency, Inc.
Community Outreach
Outpatient Substance Abuse Services
66-76 Winter Street
Haverhill, MA 01831

Phoenix East
20 Newcomb Street
Haverhill, MA 01831

Youth Assistance Program
350 Main Street
Haverhill, MA 01831

HINGHAM

Project Turnabout
224 Beal Street
Hingham, MA 02043

HOLYOKE

Holyoke Hospital Partial Hospitalization IOUTPT Program
575 Beech Street
Holyoke, MA 01040

MSPCC Family Counseling Center Outpatient Substance Abuse Services
113 Hampden Street
Holyoke, MA 01040

Providence Hospital
1233 Main Street
Holyoke, MA 01040

Substance Abuse Outpatient Programs
317 Maple Street
Holyoke, MA 01040

Honor House
40 Brightside Drive
Holyoke, MA 01040

HOPKINTON

SMOC Behavioral Health Services Serenity House
44 Wilson Street
Hopkinton, MA 01748

HYANNIS

Cape Cod Human Services
Outpatient Substance Abuse Services
460 West Main Street
Hyannis, MA 02601

CCAIRU, Inc. Transitional Care Facility
71 Pleasant Street
Hyannis, MA 02601

JAMAICA PLAIN

Arbour Substance Abuse Program
49 Robinwood Avenue
Jamaica Plain, MA 02130

Boston Alcohol Detox Project, Inc.
170 Morton Street
Jamaica Plain, MA 02130

Brigham and Women's Hospital
Brookside Community Health Center
3297 Washington Street
Jamaica Plain, MA 02130

South Jamaica Plain Health Center
FACTS Program
687 Centre Street
Jamaica Plain, MA 02130

Faulkner Hospital Addiction Recovery Program
1153 Centre Street
Jamaica Plain, MA 02130

Sullivan House
65 Glenn Road
Jamaica Plain, MA 02130

Veterans Affairs Medical Center Substance Abuse Treatment Program
150 South Huntington Avenue
Jamaica Plain, MA 02130-1831

Volunteers of America Outpatient Clinic
441 Centre Street
Jamaica Plain, MA 02130

LAWRENCE

Arbour Counseling Services
599 Canal Street
1 East
Lawrence, MA 01840

Centro Panamericano, Inc. Substance Abuse Outpatient Services
101 Amesbury Road
Suite 402
Lawrence, MA 01841

Family Services, Inc.
430 North Canal Street
Lawrence, MA 01840

Greater Lawrence Mental Health Center, Inc.
30 General Street
Lawrence, MA 01841-0007

Habit Management Institute
599 Canal Street
Lawrence, MA 01840

Psychological Center
1 South Union Street
Lawrence, MA 01840

Pegasus Youth Residence
482 Lowell Street
Lawrence, MA 01840

Women's View
582–584 Haverhill Street
Lawrence, MA 01841

LEOMINSTER

Community Health and Prevention Services
Detoxification Center
17 Orchard Street
Leominster, MA 01453

Outpatient Counseling
71 Pleasant Street
Leominster, MA 01453

LOWELL

Center for Family Development
45 Merrimack Street
Lowell, MA 01850

Family Service of Greater Lowell
97 Central Street
Suite 400
Lowell, MA 01852

Habit Management Institute
650 Suffolk Street
Lowell, MA 01854

Lowell Community Health Center, Inc. Community Health Initiatives/Outpatient
685 Lawrence Street
Lowell, MA 01852

Lowell House, Inc.
Outpatient Substance Abuse Services
555 Merrimack Street
Lowell, MA 01854

Residential Services
102 Appleton Street
Lowell, MA 01852

LYNN

Center for Addictive Behaviors, Inc.
Ryan Rehabilitation Center
100 Green Street
Lynn, MA 01902

Lynn Community Health Center
269 Union Street
Lynn, MA 01901

Project Cope
Outpatient Substance Abuse Services
117 North Common Street
Lynn, MA 01902

Willow Street Medical Center
100 Willow Street
Lynn, MA 01901

MALDEN

Adult/Adolescent Counseling, Inc.
389 Main Street
Malden, MA 02148

Eastern Middlesex Alcoholism Services
Recovery House
12 Cedar Street
Malden, MA 02148

HRI Counseling Centers, Inc.
DBA Arbour Counseling Services Recovery Network
6 Pleasant Street
Malden, MA 02148

MARBLEHEAD

Marblehead Counseling Center, Inc. Outpatient Substance Abuse Program
66 Clifton Avenue
Marblehead, MA 01945

MARLBOROUGH

UMASS Memorial Healthcare
Psychiatric and Addictions Services
57 Union Street
Marlborough, MA 01752

Advocates, Inc. Community Counseling
133 East Main Street
Marlborough, MA 01752

MARSHFIELD

North River Counseling, Inc.
769 Plain Street, Suite 1
Marshfield, MA 02050

MATTAPAN

Marathon, Inc.
River Street Detoxification Center
Stair Program
249 River Street
Mattapan, MA 02126

MIDDLEBORO

Community Care Services
94 South Main Street
Middleboro, MA 02346

MILFORD

Wayside Community Counseling Center
Substance Abuse Program
10 Asylum Street
Milford, MA 01757

NANTUCKET

Family and Children's Service/ Nantucket Outpatient Substance Abuse Services
Off Vesper Lane
Nantucket, MA 02554

NATICK

Metro West Medical Center
67 Union Street
Natick, MA 01760

NEW BEDFORD

Center for Health and Human Services
Outpatient Alcohol and Drug Program
800 Purchase Street
Suite 350
New Bedford, MA 02740

Methadone Services
88–90 Gifford Street
New Bedford, MA 02741

Marcotic Treatment Program
86 Gifford Street
New Bedford, MA 02741

Harmony House
234 Earle Street
New Bedford, MA 02746

New Bedford Child and Family Services
1061 Pleasant Street
New Bedford, MA 02740

Professional Counseling Center Outpatient Substance Abuse Services
466 County Street
New Bedford, MA 02740

NEWBURYPORT

John Ashford Link House
37 Washington Street
Newburyport, MA 01950

Turning Point, Inc. Outpatient Substance Abuse Counseling
5 Perry Way
Newburyport, MA 01950

NEWTON

Newton Outpatient Center
64 Eldredge Street
Newton, MA 02456

NORFOLK

Caritas Southwood Hospital Outpatient Substance Abuse Services
111 Dedham Street, Route 1-A
Norfolk, MA 02056

NORTH ADAMS

North Adams Regional Hospital Substance Abuse Services
Hospital Avenue
North Adams, MA 01247

NORTHAMPTON

Community Health Care, Inc. Substance Abuse Center
297 Pleasant Street
Northampton, MA 01060

Veterans' Affairs Medical Center Substance Abuse Treatment Program
421 North Main Street
Northampton, MA 01060

NORTHBOROUGH

Northborough Family and Youth Services Outpatient Substance Abuse Services
63 Main Street
Northborough, MA 01532

NORTH DARTMOUTH

Saint Lukes Hospital Psychiatric Outpatient Services
74 Faunce Corner
North Dartmouth, MA 02747

NORTON

North Cottage Program, Inc. Halfway House
69 East Main Street
Norton, MA 02766

OAK BLUFFS

Martha's Vineyard Community Services Island Counseling Center/Outpatient
Off Edgartown/Vineyard Haven Road
Oak Bluffs, MA 02557

PITTSFIELD

Berkshire Medical Center Hillcrest Hospital Thomas W. McGee Unit
165 Tor Court
Pittsfield, MA 01201

Mental Health and Substance Abuse Services of the Berkshires
131 Bradford Street
Pittsfield, MA 01202

Keenan House Recovery Home
206 Francis Avenue
Pittsfield, MA 01201

PLYMOUTH

Center for Health and Human Services/AFR
71 Christa McAuliffe Boulevard
Plymouth, MA 02360

High Point Treatment Center
Detox, Outpatient, STIT Programs
1233 State Road
Plymouth, MA 02360

QUINCY

Bay State Community Services, Inc.
Outpatient Substance Abuse Service
15 Cottage Avenue
Quincy, MA 02169

Quincy Detoxification Center, Inc. DBA Faxon Recovery Service
120 Whitwell Street
Quincy, MA 02169

South Shore Halfway House
10 Dysart Street
Quincy, MA 02169

South Shore Mental Health Center Outpatient Substance Abuse Program
6 Fort Street
Quincy, MA 02169

Spectrum Health Systems, Inc. Right Turn
1458 Hancock Street
Quincy, MA 02169

ROXBURY

Boston Public Health Commission Narcotic Addiction Clinic/Methadone Services
300 Frontage Road
Roxbury, MA 02118

Casa Esperanza, Inc.
291 Eustis Street
Roxbury, MA 02119

Dimock Community Health Center
Alcohol and Drug Detox Program
John Flowers Recovery Home
Substance Abuse Services
55 Dimock Street
Roxbury, MA 02119

Habit Management, Inc. Boston Methadone Services
99 Topeka Street
Roxbury, MA 02119

Hope House, Inc. Recovery Home
42 Upton Street and 24 Hanson Street
Roxbury, MA 02118

La Alianza Hispana, Inc. Outpatient Services
409 Dudley Street
Roxbury, MA 02119

Roxbury Comprehensive Community Health Center, Inc. Methadone Services
435 Warren Street
Roxbury, MA 02119

Salvation Army Harbor Light Center
407 Shawmut Avenue
Roxbury, MA 02118

Tecumseh House Drop In Center
107 Fisher Avenue
Roxbury, MA 02120

Victory Programs, Inc.
Victory House/Recovery Home
566 Massachusetts Avenue
Roxbury, MA 02118

Volunteers of America Hello House
686 Massachusetts Avenue
Roxbury, MA 02118

SALEM

Cab Health and Recovery Systems
27 Congress Street
Salem, MA 01970

Health and Education Services Outpatient Substance Abuse Program
162 Federal Street
Salem, MA 01970

North Shore Medical Center Addictive Disease Unit
81 Highland Avenue
Salem, MA 01970

Salem Hospital Addictive Disease Program/Outpatient
172 Lafayette Street
Salem Hospital/Professional Services Building
Salem, MA 01970

SOMERVILLE

Cambridge Health Alliance
26 Central Street
Somerville, MA 02143

Caspar, Inc.
Alcohol and Drug Education
Youth Assistance
226 Highland Avenue
Somerville, MA 02143

Intervention/Detox
245 Beacon Street
Somerville, MA 02143

Men's Recovery Home
16 Highland Avenue
Somerville, MA 02143

9 Kidder Avenue
Somerville, MA 02144

New Day
242 Highland Avenue
Somerville, MA 02143

Central Street Health Center
26 Central Street
Somerville, MA 02143

Mass Alliance of Portuguese Speakers
Acupuncture Services
Outpatient Substance Abuse Services
92 Union Square
Somerville, MA 02143

North Charles Institute
Outpatient Substance Abuse Services
260 Beacon Street
Somerville, MA 02143

Somerville Mental Health Assoc, Inc.
5 Hall Avenue
Somerville, MA 02144

SOUTH BOSTON

Arch Foundation, Inc.
675 East 4 Street
South Boston, MA 02127

Middlesex Human Services Agency
5 G Street
South Boston, MA 02127

SOUTHBRIDGE

Harrington Memorial Hospital
Wells Human Services Center
29 Pine Street
Southbridge, MA 01550

Youth Opportunities Upheld, Inc. Family Services/Youth Program
52 Charlton Street
Southbridge, MA 01550

SOUTH YARMOUTH

Habit Management Institute/ Yarmouth Methadone Services
20 Forsyth Street
South Yarmouth, MA 02664

SPRINGFIELD

Bay State Medical Center
Carlson Recovery Center
Sloan Clinic
471 Chestnut Street
Springfield, MA 01199

Opportunity House
59–61 Saint James Avenue
Springfield, MA 01109

Women's Division/My Sisters House
89 Belmont Avenue
Springfield, MA 01108

Child and Family Service, Inc.
367 Pine Street
Springfield, MA 01105

Gandara Center
Addiction Recovery Program
29-33 Arch Street
Springfield, MA 01107

Mental Health and Substance Abuse
2155 Main Street
Springfield, MA 01104

Habit Management Institute
2257 Main Street
Springfield, MA 01107

Marathon, Inc.
5 Madison Avenue
Springfield, MA 01105

Northern Educational Services, Inc.
Ethos I/Recovery Home
56 Temple Street
Springfield, MA 01105

Ethos III Outpatient Services
756 State Street
Springfield, MA 01109

Providence Hospital
Insights Program
209 Carew Street
Springfield, MA 01104

Methadone Program
227 Mill Street
Springfield, MA 01105

STONEHAM

Boston Regional Medical Center Addictions Treatment Services/Outpatient
5 Woodland Road
Stoneham, MA 02180

TAUNTON

Community Counseling of Bristol County Outpatient Substance Abuse Services
68 Church Green Street, Suite 2
Taunton, MA 02780

Greater Taunton Council on Alcoholism
71 Main Street
Taunton, MA 02780

TEWKSBURY

HART House
365 East Street
Tewksbury, MA 01876

Lowell Community Health Center, Inc. Community Health Initiatives/Detox
Tewksbury Hospital
365 East Street, Unit 1
Tewksbury, MA 01876

Middlesex Human Service Agency, Inc. DUI Program
Tewksbury Hospital
365 East Street, Hall III
Tewksbury, MA 01876

UPTON

Riverside Community Care Blackstone Valley Outpatient Care
206 Milford Street
Upton, MA 01568

WAKEFIELD

Eastern Middlesex Human Services Outpatient
338 Main Street, Suite 304
Wakefield, MA 01880

WALTHAM

Hurley House Recovery Home
12–14 Lowell Street
Waltham, MA 02154

Middlesex Regional Addiction Treatment Center
775 Trapelo Road
Waltham, MA 02154

Outpatient Services
50 Prospect Street
Suite 201
Waltham, MA 02154

WELLESLEY

Charles River Hospital Dual Diagnosis Program
203 Grove Street
Wellesley, MA 02181

WESTBOROUGH

Spectrum Addiction Services, Inc.
Primary Care
Spectrum Residential Program
155 Oak Street
Westborough, MA 01581

WEST FALMOUTH

CCAIRU Emerson House
554 West Falmouth Highway
West Falmouth, MA 02574

WESTFIELD

Community Health Care Inc. Substance Abuse Center
125 North Elm Street
Westfield, MA 01085

Providence Hospital Westfield Counseling Center
41 Church Street
Westfield, MA 01085

WESTWOOD

Westwood Lodge Hospital
45 Clapboardtree Street
Westwood, MA 02090

WOBURN

Arbour/Choate Counseling Services
500 West Cummings Park
Suite 3900
Woburn, MA 01801

WORCESTER

Adcare Hospital Substance Abuse Treatment Program
107 Lincoln Street
Worcester, MA 01605

Catholic Charities/Worcester Crozier House
10 HammondStreet
Worcester, MA 01610

Community Healthlink
Detoxification Program
Outpatient Substance Abuse Services
12 Queen Street
Worcester, MA 01610

DUI Program
72 Jaques Avenue
Worcester, MA 01610

Recovery Home
142 Burncoat Street
Worcester, MA 01606

Family Health Center of Worcester
26 Queen Street
Worcester, MA 01610

Henry Lee Willis Community Center
Channing House Recovery Home
21 Catherine Street
Worcester, MA 01605

Linda F. Griffin House
15 Northampton Street
Worcester, MA 01605

Outpatient Substance Abuse Services
44 Front Street, Suite 210
Worcester, MA 01609

Lincoln Group, The
79 June Street
Worcester, MA 01602

Saint Vincent's Hospital Deptartment of Alcohol and Drug Services/Outpatients
25 Winthrop Street
Worcester, MA 01604

Spectrum Addiction Services, Inc.
Outpatient Services
105 Merrick Street
Worcester, MA 01609

585 Lincoln Street
Worcester, MA 01605

Youth Opportunities Upheld, Inc. Structured Outpatient Services
81 Plantation Street
Worcester, MA 01604

MICHIGAN

ADRIAN

Emma L. Bixby Medical Center Sage Center for Substance Abuse Treatment
818 Riverside Avenue
Adrian, MI 49221

Family Service and Children's Aid
405 Mill Street
Adrian, MI 49221

McCullough Vargas and Associates
127 South Winter Street
Adrian, MI 49221

ALBION

Psychological Consultants of BC Chemical Dependency Resources
300-B Drive North
Albion, MI 49224

ALGONAC

Downriver Community Services, Inc. Substance Abuse Services
555 Saint Clair River Drive
Algonac, MI 48001

ALLEGAN

Allegan County Community Mental Health Services
3285 122nd Avenue
Allegan, MI 49010

CSAS, Inc. Family Recovery Center of Allegan County
138 B Hubbard Street
Allegan, MI 49010

ALLEN PARK

Pro Med Management Evergreen Counseling Centers
15101 Southfield Road
Allen Park, MI 48101

Josephine Sheehy Program
7445 Allen Road
Suite 190
Allen Park, MI 48107

ALMA

Human Aid, Inc.
1750 Wright Avenue
Alma, MI 48801

Pine River Recovery Center
300 Warwick Drive
Alma, MI 48801

ALPENA

Birchwood Center for Chemical Dependency
1501 West Chisholm Street
Alpena Hospital
Alpena, MI 49707

Catholic Human Services, Inc.
154 South Ripley Boulevard
Alpena, MI 49707

Sunrise Centre
630 Walnut Street
Alpena, MI 40707

ANN ARBOR

Ann Arbor Veterans Administration Medical Center
2215 Fuller Road
Ann Arbor, MI 48105

Catholic Social Services Substance Abuse Services
4952 Packard Road
Ann Arbor, MI 48104

Center for Behavior and Medicine
2004 Hogback Road, Suite 16
Ann Arbor, MI 48105

Chelsea Community Hospital
Older Adult Recovery Program
955 West Eisenhower Circle
Suite E
Ann Arbor, MI 48103

Chelsea Arbor Treatment Center
900 Victors Way Suite 310
Ann Arbor, MI 48108

Child and Family Service of Washtenaw Chemical Dependency Program
3879 Packard Road
Ann Arbor, MI 48104

Dawn, Inc.
Dawn Re-Entry
502 West Huron Street
Ann Arbor, MI 48104

Dawn Farm Detox
544 North Division Street
Ann Arbor, MI 48104

Home of New Vision
2500 Packard Street, Suite 201-A
Ann Arbor, MI 48104

Huron Valley Consultation Center
Carpenter Outpatient
2750 Carpenter Road
Ann Arbor, MI 48108

Eisenhower Outpatient
955 West Eisenhower Circle
Suite B
Ann Arbor, MI 48103

Institute for Psychology and Medicine
2010 Hogback Road
Suite 6
Ann Arbor, MI 48105

Jackson Counseling Agency
1900 West Stadium Boulevard
Suite 5
Ann Arbor, MI 48103

Mercy Health Service McAuley Chemical Dependency Center
2006 Hogback Road
Ann Arbor, MI 48105

Spectrum
2301 Platt Road
Ann Arbor, MI 48107

AUBURN HILLS

Havenwyck Hospital Substance Abuse Services
1525 University Drive
Auburn Hills, MI 48326

BAD AXE

Huron Counseling Services
1108 South Van Dyke Road
Bad Axe, MI 48413

List Psychological Services
65 Patrick Street, Suite 5
Bad Axe, MI 48413

BALDWIN

Family Health Care Counseling Center
1101 Washington Avenue
Baldwin, MI 49304

BARAGA

KBTCAP Outpatient Counseling Services
427 North Superior Avenue
Baraga, MI 49908

BARK RIVER

Hannaville Three Fires Halfway House Substance Abuse Program
3017 D Road
Bark River, MI 49807

BATTLE CREEK

Battle Creek Health System
165 North Washington Avenue
Battle Creek, MI 49016

Chemical Dependency Resources
151 North Avenue
Battle Creek, MI 49017

Oakridge Counseling Center
497 East Columbia Avenue
Suite 16
Battle Creek, MI 49015-4463

SPGB Services Inc
34 West Jackson Street
Suite 2, Lower Level
Battle Creek, MI 49224

Veterans' Affairs Medical Center Substance Abuse Treatment Unit
550 Armstrong Road
Battle Creek, MI 49015

BAY CITY

BASIS, Inc.
New Friendship House of Bay County
Residential Treatment Services
Riverside Outpatient
700 North Van Buren Street
Bay City, MI 48708

Riverside Center
904 6th Street
Bay City, MI 48708

Catholic Family Services
915 Columbus Avenue
Bay City, MI 48708

List Psychological Services
3741 East Wilder Road
Bay City, MI 48706

BELLAIRE

Antrim Kalkaska Community MH
205 East Cayuga Street
Bellaire, MI 49615-0220

CHIP Counseling Center
7053 M-88 Highway South
Bellaire, MI 49615

BELLEVILLE

Community Care Services Substance Abuse Service
25 Owen Street
Belleville, MI 48111

Eastwood Clinics
418 Main Street
Belleville, MI 48111

BENTON HARBOR

Berrien County Health Department Alcohol/Drug Abuse Program
769 Pipestone Street
Benton Harbor, MI 49022

Empowered Living Human Services
105 East Main Street, Suite 404
Benton Harbor, MI 49022

KADAC Holding Company Gateway Services at Benton Harbor
1610 Mall Drive
Benton Harbor, MI 49022

BENZONIA

Benzie Counseling Center
850 Michigan Avenue
Benzonia, MI 49616

Grand Traverse Band of Ottawa/Chippewa Indians Substance Abuse Services
7283 Hoadley Road
Benzonia, MI 49616

BERKLEY

Oakland Family Services Berkley Substance Abuse Services
2351 West 12 Mile Road
Berkley, MI 48072

Recovery Consultants, Inc.
2710 West Twelve Mile Road
Berkley, MI 48072-1630

Respite Counseling Center
3622 West Eleven Mile Road
Berkley, MI 48072

Smith Counseling Services Substance Abuse Services
2790 Coolidge Highway
Berkley, MI 48072

BIG RAPIDS

Nova Counseling Associates, Inc.
1724 North State Street
Big Rapids, MI 49307

BIRMINGHAM

Frazho, Joyce K., MSW
111 South Old Woodward Avenue
Suite 256
Birmingham, MI 48009

HFHS Behavior Services Chemical Dependency Program
350 North Old Woodward Street
Suite 3
Birmingham, MI 48009

Smith, Lewis, Ph.D., PC
600 North Woodward Avenue
Suite 303
Birmingham, MI 48009

BLOOMFIELD HILLS

Auro Medical Center Substance Abuse Services
111 South Woodward Avenue
Suite 120
Bloomfield Hills, MI 48304

Center for Contemporary Psychology PC Outpatient Substance Abuse
35980 Woodward Avenue, Suite 1
Bloomfield Hills, MI 48304

Family Center for Psychological Services
36700 Woodward Avenue
Suite 40, Lower Level
Bloomfield Hills, MI 48304-0928

Oakland Psychological Clinic PC Substance Abuse Services
2050 Woodward Avenue
Suite 110
Bloomfield Hills, MI 48304

Pro Med Management Evergreen Counseling Centers
1760 South Telegraph Road
Bloomfield Hills, MI 48302

Recovery Consultants, Inc.
1591 Opdyke Road
Bloomfield Hills, MI 48304

BRIGHTON

Advanced Counseling Services
7600 Grand River Street
Suite 295
Brighton, MI 48116

Brighton Hospital Alcoholism Treatment Services
12851 East Grand River Street
Brighton, MI 48116

Center for Behavior and Medicine
10299 East Grand River Street
Suite I
Brighton, MI 48116

SOS Livingston
325 South Grand River
Brighton, MI 48116

CADILLAC

Catholic Human Services
140 West River Street
Suite 7
Cadillac, MI 49601

CALUMET

Phoenix House, Inc.
422 Pine Street
Calumet, MI 49913

Up Contract Services, Inc.
1175 Calumet Avenue, Suite C
Calumet, MI 49913

CANTON

Center for Behavior and Medicine
2200 Canton Center Road
Suite 200-B
Canton, MI 48187

Downriver Mental Health Clinic Advanced Counseling Services PC
6223 Canton Road, Suite 210
Canton, MI 48187

Family Service
8564 North Canton Center Road
Canton, MI 48187-5065

Hegira Programs, Inc. Oakdale Recovery Center
43825 Michigan Avenue
Canton, MI 48188

CARO

List Psychological Services
443 North State Street
Caro, MI 48723

Thumb Area Behavioral Services Center
1309 Cleaver Road
Caro, MI 48723

CENTER LINE

New Alternatives
25501 Van Dyke Street
Center Line, MI 48015

Options Counseling Services, Inc.
25529 Van Dyke Street
Center Line, MI 48015

CENTREVILLE

Centreville Psychological Services
227 West Main Street
Centreville, MI 49032

CHARLEVOIX

Bay Area Substance Education Services, Inc.
6123 Old U.S. 31 South
Charlevoix, MI 49720

CHIP Counseling Center
6777 U.S. 31 South
Charlevoix, MI 49720

Grand Traverse Band of Ottawa/Chippewa Indians Substance Abuse Services
6429 M-66
Charlevoix, MI 49720

Northern Michigan CMH Dual Diagnosis Program
218 Garfield Street
Charlevoix, MI 49720

CHARLOTTE

Eaton Substance Abuse Program, Inc.
551 Courthouse Drive
Charlotte, MI 48813

CHEBOYGAN

CHIP Counseling Center
520 North Main Street
Suite 106
Cheboygan, MI 49721

Sue Patrick Substance Abuse Services
520 North Main Street
Suite 200
Cheboygan, MI 49721

CHELSEA

Chelsea Arbor Treatment Center
Chelsea Community Hospital
775 South Main Street
Chelsea, MI 48118

CLARE

Human Aid, Inc.
1426 North McEwan Street
Clare, MI 48617

CLARKSTON

Insight Recovery Center/ Clarkston
9075 Big Lake Road
Clarkston, MI 48347

North Oakland Counseling Associates
6401 Citation Drive, Suite C
Clarkston, MI 48346

Saint Joseph Mercy Hospital Mercy Behavioral Center
6770 Dixie Highway, Suite 308
Clarkston, MI 48346

Triad Associates PC
8062 Ortonville Road
Clarkston, MI 48348-4456

CLAWSON

Chambers and Associates Company
12 Church Avenue
Clawson, MI 48017-1110

CLINTON TOWNSHIP

Action Counseling Clinic, Inc. Substance Abuse Services
23823 15 Mile Road
Clinton Township, MI 48035-3111

Catholic Social Services of MaComb Substance Abuse Services
15980 19 Mile Street
Clinton Township, MI 48038

Chambers and Associates Company
42110 Garfield Street, Suite 200
Clinton Township, MI 48038

Eastwood Community Clinics
35455 Garfield Road
Suite C
Clinton Township, MI 48035

Metro Family Support Counseling PC
16950 19 Mile Road, Suite 2
Clinton Township, MI 48038

Options Counseling Services, Inc.
22900 East Remick Street
Clinton Township, MI 48035

Saint Joseph Mercy Center for Behavioral Medicine
43411 Garfield Street, Suite A
Clinton Township, MI 48038

Salvation Army Harbor Light Center MaComb County Satellite
42590 Stepnitz Drive
Clinton Township, MI 48036

COLDWATER

Community Health Center of Branch County Substance Treatment and Referral Service
316 East Chicago Street
Coldwater, MI 49036

DAVISBURG

Makenzie Counseling Group, Inc.
586 Broadway Street
Davisburg, MI 48350

New Oakland Child/Adolescent Family Center
12731 Andersonville Road
Davisburg, MI 48350

DEARBORN

Arab Community Center for Economic and Social Services (ACCESS)
2601 Saulino Court
Dearborn, MI 48120

Eastwood Clinics
19855 West Outer Drive
Suite 204W
Dearborn, MI 48124

Family Services, Inc.
19855 West Outer Drive
Suite 104
Dearborn, MI 48124

Henry Ford Health Systems
5111 Auto Club Drive
Dearborn, MI 48126

Insight
23400 Michigan Avenue
Suite 405
Dearborn, MI 48124

Oakwood Healthcare Systems
18101 Oakwood Boulevard
Dearborn, MI 48123

Personal Dynamics Center Substance Abuse Program
23810 Michigan Avenue
Dearborn, MI 48124

Serenity Manor, Inc.
1637 Ferney Street
Dearborn, MI 48120

DEARBORN HEIGHTS

Catholic Social Services of Wayne County Substance Abuse Services
20382 Van Born Road
Dearborn Heights, MI 48125

Parkview Counseling Center
25639 Ford Road
Dearborn Heights, MI 48127

Westside Mental Health Services
24548 West Warren Avenue
Dearborn Heights, MI 48127

DETROIT

Adult Psychiatric Clinic North Central
4321 East McNicholse Road
Detroit, MI 48212

American Indian Health and Family Services of Southeast Michigan
4880 Lawndale Street
Detroit, MI 48210

BAPCO Substance Abuse Treatment and Prevention Program
17357 Klinger Street
First Community Baptist Church
Detroit, MI 48212

Boniface Fort Street Clinic
5882 Fort Street
Detroit, MI 48209

Boniface Human Services
Outpatient Program
5884 West Fort
Detroit, MI 48209

Boniface Youth Services
1025 East Forest Street
Room 315
Detroit, MI 48201

Catholic Social Services of Wayne County
9851 Hamilton Avenue
Detroit, MI 48202

Center of Behavioral Therapy PC
24453 Grand River Avenue
Detroit, MI 48219

Childrens Center of Wayne County
79 West Alexandrine Street
Detroit, MI 48201

Community Treatment Center Monica House
15380 Monica Street
Detroit, MI 48238

Comprehensive Services, Inc.
4630 Oakman Boulevard
Detroit, MI 48204

Deaf Options, Inc.
220 Bagley Street, Suite 1020
Detroit, MI 48226

Department of Human Services Gratiot Clinic
3506 Gratiot Avenue
Detroit, MI 48207

Detroit Central City Community Mental Health, Inc.
10 Peterboro Street
Detroit, MI 48201

Detroit East, Inc. Community Mental Health
1970 East Larned Street
Detroit, MI 48207

Detroit Light House Program
3750 Woodward Avenue
Suite C-40
Detroit, MI 48201

Detroit Rescue Mission
3535 3 Street
Detroit, MI 48201

Genesis III
11017 Mack Avenue
Detroit, MI 48214

Eastwood Clinics
15085 East 7 Mile Road
Detroit, MI 48205

11542 Conner Street
Detroit, MI 48205

Outpatient
15125 Gratiot Avenue
Detroit, MI 48205

Eleonore Hutzel Recovery Center
301 East Hancock Street
Detroit, MI 48201

13301 Mound Road
Detroit, MI 48213

Emmanuel House Recovery Program
18570 Fitzpatrick Court
Detroit, MI 48228

Family Services of Detroit/ Wayne County
Downtown Detroit Office
220 Bagley Street
Michigan Building Suite 700
Detroit, MI 48226

18585 Mack Street
Detroit, MI 48236

Harper Hospital
50 East Cnafield Street
Detroit, MI 48201

Harper House/Change Alternative Living/Outpatient
2940 East 8 Mile Road
Detroit, MI 48234

Heartline Inc
8201 Sylvester Street
Detroit, MI 48214

Insight Recovery Center
7430 2nd Avenue
Detroit, MI 48202

Islamic Health and Human Services, Inc.
1249 Washington Boulevard
Book Tower Building
Suite 2040-41
Detroit, MI 48226

Jefferson House
8311 East Jefferson Avenue
Detroit, MI 48214

Latino Family Services, Inc.
3815 West Fort Street
Detroit, MI 48216

Mariners Inn
445 Ledyard Street
Detroit, MI 48201

Mercy Hospital Chemical Dependency Services
5555 Conner Avenue
Detroit, MI 48213

12535 Harper Street
Detroit, MI 48213

Metro Arts Therapy Services
1274 Library Street, Suite 301
Detroit, MI 48226

11000 West McNichols Road
Detroit, MI 48221

Metro East Substance Abuse Treatment Corporation
8047 East Harper Avenue
2nd Floor
Detroit, MI 48213

13627 Gratiot Avenue
Detroit, MI 48205

13929 Harper Avenue
Detroit, MI 48213

Metro Matrix Human Services
Peter Claver Career
450 Elliott Street
Detroit, MI 48201

Project Transition
16260 Dexter Avenue
Detroit, MI 48221

Nardin Park Recovery Center
9605 West Grand River Avenue
Detroit, MI 48204

Neighborhood Service Organization (NSO)
24 Hour Walk-In Center
3430 3rd Street
Detroit, MI 48201

Calvin Wells Treatment Center
8600 Woodward Street
Detroit, MI 48202

Gratiot Services Center
3506 Gratiot Avenue
Detroit, MI 48207

Neighborhood Services Department
Detroit Department of Human Services
8809 John C. Lodge
Herman Keifer Hospital
Building 5
Detroit, MI 48202

New Center Community Mental Health Services
2051 West Grand Boulevard
Grand Dex Plaza
Detroit, MI 48208

North Park
1001 Puritan Street
Detroit, MI 48202

Metro Youth and Family Services Program
1249 Washington Boulevard
Book Tower, Suite 1537
Detroit, MI 48226

New Life Home for Recovering Women
17131 Gitre Street
Detroit, MI 48205

New Life Recovery, Inc.
6690 Michigan Avenue
Detroit, MI 48210

New Light Recovery Center, Inc.
300 West McNichols Street
Detroit, MI 48203

Northeast Guidance Center
Specialty Services Program
2070 Chalmers Street
Detroit, MI 48215

Northeast Health Services, Inc.
3800 Woodward Avenue
Suite 1002
Detroit, MI 48201-2030

Parkview Counseling Center
18609 West 7 Mile Road
Detroit, MI 48219

Positive Images
694 East Grand Boulevard
Detroit, MI 48207

Quality Behavioral Health, Inc.
3455 Woodward Avenue
Suite 101
Detroit, MI 48201

Renaissance Education and Training Center
18420 West McNichols Road
Detroit, MI 48219

Renaissance West Community Mental Health Chemical Dependency Service
13940 Tireman Street
Detroit, MI 48228

Sacred Heart Rehabilitation Center, Inc. Alcohol and Drug Treatment Services
220 Bagley Street
Michigan Building, Suite 1022
Detroit, MI 48201

Salvation Army Evangeline Center for Women/Children
130 West Grand Boulevard
Detroit, MI 48216

Salvation Army Harbor Light Substance Abuse Center
2643 Park Avenue
Detroit, MI 48201

Self-Help Addiction Rehab (SHAR)
1852 West Grand Boulevard
Detroit, MI 48208

Aftercare
5675 Maybury Grand Avenue
Detroit, MI 48208

Clark Center
174 South Clark Street
Detroit, MI 48209

Day Treatment
14301 Longview Street
Detroit, MI 48213

East Center
4216 McDougall Street
Detroit, MI 48207

Sobriety House, Inc.
2081 West Grand Boulevard
Detroit, MI 48208

Southwest Detroit Community Mental Health Services, Inc. Substance Abuse Services
1700 Waterman Street
Detroit, MI 48209

Star Center, Inc.
13575 Lesure Street
Detroit, MI 48227

UPC Jefferson Research Clinic
2761 East Jefferson Avenue
Detroit, MI 48207

Veterans Affairs Medical Center Chemical Dependence Treatment Services
4646 John Road
Detroit, MI 48201

Wayne County Juvenile Detention Chemical Dependency Program
1333 East Forest Street
Detroit, MI 48207

Wendie D. Lee Institute of Life Management, Inc.
11000 West McNichols Street
Suite 212
Detroit, MI 48221

DOWAGIAC

Pokagon Band of Potawatomi Indian Tribe Keepers of The Fire Substance Abuse Program
714 North Front Street
Dowagiac, MI 49047

SACSJC Myrtle Treatment Center
420 West High Street
Dowagiac, MI 49047

EAST LANSING

Gateway Community Services First Step
910 Abbott Road
Suite 100
East Lansing, MI 48823

Lansing Psychological Associates
234 Michigan Avenue
East Lansing, MI 48823

Meridian Professional Psychological Consultants
5031 Park Lake Road
East Lansing, MI 48823

Psychological Associates in Rehab
780 West Lake Lansing Road, Suite 300
East Lansing, MI 48823

Total Health Care of Michigan
2900 Hannah Boulevard Suite 200
East Lansing, MI 48823

EASTPOINTE

Eastwood Clinics
20811 Kelly Road, Suite 103
Eastpointe, MI 48021-3139

ESCANABA

Delta Menominee DHD and other Drug Services
2920 College Avenue
Delta County Service Center
Escanaba, MI 49829

Marquette Medical Center
2500 7th Avenue
South Doctors Park, Suite 102
Escanaba, MI 49829

FARMINGTON

Eastwood Center at Botsford General Hospital
28050 Grand River Avenue
Farmington, MI 48336

FARMINGTON HILLS

Broe Rehabilitation Services, Inc.
33634 West Eight Mile Road
Farmington Hills, MI 48335

Catholic Social Services of Oakland County
29475 Inkster Road
Farmington Hills, MI 48334

Chambers and Associates
32330 West Twelve Mile Road
Suite 12
Farmington Hills, MI 48334

Davis Counseling Center
37923 West 12 Mile Road
Entry A
Farmington Hills, MI 48331

Gerger Spivack and Associates
37923 West 12 Mile Road
Farmington Hills, MI 48331

Key Psychological Services
30630 12 Mile Road, Suite D
Farmington Hills, MI 48334

Oakland Family Services
23332 Orchard Lake Road
Farmington Hills, MI 48336

23450 Middlebelt Road
Farmington Hills, MI 48336

Pioneer Counseling Centers
28511 Orchard Lake Road
Suite A
Farmington Hills, MI 48334-2951

FERNDALE

Community Services of Oakland
345 East 9 Mile Road
Ferndale, MI 48220

HFHS Second Step Program
Kingswood Hospital
10300 West 8 Mile Road
Ferndale, MI 48220

FLINT

Auburn Counseling Associates
400 North Saginaw Street
Suite 300
Flint, MI 48502

Catholic Social Services
901 Chippewa Street
Flint, MI 48503

Community Recovery Services
711 North Saginaw Street
Suite 323
Flint, MI 48503

CRS at Flint Corrections Center
411 East 3 Street
Flint, MI 48503

CRS at Flint New Paths
765 East Hamilton Avenue
Flint, MI 48505

Daniels, Dan, ACSW
4511-G Miller Road
Flint, MI 48507

Dot Caring Centers, Inc.
3500-G Flushing Road, Suite 100
Flint, MI 48504

Flint Odyssey House, Inc.
1225 Martin Luther King Avenue
Flint, MI 48503

1013 Garland Street
Flint, MI 48503-1445

Genesis Regional Medical Center Addiction Treatment
2811 East Court Street
Flint, MI 48506

Hurley Mental Health Associates
1125 South Linden Road
Flint, MI 48502

Insight Recovery Center
1110 Eldon Baker Drive
Flint, MI 48507

4413-G Corunna Road
Flint, MI 48532

McLaren Behavioral Health Center
5057-G West Bristol Road
Flint, MI 48532

National Council on Alcoholism and Addictions/Greater Flint Area
202 East Boulevard Drive
Suite 310
Flint, MI 48503

Taylor Psychological Clinic
1172 Robert T Longway Street
Flint, MI 48503

Transition House, Inc.
931 Martin Luther King Boulevard
Flint, MI 48503

Woodward Counseling, Inc.
1207 North Ballenger Highway, Suite G
Flint, MI 48504

FORT GRATIOT

Blue Water Mental Health Clinic
1501 Krafft Road
Fort Gratiot, MI 48059

FRANKLIN

Beacon Hill Clinic
31000 Lahser Road, Suite 1
Franklin, MI 48025

FRASER

Oakland Psychological Clinic Substance Abuse Services
16664 15 Mile Road
Fraser, MI 48026

GARDEN CITY

Garden City Hospital
Brookfield Clinic
6245 North Inkster Road
Garden City, MI 48135

GAYLORD

Catholic Human Services, Inc. Alcohol and Drug Services
111 South Michigan Avenue
Gaylord, MI 49735

Counseling and Health Substance Abuse Services
651 North Otsego Avenue
Gaylord, MI 49735

Northern Michigan Community Mental Health Dual Diagnosis Program
800 Livingston Boulevard
Suite 2-A
Gaylord, MI 49735

GLADWIN

Human Aid, Inc. Substance Abuse Services
137 Commerce Court
Gladwin, MI 48624

GRAND BLANC

Oakland Psychological Clinic PC
8341 Office Park Drive
Grand Blanc, MI 48439

GRAND HAVEN

Child/Family Services of Western Michigan, Inc.
321 South Beechtree Street
Grand Haven, MI 49417

Ottagan Addictions Rehab, Inc.
57 Robbins Road
Grand Haven, MI 49417

GRAND RAPIDS

ACAC Inc
3949 Sparks Street SE, Suite 103
Grand Rapids, MI 49546

Advanced Therapeutics Corporation Solutions
738 Lafayette Street NE
Grand Rapids, MI 49503

Anderson Substance Abuse Treatment Center
3501 Lake Eastbrook Boulevard
Suite 120
Grand Rapids, MI 49546

Beauchamp Consulting and Associates
6159 28th Street SE, Suite 16
Grand Rapids, MI 49546-6911

Bethany Christian Services Substance Abuse Counseling Program
901 Eastern Avenue NE
Grand Rapids, MI 49503

Center for Family Recovery
4477 Cascade Road SE
Grand Rapids, MI 49546

Community Alternatives Program Project Rehab
801 College Street SE
Grand Rapids, MI 49507

Eastern Clinic
1555 Eastern Street SE
Grand Rapids, MI 49507

Family Outreach Center Outpatient Substance Abuse Counseling
1939 South Division Avenue
Grand Rapids, MI 49507

Forest View Psychiatric Hospital Dual Diagnosis Program
1055 Medical Park Drive SE
Grand Rapids, MI 49546

Fountain Hill Center for Counseling Consultation
534 Fountain Street NE
Grand Rapids, MI 49503

Grand Rapids Center for Psychotherapy
3350 Eagle Park Drive
Suite 102-B
Grand Rapids, MI 49505

Kooistra, Jansma, Teitsma, DiNallo, and Van Hoek
3330 Claystone Street SE
Grand Rapids, MI 49546

Life Guidance Services
3351 Claystone Street SE
Suite 112
Grand Rapids, MI 49546

1400 Leonard Street NE
Grand Rapids, MI 49505

Longford Care Unit of Kent Community Hospital
750 Fuller Avenue NE
Grand Rapids, MI 49503

Mel Trotter Ministries
225 Commerce Street SW
Grand Rapids, MI 49503

Montiegel and Miller Company
161 Ottawa Avenue NW
Suite 200-F
Grand Rapids, MI 49503

North Kent Guidance Services
5270 Northland Drive, Suite A
Grand Rapids, MI 49525-1040

Our Hope Association
324 Lyon Street NE
Grand Rapids, MI 49503

Pathfinder Resources, Inc.
Demey Center
245 State Street SE
Grand Rapids, MI 49503

Jellema House
523 Lyon Street NE
Grand Rapids, MI 49503

Pine Rest Christian Mental Health Services
300 68 Street SE
Grand Rapids, MI 49501

Project Rehabilitation
Adult Residential Services
200 Eastern Avenue SE
Grand Rapids, MI 49503

Community Services
Hispanic Residential Program
822 Cherry Street SE
Grand Rapids, MI 49503

Shiloh and Dakota
130 68th Street
Grand Rapids, MI 49548

Psychology Associates
1000 Parchment Street
Grand Rapids, MI 49546-3663

Reality Counseling Services
2420 Burton Street, Suite 201
Grand Rapids, MI 49546

Salvation Army Turning Point
1931 Boston Street SE
Grand Rapids, MI 49506

Wedgewood Christian Youth and Family Services, Inc.
3300 36th Street SE
Grand Rapids, MI 49512

Cutlerville Recovery
300 68th Street SE
Grand Rapids, MI 49548

West Michigan Addiction Consultants PC Professional Recovery System
3001 Fuller Ave NE
Grand Rapids, MI 49505

GRAYLING

Grace Center/Saint Francis Human Resource Center
6459 West Street, Suite M-72
Grayling, MI 49738

GREENVILLE

North Kent Guidance Services
106 South Greenville West Drive
Greenville, MI 48838

GROSSE POINTE

Eastwood Clinics Grosse Point Woods
19251 Mack Avenue
Mack Office Building Suite 300
Grosse Pointe, MI 48236

GROSSE POINTE FARMS

Vonschwarz Associates Community Resource Services
456 Touraine Street
Grosse Pointe Farms, MI 48236

GROSSE POINTE PARK

Catholic Social Services of Wayne County Substance Abuse Services
15200 East Jefferson Street
Suite 105
Grosse Pointe Park, MI 48230

HANCOCK

Christian Counseling
100 Quincy Street
Hancock, MI 49930

Marquette Medical Clinic
1045 Quincy Street
Hancock, MI 49930

Western UP District Health Department Substance Abuse Services
540 Depot Street
Hancock, MI 49930

HARRISON TOWNSHIP

Saint John Hospital/MaComb Center Chemical Dependency Unit
26755 Ballard Road
Harrison Township, MI 48045

HART

New Life Recovery and Prevention Services, Inc.
220 Washington Street
Hart, MI 49420

HARTFORD

Van Buren County Health Department Substance Abuse Services
57418 County Road 681
Hartford, MI 49057

HASTINGS

Barry County Substance Abuse Services
220 West Court Street
Hastings, MI 49058

HIGHLAND PARK

Black Family Development, Inc. Family Abstinence Commitment to Empower (FACE)
211 Glendale Street, Room 206
Riverview Medical Center
Highland Park, MI 48203

Christian Guidance Center
13220 Woodward Avenue
Highland Park, MI 48203

New Center Community Mental Health Services
211 Glendale Road, 4th Floor
Highland Park, MI 48203

New Era Alternative Treatment Center
211 Glendale Street, Suite SB
Highland Park, MI 48203

HILLSDALE

Bridgeway Center of Foote Hospital
1360 South Hillsdale Road
Hillsdale, MI 49242

Hillsdale Community Health Center
170 South Howell Street
Hillsdale, MI 49242

HOLLAND

Child/Family Services of Western Michigan, Inc. Substance Abuse Services
412 Century Lane
Holland, MI 49423

Holland Community Hospital
602 Michigan Avenue
Holland, MI 49423

Behavioral Health Services
854 South Washington Avenue
Suite 330
Holland, MI 49423-7132

Mercy Glen Family Recovery Center Substance Abuse Services
603 East 16 Street
Holland, MI 49423

Ottagan Addictions Rehab, Inc.
483 Century Lane
Holland, MI 49423

Chester A. Ray Center
231 Washington Boulevard
Holland, MI 49423

Harbor House for Women
377 Lincoln Street
Holland, MI 49423

Pine Rest Christian Mental Health Services
926 South Washington Street
Holland, MI 49423

HOLLY

Highland Waterford Center, Inc. Holly Gardens
4501 Grange Hall Road
Holly, MI 48442

North Oakland Center for Human Potential
521 East Street
Holly, MI 48442

HOLT

Child and Family Services of Michigan, Inc. Capitol Area Substance Abuse Services
4801 Willoughby Street
Suite 2
Holt, MI 48842

HOUGHTON LAKE

Human Aid, Inc. Substance Abuse Services
202 Health Parkway
Houghton Lake, MI 48617

HOWELL

Brighton Hospital Livingston Counseling and Assessment Services, Inc.
3744 East Grand River Avenue
Howell, MI 48843

McAuley/McPherson Behavioral Services
620 Byron Road, 3rd Floor
Howell, MI 48843

IONIA

Arbor Circle Corporation DBA
848 East Lincoln Avenue
Ionia, MI 48846

Inner Access Therapy Center
227 West Main Street, Suite 206
Ionia, MI 48846

IRON MOUNTAIN

Dickinson/Iron Substance Abuse Services, Inc. Outpatient
427 South Stephenson Avenue
Iron Mountain, MI 49801

Veterans' Affairs Medical Center Substance Abuse Treatment Program
325 East H Street
Iron Mountain, MI 49801

IRON RIVER

Dickinson/Iron Substance Abuse Services, Inc.
117 West Genesee Street
Iron River, MI 49935

IRONWOOD

Lutheran Social Services of Wisconsin and Upper Michigan, Inc.
Villa Manor
126 West Arch Street
Ironwood, MI 49938

JACKSON

Bridgeway Center of Foote Hospital
900 E Michigan Avenue
Jackson, MI 49201

Family Service and Children's Aid
330 West Michigan Street
Jackson, MI 49201

Michigan Therapeutic Consultants PC
605 West Michigan Avenue
Jackson, MI 49201

National Council on Alcoholism
950 West Monroe Street
Suite G-400
Jackson, MI 49202

Washington Way Recovery Center
2424 West Washington Street
Jackson, MI 49203

KALAMAZOO

Child and Family Psychological Services
5380 Holiday Terrace
Kalamazoo, MI 49009

Gateway New Beginnings KADAC Holding Company
1625 Gull Road
Kalamazoo, MI 49001

Gateway Northside Outreach Services
118 Roberson Street
Kalamazoo, MI 49007

Gateway Outpatient Services
5360 Holiday Terrace
Kalamazoo, MI 49006

Gateway Villa
1910 Shaffer Road
Kalamazoo, MI 49001

Guidance Clinic
2615 Stadium Drive
Kalamazoo, MI 49008

Kalamazoo Psychology PC
122 West South Street
Suite 207
Kalamazoo, MI 49007

New Way Counseling Center
1128 South Westnedge
Kalamazoo, MI 49008

Sandra Fields/Neal and Associates, Inc.
535 South Burdick Street
Suite 165
Kalamazoo, MI 49007-5261

Senior Services, Inc. Older Adult Recovery Program
918 Jasper Street
Kalamazoo, MI 49001

University Substance Abuse Clinic SPADA
1000 Oakland Drive
Kalamazoo, MI 49008

Victory Clinical Services
1020 South Westnedge Street
Kalamazoo, MI 49008

Western Michigan University
Substance Abuse Services
Sindecuse Health Center
Room 3235
Kalamazoo, MI 49008

Womancare, Inc.
2836 West Main Street
Kalamazoo, MI 49006

KALKASKA

Antrim Kalkaska Community Mental Health Center
509 North Birch Street
Kalkaska, MI 49646-0267

Interventions Counseling Service
556 South Cedar Street
Kalkaska, MI 49646

KENTWOOD

Pathfinders Resources, Inc. Women and Children's Center
3333 36th Street NE
Kentwood, MI 49512

KINGSFORD

Community Substance Abuse Services, Inc.
373 Woodward Avenue
Kingsford, MI 49801

LAKE ORION

Guest House
1840 West Scripps Road
Lake Orion, MI 48361

Oakland Psychological Clinic PC Substance Abuse Services
2633 South Lapeer Road
Lake Orion, MI 48360

L'ANSE

Keweenaw Bay Tribal Alcohol Program
Brewry Road, Route 2
L'Anse, MI 49946

LANSING

Comprehensive Substance Abuse Treatment
House of Commons
517 North Walnut Street
Lansing, MI 48933

Older Adult Prevention and Treatment Program
808 Southland Street, Suite A
Lansing, MI 48910

Southland Counseling Center
808 Southland Street, Suite C
Lansing, MI 48910

Cristo Rey Counseling Services Substance Abuse Program
1717 North High Street
Lansing, MI 48906

Dimensions of Life
510 West Willow Street
Lansing, MI 48906

Glass House
419 North Martin Luther King Boulevard
Lansing, MI 48915

Holden House
3300 South Pennsylvania Avenue
Lansing, MI 48910

Insight Recovery Center
2929 Covington Court
Lansing, MI 48912

Marina Levine Rehab Services
1808 South Pennsylvania Avenue, Suite C
Lansing, MI 48910

National Council on Alcoholism Lansing Regional Area
3400 South Cedar Street
Suite 200
Lansing, MI 48910

Reality Counseling Services
610 East Grand River
Lansing, MI 48906

Total Health Education, Inc.
2627 North East Street
Lansing, MI 48906

Treatment Works, Inc.
3401 East Saginaw Street
Lansing, MI 48912

LAPEER

Alcohol Information and Counseling Center
1575 Suncrest Drive
Lapeer County Health Department
Lapeer, MI 48446

Christian Family Services of Lapeer County
441 Clay Street
Lapeer, MI 48446

Completion House, Inc. DBA Turning Point
24 East Park Street
Lapeer, MI 48446

Lapeer County Community Mental Health
1570 Suncrest Drive
Lapeer, MI 48446

Lapeer Regional Hospital Vail Center
1375 North Main Street
Lapeer, MI 48446

List Psychological Services
350 North Court Street
Lapeer, MI 48446

LINCOLN PARK

Boniface Human Services
25050 West Outer Drive
Suite 201
Lincoln Park, MI 48146

Community Care Services
Counseling and Resource Center
26184 West Outer Drive
Lincoln Park, MI 48146

LIVONIA

Arbor Hills Medical Center
27550 Joy Road
Livonia, MI 48150

Butterfly Center The Recovery Corporation
27485 5 Mile Road
Livonia, MI 48154

Catholic Social Service of Wayne County
17316 Farmington Road
Livonia, MI 48152

Eastwood Clinics
17250 Farmington Road
Livonia, MI 48154

Employee Assistance Associates, Inc.
38705 7 Mile Road
Suite 130
Livonia, MI 48152

Family Services, Inc.
16755 Middlebelt Road
Livonia, MI 48154

Hegira Programs, Inc. Livonia Counseling Center
13325 Farmington Road
Livonia, MI 48150

New Directions Center for Christian Counseling
37625 Ann Arbor RoadSuite 107
Livonia, MI 48150

Oakland Psychological Clinic Substance Abuse Services
29865 6 Mile Road, Suite 112
Livonia, MI 48152

Pioneer Counseling Centers
37650 Professional Center Drive
Suite 145-A
Livonia, MI 48154

Saint Mary Hospital Chemical Dependency Services
36475 5 Mile Road
Livonia, MI 48154

University Psychiatric Center Livonia Substance Abuse Program
16832 Newburgh Road
Livonia, MI 48154

LUDINGTON

New Life Recovery and Prevention Services
1105 South Washington Street
Ludington, MI 49431

MADISON HEIGHTS

Gateway Counseling Center
27301 Dequindre Road
Madison Heights, MI 48071

Medical Resource Center, Inc.
1400 East 12 Mile Road
Madison Heights, MI 48071

MANISTEE

Manistee/Benzie Community Mental Health Counseling Center
395 3rd Street
Manistee, MI 49660

MANISTIQUE

Hiawatha Behavioral Health Authority
125 North Lake Street
Manistique, MI 49854

LMAS Addiction Services
300 Walnut Street
Manistique, MI 49854

MARLETTE

Family Resource Counseling and Learning Center, Inc.
6444 Morris Street
Marlette, MI 48453

MARQUETTE

Bell Behavioral Services
425 Corning Street, Suite B
Marquette, MI 49855

Great Lakes Recovery Center, Inc.
241 Wright Street
Marquette, MI 49855

228 West Washington Street
Suite 3
Marquette, MI 49855

Lutheran Social Services of Wisconsin and Upper Michigan
1009 West Ridge Street
Marquette, MI 49855

Marquette General Hospital Addiction Services
420 West Magnetic Street
Marquette, MI 49855

MARYSVILLE

Eastern Michigan Counseling Associates
1600 Gratiot Boulevard
Building A, Suite 3
Marysville, MI 48040

MASON

Correctional Assessment and Treatment Services Comp. Substance Abuse Treatment Program
630 North Cedar Street
Ingham County Jail
Mason, MI 48854

MEMPHIS

Sacred Heart Rehabilitation Center, Inc.
400 Stoddard Road
Memphis, MI 48041

MENOMINEE

Beacon/Bay Area Program for Behavioral Medicine
1110 10th Avenue
Menominee, MI 49858

Delta Menominee District Health Department
2608 10th Street
Menominee, MI 49858

MIDLAND

Family and Children's Services of Midland
1714 Eastman Avenue
Midland, MI 48641

Focus Substance Abuse Counseling and Information Service
4604 North Saginaw Road
Suite C
Midland, MI 48640

H. G. Swift Counseling Services
5100 Eastman Avenue, Suite 2
Midland, MI 48640

Ten Sixteen Treatment Center
1016 Eastman Avenue
Midland, MI 48640

MILFORD

Oakland Psychological Clinic PC Substance Abuse Services
1203 North Milford Road
Suite A
Milford, MI 48381

MIO

Ausable Valley Community Mental Health Substance Abuse Services
325 North Mount Tom Road
Mio, MI 48647

MONROE

Catholic Social Services of Monroe County
16 East 5th Street
Monroe, MI 48161

Substance Abuse Services
123 West First Street
Gateway Building
Monroe, MI 48161

Eastwood Clinics
708 South Monroe Street
Monroe, MI 48161-2126

Mercy Memorial Hospital
Family Center
700 Stewart Road
Monroe, MI 48162

Substance Abuse Services
718 North Macomb Street
Monroe, MI 48162

Monroe County Jail Substance Abuse Education and Counseling Program
100 East 2 Street
Monroe, MI 48163

Salvation Army Harbor Light
Monroe County Alcohol Center
3580 South Custer Road
Monroe, MI 48161

Monroe County Center
25 South Monroe Street
Monroe, MI 48161

Vets Incorporated
14 South Monroe Street
Monroe, MI 48161

MOUNT CLEMENS

Clinton Counseling Center
Comprehensive Youth Services
2 Crocker Boulevard
Suite 101
Mount Clemens, MI 48043

43565 Elizabeth Road
Mount Clemens, MI 48043

Macomb Family Services Inc I
2 Crocker Boulevard
Suite 202
Mount Clemens, MI 48043

New Beginnings Counseling
39 B Crocker Boulevard
Mount Clemens, MI 48043

MOUNT PLEASANT

Choices of Mount Pleasant, Inc.
1234 East Broomfield Road
Building A, Suite 5
Mount Pleasant, MI 48858

Mount Pleasant Counseling Services
3480 South Isabella Road
Mount Pleasant, MI 48858

OJIBWE Substance Abuse Program
2250 Enterprise Drive
Mount Pleasant, MI 48855

Omega Counseling Centers
105 South Franklin Street
Suite 221
Mount Pleasant, MI 48858

MUSKEGON

Child and Family Services
1352 Terrace Street
Muskegon, MI 49442

Mercy Counseling and Recovery Center
1771 Wells Street
Muskegon, MI 49442

West Michigan Therapy, Inc.
130 East Apple Avenue
Muskegon, MI 49442

MUSKEGON HEIGHTS

East Side Substance Abuse Clinic
445 East Sherman Boulevard
Muskegon Heights, MI 49444

NEW BALTIMORE

Harbor Oak Hospital/Pioneer Health Care
35031 23 Mile Road
New Baltimore, MI 48047

Personal Home Care Services, Inc. Center for Counseling
32743 23 Mile Road
New Baltimore, MI 48047-1985

Self and Others
33497 23 Mile Road, Suite 130
New Baltimore, MI 48047

NEWBERRY

LMAS Addiction Services
County Road 428
Hamilton Lake Road
Newberry, MI 49868

NEW HAVEN

Community Human Services, Inc.
57737 Gratiot Avenue
New Haven, MI 48048

NILES

Addiction Recovery Centers, Inc.
306 East Main Street
Niles, MI 49120

Lakeland KADAC Holding Co.
1209 South 11th Street, Unit 14
Niles, MI 49120

NORTHVILLE

Hegira Programs, Inc. Northville Counseling Center
115 North Center Street
Suite 202
Northville, MI 48167

Northville Psychiatric Hospital
41001 West Seven Mile Road
Northville, MI 48167

NORTON SHORES

Alcohol and Chemical Abuse Consultants, Inc.
427 Seminole Street
Norton Shores, MI 49441

NOVI

Insight Recovery Center
24230 Karim Boulevard
Suite 303
Novi, MI 48375

Orchard Hills Psychiatric Center Substance Abuse Services
40000 Grand River, Suite 306
Novi, MI 48375

Saint Joseph Mercy Hospital
39575 West Ten Mile Road
Suite 202
Novi, MI 48375

OAK PARK

Lutheran Child and Family Services of Michigan Substance Abuse Services
15160 West Eight Mile Road
Oak Park, MI 48237

Metropolitan Rehabilitation Clinics
21700 Greenfield Street
Suite 130
Oak Park, MI 48237

OSCODA

Birchwood Center for Chemical Dependency
5671 Skeel Avenue
Oscoda, MI 48750

OTTER LAKE

Turning Point Recovery Center Otter Lake Residential Unit
6727 Sherman Drive
Otter Lake, MI 48464

OWOSSO

Catholic Social Services
120 West Exchange Street
Suite 204
Owosso, MI 48867

Memorial Healthcare Plus Positive Alts Counseling/ Education
1488 North M-52
Owosso, MI 48867

PAW PAW

Gateway
181 West Michigan Street
Paw Paw, MI 49079

New Journey Substance Abuse Program
410 East Michigan Street
Paw Paw, MI 49079

PETOSKEY

CHIP Counseling Center
2503 Charlevoix Avenue
Petoskey, MI 49770

Harbor Hall
704 Emmet Street
Petoskey, MI 49770

Little Traverse Bay Bands of Odawa Indians Substance Abuse Programs
1345 U.S. 31 North
Petoskey, MI 49770

Northern Michigan Community Mental Health Dual Diagnosis Program
1 MacDonald Drive, Suite B
Petoskey, MI 49770

Northern Michigan Hospitals Harbor Hall Outpatient Substance Abuse Program
820 Arlington Street
Petoskey, MI 49770

Women's Resource Center of Northern Michigan, Inc.
423 Porter Street
Petoskey, MI 49770

PLAINWELL

Pathways Psychological Associates
112 East Chart Street
Plainwell, MI 49080

PLYMOUTH

Growth Works Counseling and Intervention Services
271 South Main Street
Plymouth, MI 48170

Orchard Hills Psychiatric Center
199 North Main Street, Suite 202
Plymouth, MI 48170

Personalized Nursing Light House, Inc.
575 Main Street, Suite 6
Plymouth, MI 48170

PONTIAC

Catholic Social Services of Oakland County
53 Franklin Boulevard
Pontiac, MI 48341

El Centro La Familia
35 West Huron Street, Suite 200
Pontiac, MI 48342

Mercy Network Central
35 West Huron Street
Pontiac, MI 48342

Oakland Family Services Substance Abuse Services
114 Orchard Lake Road
Pontiac, MI 48341

Parkview Company Counseling Center
989 University Drive, Suite 2
Pontiac, MI 48342

Pontiac General Hospital and Medical Center North Oakland Medical Center
461 West Huron Street
Pontiac, MI 48341-1651

Procare at Pontiac Osteopathic Hospital
24 East Huron Street
Pontiac, MI 48342

Chemical Dependency Unit
50 North Perry Street
Pontiac, MI 48058

Residential Unit
16 1/2 East Huron Street
Pontiac, MI 48342

Saint Joseph Mercy Hospital
900 Woodward Avenue
Pontiac, MI 48341

Sequoia Recovery Services
363 West Huron Street
Pontiac, MI 48341

Turning Point Recovery Center
Completion House
54 Seneca Street
Pontiac, MI 48342-2349

University Unit/Outpatient Counseling
131 University Drive
Pontiac, MI 48342

Woodward Counseling, Inc.
35 South Johnson Street
Suite 3D
Pontiac, MI 48341

PORTAGE

Mid-America Psychological Services
8036 Moorsbridge Road
Portage, MI 49024

PORT HURON

Blue Lake Residential Care Facilities Clearview Substance Abuse Services
1406 8th Street
Port Huron, MI 48060

Catholic Social Services of Saint Clair County/Substance Abuse Services
2601 13th Street
Port Huron, MI 48060

Center for Human Resources Military Street
1001 Military Street
Port Huron, MI 48060

Cornell Center
1025 Court Street
Port Huron, MI 48060

Professional Counseling Center PC
520 Superior Street
Port Huron, MI 48060

REDFORD

Botsford Family Service Center Substance Abuse Services
26905 Grand River Avenue
Redford, MI 48240

Redford Counseling Center
25945 West 7 Mile Road
Redford, MI 48240

REED CITY

Human Aid, Inc. Substance Abuse Services
834 South Chestnut Street
Reed City, MI 49677

RICHMOND

MaComb Family Services, Inc.
67515 Main Street, Suite C
Richmond, MI 48062

ROCHESTER HILLS

Eastwood Clinics
725 Barclay Circle Drive
Suite 215
Rochester Hills, MI 48307-4512

Oakland Family Services
1460 Walton Boulevard, Suite 220
Rochester Hills, MI 48309

ROMEO

Community Human Services, Inc.
332 South Main Street
Romeo, MI 48065

ROMULUS

Hegira Programs, Inc. Romulus Help Center
9340 Wayne Road
Suite A
Romulus, MI 48174

Transitions of Michigan
9844 Harrison Road
Romulus, MI 48174

ROSEVILLE

Parkview Counseling Center
27115 Gratiot Street
Roseville, MI 48066

ROYAL OAK

Catholic Social Services of Oakland County/Talbott Center
1424 East 11 Mile Road
Royal Oak, MI 48067

Eastwood Community Clinics
30701 North Woodward Avenue
Suite 200
Royal Oak, MI 48073

Residential Substance Abuse Treatment Program
1515 North Stephenson Highway
Royal Oak, MI 48067

SAGINAW

Aleda E. Lutz VA Medical Center
1500 Weiss Street
Saginaw, MI 48602

American Comprehensive Treatment Services, Inc.
1527 South Washington Street
Saginaw, MI 48601

Arete Community Treatment Centers
709 Lapeer Street
Saginaw, MI 48607

Boysville of Michigan, Inc. Holland House
614 East Holland Avenue
Saginaw, MI 48601

Catholic Family Service Family Counseling Services
710 North Michigan Avenue
Saginaw, MI 48602

Dot Caring Centers, Inc.
Halfway House/Residential Center
1915 Fordney Street
Saginaw, MI 48601

Saginaw Valley Center
3190 Hallmark Court
Saginaw, MI 48603

Health Source Saginaw Pathway Chemical Dependency Services
3340 Hospital Road
Saginaw, MI 48603

Insight Recovery Center
3216 Christy Way
Saginaw, MI 48602

Intervention and Rehab Associates, Inc.
1616 Court Street
Saginaw, MI 48602

Restoration Community Outreach
1205 Norman Street
Saginaw, MI 48601

Saginaw Odyssey House
128 North Warren Street
Saginaw, MI 48607

Saginaw Psychological Services, Inc.
2100 Hemmeter Street
Saginaw, MI 48603

Samaritan Counseling Center of Saginaw Valley
2405 Bay Street
Faith Lutheran Church
Saginaw, MI 48602

STM Clinic Mental Health and Substance Abuse Services
1 Tuscola Street, Suite 302
Saginaw, MI 48607

SAINT CLAIR SHORES

Cube
22811 Greater Mack Avenue
Hampton Square Building
Suite 107
Saint Clair Shores, MI 48080

Down River Mental Health Clinic Advanced Counseling Services
19501 East Eight Mile Road
Saint Clair Shores, MI 48080

Henry Ford Health Systems Behavioral Services
21603 Eleven Mile Road, Suite 1
Saint Clair Shores, MI 48081

Pro Med Management Evergreen Counseling Centers
19900 10 Mile Road
Saint Clair Shores, MI 48081

SAINT IGNACE

American Indian Substance Abuse Program
225 Waseh Drive
Saint Ignace, MI 49781

LMAS Health Department Substance Abuse Program/ Mackinac County
749 Hombach Street
Saint Ignace, MI 49781

SAINT JOHNS

Clinton County Counseling Center
1000 East Sturgis Street
Saint Johns, MI 48879

SAINT JOSEPH

Kadac Hold Gateway Services
1234 Napier Avenue
Saint Joseph, MI 49085

SAINT LOUIS

Recovery Unlimited
215 West Saginaw Street
Saint Louis, MI 48880

SANDUSKY

Sanilac County Health Department Alcohol and Drug Program
171 Dawson Street
Sandusky, MI 48471

SAULT SAINTE MARIE

American Indian Substance Abuse Program
2154 Shunk Road
Sault Sainte Marie, MI 49783

Great Lakes Recovery Center, Inc.
New Hope House/Men
301 East Spruce Street
Sault Sainte Marie, MI 49783

New Hope House/Women
1111 Minneapolis Street
Sault Sainte Marie, MI 49783

Upper Michigan Behavioral Health Services
500 Osborne Boulevard
Sault Sainte Marie, MI 49783

SHELBY TOWNSHIP

Devon Center
52188 Van Dyke Street, Suite 320
Shelby Township, MI 48316-1863

MaComb Family Services
45445 Mound Road Suite 109
Shelby Township, MI 48316

Pro Med Management Evergreen Counseling Centers
53950 Van Dyke Street
Shelby Township, MI 48087

SOUTHFIELD

Burdette and Doss Associates Psychological Services
17352 West 12 Mile Road
Suite 100
Southfield, MI 48076

Central Therapeutic Services, Inc.
17600 West 8 Mile Road
Suite 7
Southfield, MI 48075

Clark and Associates Psychological Services
16250 Northland Drive
Suite 245
Southfield, MI 48075

Counseling Associates
26699 West 12 Mile Road
Suite 100
Southfield, MI 48034

Family Service of Detroit and Wayne Counties
15565 Northland Street
Suite 505 West
Southfield, MI 48075

Oakland Psychological Clinic PC Substance Abuse Services
21700 Northwestern Highway
Suite 750
Southfield, MI 48075

Pathway Family Center
22190 Providence Road Suite 300
Southfield, MI 48075

Providence Hospital and Medical Center
16001 West 9 Mile Road
Southfield, MI 48037

Wedgewood Christian Counseling Center
17117 West 9 Mile Road
Suite 1325
Southfield, MI 48075

SOUTHGATE

Downriver Guidance Clinic
131010 Allen Road
Southgate, MI 48195

Family Services, Inc
13331 Reeck Road
Southgate, MI 48195

SOUTH HAVEN

Black River Counseling Group
352 Blue Star Highway
South Haven, MI 49090

New Journey Substance Abuse Program
300 Kalamazoo Street
South Haven, MI 49090

STANTON

Omega Counseling Centers
111 East Main Street, Suite B
Stanton, MI 48888

STERLING

Sterling Area Health Center
725 East State Street
Sterling, MI 48659

STERLING HEIGHTS

Crossroads Counseling Center
38850 Van Dyke Street, Suite 102
Sterling Heights, MI 48312

Pro Med Management Evergreen Counseling Centers
33200 Dequindre Road
Suite 200
Sterling Heights, MI 48310

Pioneer Counseling Center
36250 Dequindre Road
Suite 310
Sterling Heights, MI 48310

Professional Counseling Associates
36250 Dequindre Road, Suite 320
Sterling Heights, MI 48310

STURGIS

Michiana Addiction and Prevention Services
300 West Chicago Street
Suite 1212
Sturgis, MI 49091

SUTTONS BAY

Grand Traverse Band of Ottawa Chippewa Indians Substance Abuse Services
2300 North Stallman Road
Suttons Bay, MI 49682

TAWAS CITY

Ausable Valley Community Mental Health Substance Abuse Services
1199 West Harris Avenue
Tawas City, MI 48763

TAYLOR

Community Care Services Substance Abuse Service
26650 Eureka Road
Taylor, MI 48180

Downriver Mental Health/ Advanced Psychiatric Services Chemical Dependency Program
20600 Eureka Road, Suite 819
Taylor, MI 48180

TECUMSEH

Sage Center for Substance Abuse Treatment at Bixby Medical Center
415 East Kilbuck Street
Tecumseh, MI 49286

TEMPERANCE

Catholic Social Services of Monroe County
8330 Lewis Street
Temperance, MI 48182

THREE RIVERS

Michiana Addiction and Prevention Services
222 South Main Street
Three Rivers, MI 49093

TRAVERSE CITY

Addiction Treatment Services Inc
940 East 8th Street
Traverse City, MI 49686

Bay Area Counseling
2226 South Airport Road West
Suite C
Traverse City, MI 49684

Catholic Human Services
1000 Hastings Street
Traverse City, MI 49686

Charles Bethea Associates
2046B South Airport Road
Traverse City, MI 49684

Grand Traverse Band of Ottawa/Chippewa Indians Substance Abuse Services
940 East 8th Street
Traverse City, MI 49684

Great Lakes Community Health
701 South Elmwood Street
Suite 19
Traverse City, MI 49684

Rubritius, Jeffrey W., MSW
13685 Southwest Bay Shore Drive
Suite 106-W
Traverse City, MI 49684

Munson Medical Center Alcohol and Drug Treatment Center
1105 6 Street
Traverse City, MI 49684

Northern Michigan Alcoholism and Addiction Treatment Services, Inc.
116 East 8 Street
Traverse City, MI 49684

Phoenix Hall
445 East State Street
Traverse City, MI 49684

Wedgewood Christian Counseling Center
3301 Veterans Drive
Suite 125
Traverse City, MI 49684

TRENTON

Oakwood Healthcare Systems
5450 Fort Street
Trenton, MI 48183

TROY

Insight
631 East Big Beaver Road
Suite 111
Troy, MI 48083

Perspectives of Troy PC
2690 Crooks Road
Suite 300
Troy, MI 48084

Rivers Bend PC
33975 Dequindre Street, Suite 5
Troy, MI 48083

WAKEFIELD

Gogebic Community Mental Health
103 West U.S. 2
Wakefield, MI 49968

WALKER

Northwest Counseling Center
3755 Remembrance Road NW
Walker, MI 49504

WALLED LAKE

Oakland Family Services
2045 West Maple Road
Suite D 405
Walled Lake, MI 48088

WARREN

Catholic Social Services of Macomb Substance Abuse Program
12434 East 12 Mile Road
Suite 201
Warren, MI 48093

Harper/Warren Chemical Dependency Program
4050 East 12 Mile Road
Warren, MI 48092

Horizon Health System Community Hospital
26091 Sherwood Street, Suite 4-A
Warren, MI 48091-1296

Medical Resource Center, Inc. Michigan Counseling Services
23700 Van Dyke Avenue
Warren, MI 48089

Michigan Psychological Center, Inc.
26451 Ryan Road
Warren, MI 48091

Sacred Heart Rehabilitation Center, Inc.
28573 Schoenherr Street
Warren, MI 48093

WATERFORD

Catholic Social Services of Oakland County/Waterford
6637 Highland Road
Waterford, MI 48327

Community Programs, Inc.
1435 North Oakland Boulevard
Waterford, MI 48327

Perfect Solutions, Inc.
2710 Dixie Highway, Suite C
Waterford, MI 48328-1711

WATERSMEET

Lac Vieux Desert Substance Abuse Program
Choate Road
Watersmeet, MI 49969

WEST BLOOMFIELD

Affordable Counseling
5745 West Maple Road
Suite 207
West Bloomfield, MI 48322

Henry Ford Maplegrove Center Behavioral Services
6773 West Maple Road
Maplegrove Center
West Bloomfield, MI 48322

New Oakland Child and Adolescent Family Center
5600 West Maple Road
Suite D-402
West Bloomfield, MI 48322

New Start, Inc.
5839 West Maple Road
Suite 112
West Bloomfield, MI 48322

WEST BRANCH

Ausable Valley Comm. Mental Health Center
Substance Abuse Program
511 Griffin Street
West Branch, MI 48661

Substance Acute Detox
403 East Houghton Avenue
West Branch, MI 48661

WESTLAND

Hegira Programs, Inc. Westland Counseling Center
8623 North Wayne Street
Suite 310
Westland, MI 48185

Oakwood Healthcare Systems
2001 South Merriman Road, Suite 500
Westland, MI 48186

Pro Med Management Evergreen Counseling Centers
8623 North Wayne Road
Suite 200
Westland, MI 48185

WETMORE

LMAS Addiction Services Alger County
9526 Prospect Avenue
Wetmore, MI 49895

WILSON

Hannahville Health Center
14925-N Hannahville Road
Suite B-1
Wilson, MI 49896

WYANDOTTE

Henry Ford Wyandotte Hospital
2333 Biddle Avenue
Wyandotte, MI 48192

Wyandotte Health Center Substance Abuse Services
1622 Eureka Street
Wyandotte, MI 48192

YPSILANTI

Beyer Hospital Chemical Dependency Services
135 South Prospect Street
Ypsilanti, MI 48198

Christine Morgan and Therry Ministering Center
948 Watling Boulevard
Ypsilanti, MI 48197

Dawn, Inc. Dawn Farm
6633 Stony Creek Road
Ypsilanti, MI 48197

MINNESOTA

AH-GWAH-CHING

Lakeside Center Chemical Dependency Services
723 Ah-Gwah-Ching Road
Ah-Gwah-Ching, MN 56430

AITKEN

Northland Counseling Center
936 2nd Street NW
Aitkin, MN 56431-1104

ALBERT LEA

Fountain Lake Treatment Center
408 Fountain Street
Albert Lea, MN 56007

ALEXANDRIA

Douglas County Hospital Chemical Dependency Unit
700 Cedar Street
Marian Building, Suite 154
Alexandria, MN 56308

ANOKA

Anoka/Metro Regional Treatment Center
3300 4 Avenue North
Anoka, MN 55303

Riverplace Counseling Center
1814 South Ferry Street
Anoka, MN 55303

Transformation House
1410 South Ferry Street
Anoka, MN 55303

Transformation House II
2532 North Ferry Street
Anoka, MN 55303

AUSTIN

Agape Halfway House, Inc.
200 5 Street SW
Austin, MN 55912

Austin Med Center Behavioral Health Center Chemical Dependency Services
101 14th Street NW, Suite 4
Austin, MN 55912

BARNESVILLE

Red River Serenity Manor, Inc.
123 2 Street NE
Barnesville, MN 56514

BEMIDJI

Counseling Associates of Bemidji
3217 Bemidji Avenue North
Bemidji, MN 56601

Lakes Region Chemical Dependency
1411 Bemidji Avenue
Bemidji, MN 56601

Upper MS Mental Health Center Program for Addictions Recovery
722 15 Street
Bemidji, MN 56601

BRAINERD

Adapt of Minnesota
510 Bluff Avenue
Brainerd, MN 56401

Brainerd Regional Human Services Center Aurora Chemical Dependency Program
1777 Highway 18 East
Brainerd, MN 56401

Break Free Adolescent Outpatient
2801 Andrew Street
Brainerd, MN 56401

Saint Joseph's Medical Center Focus Unit
523 North 3 Street
Brainerd, MN 56401

BRECKENRIDGE

Saint Francis Medical Center Hope Unit
401 Oak Street
Breckenridge, MN 56520

BROOKLYN CENTER

Allina Behavioral Health Services Brooklyn Center Program
6200 Shingle Creek Parkway
Brookdale Corporate Center
Suite 480
Brooklyn Center, MN 55430

BUFFALO

Central Minnesota Mental Health Center
105 2nd Avenue NE
Buffalo, MN 55313

Professional Counseling Center of Buffalo
Wright One Plaza
Highway 55 West
Buffalo, MN 55313

BURNSVILLE

Fairview Ridges Hospital
Adult Chemical Program
156 Cobblestone Lane
Burnsville, MN 55337

River Ridge Nonresidential Treatment Center
1515 East Highway 13
Burnsville, MN 55337

Riverside Medical Center
1510 East 122nd Street
Burnsville, MN 55337

CAMBRIDGE

Cambridge Memorial Hospital Dellwood Recovery Center
701 South Dellwood Avenue
Cambridge, MN 55008

CASS LAKE

Ahnji-Be-Mah-Diz Center Leech Lake Halfway House
421 3rd Street NE
Cass Lake, MN 56633

CENTER CITY

Hazelden Foundation
15245 Pleasant Valley Road
Center City, MN 55012

CHASKA

Stafford CD Treatment Center, Inc.
212 Walnut Street
Chaska, MN 55318

CLOQUET

Liberalis Womens Program
512 Skyline Boulevard
Cloquet, MN 55720

COTTAGE GROVE

Anthony Lewis Center
7064 West Point Douglas Road
Suite 102
Cottage Grove, MN 55016

CROOKSTON

Glenmore Recovery Center
323 South Minnesota Street
Crookston, MN 56716

DELANO

Professional Counseling Center of Delano
500 Highway 12
Delano, MN 55328

DETROIT LAKES

Glenmore Clinic Outpatient Program
714 Lake Avenue
Detroit Lakes, MN 56501

Lakes Counseling Center
211 West Holmes Street
Detroit Lakes, MN 56501

DULUTH

Equay-Say-Way Treatment Center
205 West 2nd Street, Suite 150
Duluth, MN 55802

Marty Mann Halfway House
714 North 11 Avenue East
Duluth, MN 55805

Messabi Work Release Program
23 Mesaba Avenue
Duluth, MN 55806

Miller Dawn Medical Center, Inc. Chemical Dependency
502 East 2nd Street
Duluth, MN 55805

Port Rehabilitation Center
23 Mesaba Avenue
Duluth, MN 55806

Pride Institute Outpatient Program
205 West 2nd Street, Suite 448
Duluth, MN 55801

Thunderbird and Wren Halfway House
229 North 4 Avenue West
Duluth, MN 55806

EAGAN

Twin Town Treatment Center Eagan Outpatient
2121 Cliff Drive, Suite 101
Eagan, MN 55122

EAST GRAND FORKS

Northwest Recovery Center, Inc.
910 Central Avenue
East Grand Forks, MN 56721

EDEN PRAIRIE

Pride Institute
14400 Martin Drive
Eden Prairie, MN 55344

Regents Hospital/New Connections Programs Eden Prairie Outpatient Treatment
6446 City West Parkway
Suite 205
Eden Prairie, MN 55344

EDINA

Allina Behavioral Health Services
3400 West 66th Street
Southdale Place, Suite 385
Edina, MN 55435

Fairview Recovery Services
Chemical Dependency Treatment Program
3101 West 69 Street
Edina, MN 55435

Outpatient
Services
7600 France Avenue
Edina, MN 55435

ELY

Arrowhead Center, Inc.
118 South 4 Avenue East
Ely, MN 55731

FAIRMONT

Chain of Lakes Behavioral Health Services Inc
Rural Route 1
Fairmont, MN 56031

Sunrise Recovery Center
Rural Route 1
Fairmont, MN 56031

FARIBAULT

Faribault Family Focus
303 NE 1st Ave
Suite 110
Faribault, MN 55021

New Dimensions Program
1101 Linden Lane
Faribault, MN 55021-6400

FARMINGTON

Journey Counseling Services
209 Oak Street
Farmington, MN 55024

FERGUS FALLS

Fergus Falls Regional Treatment Center Chemical Dependency Services
1400 North Union Avenue
Fergus Falls, MN 56537

Lakeland Mental Health Center, Inc. Chemical Dependency Outpatient Program
126 East Alcott Avenue
Fergus Falls, MN 56537

Lakes Region Halfway House
217 North Union Avenue
Fergus Falls, MN 56537

FOREST LAKE

Fairview Recovery Services
Adolescent Residential Treatment Program
246 11th Avenue SE
Forest Lake, MN 55025

Outpatient Program
1120 SE 4th Street
Forest Lake, MN 55025

FRIDLEY

Transformation House I
351-7 4th Avenue NE
Fridley, MN 55432

GRAND MARAIS

Cook County Social Services North Shore Chemical Dependency Outpatient Program
Arrowhead Professional Building
Grand Marais, MN 55604

GRAND PORTAGE

Grand Portage Chemical Dependency Services
Grand Portage, MN 55605

GRAND RAPIDS

Hope House of Itasca County
604 South Pokegama Avenue
Grand Rapids, MN 55744

North Homes, Inc. Adolescent Outpatient Program
924 County Home Road
Grand Rapids, MN 55744

Northland Recovery Center Substance Abuse Services
1215 7 Avenue SE
Grand Rapids, MN 55744

Rapids Counseling Services, Inc.
717 NE 4th Street
Grand Rapids, MN 55744

GRANITE FALLS

Project Turnabout
660 18 Street
Granite Falls, MN 56241

HASTINGS

Cochran Programs
1200 East 18th Street, Building 4
Hastings, MN 55033

Dakota County Receiving Center
1200 East 18 Street, Building 1
Hastings, MN 55033

Twin Town Dakota County Jail Program Dakota County Workhouse
1580 West Highway 55
Hastings, MN 55033

HIBBING

University Medical Center Mesabi Outpatient
750 34th Street East
Hibbing, MN 55746

HOPKINS

Omegon Inc
2000 Hopkins Crossroads
Hopkins, MN 55305

HUTCHINSON

Hutchinson Community Hospital
Outpatient Chemical Dependency Program
Hutchinson Receiving Center
1095 Highway 15 South
Hutchinson, MN 55350

INTERNATIONAL FALLS

Northland Counseling Center
1404 Highway 71
International Falls, MN 56649

Rational Alternatives, Inc.
206 14 Street East
International Falls, MN 56649

JACKSON

Ashley House, Inc. DBA Road to Recovery
308 West Ashley Street
Jackson, MN 56143

LA CRESCENT

Counseling Clinic of La Crescent
33 South Walnut Street
La Crescent, MN 55947

LITCHFIELD

Charter Behavioral Health Systems of Litchfield
114 North Holcombe Street
Litchfield, MN 55355

LITTLE FALLS

Effective Living Center, Inc.
72 East Broadway
Little Falls, MN 56345

Saint Gabriel's Hospital Chemical Dependency Unit
815 SE 2 Street
Little Falls, MN 56345

LITTLEFORK

Pineview Recovery Center
912 Main Street
Littlefork, MN 56653

LORETTO

Vinland National Center
Lake Independence
Loretto, MN 55357

LUVERNE

Southwestern Mental Health Center
2 Round Wind Road
Luverne, MN 56156

MAHNOMEN

Mahnomen County Human Services Outpatient Treatment
311 North Main Street
Mahnomen, MN 56557

MANKATO

Addictions Recovery Technologies
12 Civic Center Plaza
Suite 2116
Mankato, MN 56001

House of Hope
119 Fulton Street
Mankato, MN 56001

Immanuel/Saint Joseph's Hospital Family Recovery Program
1025 Marsh Street
5th Floor
Mankato, MN 56001

MAPLE LAKE

Maple Lake Recovery Center
207 Division Street
Maple Lake, MN 55358

MARSHALL

Project Turnabout
1220 Birch Street
Marshall, MN 56258

MINNEAPOLIS

African American Family Services
2616 Nicollet Avenue South
Minneapolis, MN 55408

Allina Behavioral Health Services
825 Nicollet Avenue, Suite 1020
Minneapolis, MN 55402-2614

American Indian Services, Inc.
2200 Park Avenue South
Minneapolis, MN 55404

Anthony Louis Center
1000 Paul Parkway
Minneapolis, MN 55434

Bridgeway Treatment Center
22 27 Avenue SE
Minneapolis, MN 55414

Changing Lifestyle Counseling of Saint Louis Park
7515 Wayzata Boulevard
Suite 202
Minneapolis, MN 55426

Chemical Health Advisory Services, Inc. DBA Basics
2415 Emerson Avenue South
Minneapolis, MN 55405

Chicanos Latinos Unidos En Servicio
2110 Nicollet Avenue
Minneapolis, MN 55404-2528

Chrysalis Center for Women
2650 Nicollet Avenue South
Minneapolis, MN 55408

Community Health and Human Services
4149 Lyndale Avenue North
Minneapolis, MN 55412

Community University Health Care Center Southeast Asian Outpatient Program
2001 Bloomington Avenue South
Minneapolis, MN 55404

Create, Inc.
1911 Pleasant Avenue
Minneapolis, MN 55403

Telesis
1345 Shenandoah Lane
Hennepin County Adult Workhouse
Minneapolis, MN 55447

Eden Programs, Inc.
Eden Day Mens Program
1025 Portland Avenue South
Minneapolis, MN 55404

Eden Renew Outpatient Program
Eden Women's Program
2649 Park Avenue South
Minneapolis, MN 55407

Fairview Recovery Services
Adolescent Program
Adult Inpatient Program
Hearing Impaired Chemical Dependency Program
2450 Riverside Avenue
Minneapolis, MN 55454

Adolescent Outpatient
2960 Winnetka Avenue North
Suite 101
Minneapolis, MN 55427

Hazelden Center for Youth and Families
11505 36th Avenue North
Minneapolis, MN 55441

Health Recovery Center, Inc.
3255 Hennepin Avenue South
Minneapolis, MN 55408

HFA Addiction Medicine
914 South 8th Street, Suite D-131
Minneapolis, MN 55404

Intervention Institute
349 13th Avenue NE
Minneapolis, MN 55413

Lifestyle Counseling of Richfield/Bloomington
9607 Girard Avenue South
Minneapolis, MN 55431

Living Word Recovery Services
7308 Aspen Lane, Suite 153-A
Minneapolis, MN 55428

Minnesota Indian Women's Resource Center
2300 15 Avenue South
Minneapolis, MN 55404

Minneapolis Psychiatric Institute Abbott/Northwestern Hospital Campus
800 East 28th Street
Wasie Center, 4th Floor
Minneapolis, MN 55407-3799

Mission Care Detox Center
3409 East Medicine Lake Boulevard
Minneapolis, MN 55441

My Home/Excelsior Project Inc Outpatient Program
2344 Nicollet Avenue South
Suite 20
Minneapolis, MN 55401

New Connection Programs Blaine Outpatient Treatment
10267 University Avenue NE
Minneapolis, MN 55434

Nuway House II
2518 First Avenue South
Minneapolis, MN 55404

On Belay House
115 Forestview Lane North
Minneapolis, MN 55441-5910

Park Avenue Center
2525 Park Avenue
Minneapolis, MN 55404

Pathways Psychological Services Outpatient Program
7575 Golden Valley Road
Suite 119
Minneapolis, MN 55427

Pride Institute Outpatient Program
1406 West Lake Street, Suite 204
Minneapolis, MN 55408

Prodigal House
5103 Minnehaha Avenue South
Minnesota Veterans Home Bldg 1
Minneapolis, MN 55417

Progress Valley I
3033 Garfield Avenue South
Minneapolis, MN 55408

Progress Valley II
308 East 78th Street
Minneapolis, MN 55423

Recovery Resource Center
1900 Chicago Avenue
Minneapolis, MN 55404

River Ridge Treatment Center
700 South 3rd Street, Suite 101
Minneapolis, MN 55415

Salvation Army Harbor Light
Beacon Program
1010 Currie Avenue North
Minneapolis, MN 55405

3 RS Counseling Center
2220 Central Avenue NE
Minneapolis, MN 55413

Turning Point, Inc.
1500 Golden Valley Road
Minneapolis, MN 55405

1105 16 Avenue North
Minneapolis, MN 55411

Unity Hospital Substance Abuse Services
550 Osborne Road 2 East
Minneapolis, MN 55432

Veterans' Affairs Medical Center Addictive Disorders Section
1 Veterans Drive
Highway 55 and County 62
Minneapolis, MN 55417

Wayside House, Inc.
3705 Park Center Boulevard
Minneapolis, MN 55416

West Metro Recovery Services
5810 North 42nd Avenue
Minneapolis, MN 55331

MINNETONKA

New Connection Programs
Adolescent Outpatient Program
Hennepin County Home School
14300 County Road 62
Minnetonka, MN 55345

Regents Hospital Home School Program
14300 County Road, Suite 62
Minnetonka, MN 55345

River Ridge Nonresidential Treatment Center
15612 West Highway 7
Highwood Office Center Suite 150
Minnetonka, MN 55345

MONTICELLO

Big Lake Community Hospital Counseling Center
407 Washington Street
Monticello, MN 55362

MOORHEAD

Clay County Receiving Center
715 North 11 Street
Moorhead, MN 56560

Wellness Center of Fargo/ Moorhead
403 Center Avenue
Suite 409
Moorhead, MN 56560

MORRIS

Stevens Community Memorial Hospital New Beginning Center
400 East First Street
Morris, MN 56267

MORTON

Lower Sioux Alcoholism Program
Route 1
Morton, MN 56270

NAVARRE

Lifestyle Counseling of Mound
2389 Blaine Avenue
Navarre, MN 55392

NETT LAKE

Anishinaabe Miikana Gidamaajitaamin Bois Forte
13090 Westley Drive
Nett Lake, MN 55772

NEVIS

Pine Manor, Inc. Chemical Dependency Services
Route 2
Nevis, MN 56467

NEW BRIGHTON

Amethyst Counseling Services Outpatient Chemical Dependency Treatment Services
1405 Silver Lake Road
New Brighton, MN
55112

NEW ULM

Brown County Detox and Evaluation Center
510 North Front Street
New Ulm, MN 56073

New Ulm Medical Center
1324 North 5 Street
New Ulm, MN 56073

NORTHFIELD

Northfield Family Focus
220 Division Street
Northfield, MN 55057

OAKDALE

We Care Counseling Center
6060 50 Street North, Suite 1
Oakdale, MN 55128

OWATONNA

Owatonna Family Focus
215 Sout Oak Avenue
Owatonna, MN 55060

West Hills Lodge, Inc.
545 Florence Avenue
Owatonna, MN 55060

PINE CITY

Meadow Creek
Route 4
Pine City, MN 55063

Pine Shores Chemical Dependency Services
Route 2
Pine City, MN 55063

PIPESTONE

Southwest Mental Health Center
1016 8th Avenue SW
Pipestone, MN 56164

PLYMOUTH

Ark Counseling of Plymouth
1884 Berkshire Street
Plymouth, MN 55447

PRESTON

Visions
124 Main Street
Preston, MN 55965

PRIOR LAKE

Lifestyle Counseling Services
16511 Anna Trail SE
Suite C
Prior Lake, MN 55372

REDBY

Northern Winds Treatment Center Oosh Kii Mii Kah Nah
Redby, MN 56670

Red Lake Group Home Adolescent Inpatient Program
Redby, MN 56670

Red Lake Tribal Substance Abuse Prevention Programs
Redby, MN 56670

REDWOOD FALLS

Project Turnabout/Redwood Falls Outpatient Program
334 South Jefferson Street
Redwood Falls, MN 56283

RICHFIELD

Progress Valley II
308 East 78 Street
Richfield, MN 55423

ROCHESTER

Aiimsonion Clinic Chemical Dependency Program
300 3 Avenue SE
Ironwood Square Suite 206
Rochester, MN 55904

Charter Behavioral Health Systems of Rochester
333 16th Avenue NW
Rochester, MN 55901

Dunatos Outpatient Program
Rochester, MN 55903

Fountain Center
4104 18 Street NW
Cedarwood Mall
Rochester, MN 55901

Franciscan Skemp Health Care Center
1623 4th Street NW
Rochester, MN 55901-1827

Gables, The
604 5 Street SW
Rochester, MN 55902

Guest House
4800 48 Street NE
Rochester, MN 55903

Mayo Adult Chemical Dependency Treatment Center
1216 2nd Street SW
Generose Building, First Floor East
Rochester, MN 55902

Mayo Foundations Outpatient Addictions Service
121 2nd Street SW
Generose Building
Rochester, MN 55905

Pathway House
613 2nd Street SW
Rochester, MN 55902

Pathway to Parenthood
103 6 Avenue SW
Rochester, MN 55902

Zumbro Valley Mental Health Center, Inc.
Crisis Receiving Unit
2116 SE Campus Drive
Suite 105
Rochester, MN 55904

Recovery Basics
1932 Viking Drive NW
Rochester, MN 55906

Right to Recovery Program
917 North Broadway Street
Rochester, MN 55906

ROSEAU

Glenmore Recovery Center Outpatient Clinic
101 South Main Street
Roseau, MN 56751

SAINT CLOUD

Central Minnesota Mental Health Center Alcohol and Drug Abuse Services
1321 13th Street North
Saint Cloud, MN 56303

Effective Living Center, Inc.
114 1st Avenue West
Saint Cloud, MN 56301

Focus 12 Halfway House
3220 North 8 Street
Saint Cloud, MN 56303

Journey Home
210 5 Avenue NE
Saint Cloud, MN 56304

Passage Home
1003 South 8 Avenue
Saint Cloud, MN 56301

Saint Cloud Hospital Recovery Plus
1406 North 6 Avenue
Saint Cloud, MN 56301

Veterans' Affairs Medical Center Alcohol/Drug Dependence Treatment Program
4801 North 8 Street
Unit 116C
Saint Cloud, MN 56303

SAINT PAUL

African American Family Services
1041 Selby Avenue
Saint Paul, MN 55104

Ahrens Residence
1609 Jackson Street
Saint Paul, MN 55117

Charter Behavioral Health Systems of West Saint Paul
1555 Livingston Avenue
Suite 101
Saint Paul, MN 55118

Conceptual Counseling, Inc.
245 East 6 Street
Suite 435
Saint Paul, MN 55101

Hazelden/Fellowship Club
680 Stewart Avenue
Saint Paul, MN 55102

Juel Fairbanks Chemical Dependency Services, Inc.
806 North Albert Street
Saint Paul, MN 55104

Kelly Institute
2700 University Avenue West
Suite 20
Saint Paul, MN 55114

Model Cities Family Development Center
839 University Avenue
Saint Paul, MN 55104

Pride Institute Outpatient Program
405 Sibley Street, Suite 125
Saint Paul, MN 55101

Ramsey County Receiving Center
155 East 2nd Street
Saint Paul, MN 55101-1424

Regions Hospital
445 Etna Street, Suite 55
Saint Paul, MN 55106

Saint Joseph Hospital/Health East
Adolescent Behavioral Health Services
Chemical Dependency Program
69 West Exchange Street
Saint Paul, MN 55102

Senior Chemical Dependency Program
1380 Frost Avenue
Saint Paul, MN 55109

Twin Town Treatment Center
1706 University Avenue
Saint Paul, MN 55104

United Hospital Chemical Dependency Services
333 North Smith Avenue
Suite 4900
Saint Paul, MN 55102

SAINT PETER

Charter Behavioral Health System of Saint Peter Outpatient Program
116 South 3rd Street
Saint Peter, MN 56082

Johnson Chemical Dependency Center
100 Freeman Drive
Johnson Hall
Saint Peter, MN 56082

SAWYER

Mash Ka Wisen Treatment Center
Sawyer, MN 55780

SHAKOPEE

Stafford CD Treatment Center, Inc.
1100 East 4 Avenue
Suite 60
Shakopee, MN 55379

STILLWATER

Cedar Ridge, Inc. Extended Care Program
11400 Julianne Avenue North
Stillwater, MN 55082

Stillwater Outpatient
6381 Osgood Avenue
Stillwater, MN 55082

Washington County Jail Program Human Services, Inc.
14900 61st Street North
Stillwater, MN 55082

THIEF RIVER FALLS

Glenmore Recovery Center Outpatient Clinic
621 North Labree Avenue
Thief River Falls, MN 56701

Northwest Recovery Center, Inc.
115 6th Street West
Thief River Falls, MN 56701

TWO HARBORS

Lake View Memorial Hospital Outpatient Chemical Dependency Unit
325 11 Avenue
Two Harbors, MN 55616

VIRGINIA

Arrowhead Center, Inc.
505 12 Avenue West
Virginia, MN 55792

Halfway House
450 Pine Mill Court
Virginia, MN 55792

Range Mental Health Center, Inc. Detoxification Service
901 9 Avenue
Virginia, MN 55792

Twelfth Step House, Inc.
512 2 Street North
Virginia, MN 55792

WABASHA

Hiawatha Valley Mental Health Center
611 Broadway Avenue, Suite 100
Wabasha, MN 55981

WACONIA

Counseling Center of Waconia
24 South Olive Street
Waconia, MN 55387

Cornerstone Recovery Center
301 Industrial Boulevard
Waconia, MN 55387

WADENA

Bell Hill Recovery Center
Wadena, MN 56482

Neighborhood Counseling Center
11 2nd Street SW
Wadena, MN 56482

WASECA

Waseca Family Focus
203 South State Street
Waseca, MN 56093

WAVERLY

Charter Behavioral Health Services
109 North Shore Drive
Waverly, MN 55390

WAYZATA

Way 12 Halfway House
645 East Wayzata Boulevard
Wayzata, MN 55391

WHITE EARTH

Chi-Ska-Wes-Eh Halfway Home
White Earth, MN 56591

White Earth Chemical Dependency Program
Richwood Road
White Earth, MN 56591

WILLMAR

Bradley Center
1550 Highway 71 NE
Willmar Regional Treatment Center
Willmar, MN 56201

Cardinal Recovery Center/ Willmar Regional Treatment Center
316 Becker Avenue SW
Cardinal Square Suite 323
Willmar, MN 56201

Cardinals Prairie Youth Program
1550 Highway 71 NE
Willmar, MN 56201

Woodland Centers
Apple Tree Square
Highway 12
Willmar, MN 56201

WINNEBAGO

Adolescent Treatment Center of Winnebago
550 Cleveland Avenue West
Winnebago, MN 56098

WINONA

Franciscan Skemp Behavioral Health
Amethyst House
428 West Broadway
Winona, MN 55987

1600 Gilmore Avenue
Suite 110-A
Winona, MN 55987

Winona Counseling Clinic Chemical Dependency Services
111 Market Street
Winona, MN 55987

WINSTED

Counseling Center of Winsted
551 4 Street North
Winsted, MN 55395

WOODSTOCK

New Life Treatment Center County Road
120 East Dakota
Woodstock, MN 56186

WORTHINGTON

Addiction Recovery Technologies of Worthington
424 10th Street
Worthington, MN 56187

Southwest Mental Health Center Challenges
701 11th Street
Worthington, MN 56187

MISSISSIPPI

BILOXI

Veterans Affairs Medical Center
400 Veterans Avenue
Biloxi, MS 39531

BRANDON

Region 8 Community Mental Health Center New Roads Alcohol and Drug Services
105 Office Park, Box 88
Brandon, MS 39043

CLARKSDALE

Region I Mental Health Center
Alcohol and Drug Services
1742 Cheryl Street
Health Services Building
Clarksdale, MS 38614

CLINTON

Victory Manor Recovery Center
100 West Northside Drive
Clinton, MS 39056

COLUMBUS

Baptist Memorial Hospital Chemical Dependency Unit
525 Willowbrook Road
Columbus, MS 39703

Recovery House, Inc.
770 Golding Road
Columbus, MS 39704

COLUMBUS AFB

Columbus Air Force Base Substance Abuse Program
14 MDOS/SGOMH
201 Independence Drive
Suite 101
Columbus AFB, MS 39701-5300

CORINTH

Magnolia Regional Health Center Crossroads Psychiatric Unit
611 Alcorn Drive
Corinth, MS 38834

Timber Hills Mental Health Services
601 Foote Street
Corinth, MS 38834

GREENVILLE

Delta Community Mental Health Services Substance Abuse Services
1654 East Union Street
Greenville, MS 38701

GREENWOOD

Region 6 Community Mental Health Center
Old Browning Road, Box 1505
Greenwood, MS 38935-1505

GRENADA

Grenada Lake Medical Center
960 Avent Drive
Grenada, MS 38901

GULFPORT

BHC Hill Hospital
11150 Highway 49 North
Gulfport, MS 39503

Branch Medical Clinic Addiction Treatment Facility
5501 Marvin Shield Boulevard
Code 100
Gulfport, MS 39501

HATTIESBURG

Pine Belt Mental Healthcare Resources
Programs for Chemical Dependency
820 South 28 Avenue
Hattiesburg, MS 39401

Pine Grove Recovery Center
2255 Broadway Drive
Hattiesburg, MS 39401

JACKSON

Alcohol Services Center, Inc. Drug Treatment Unit
950 North West Street
Jackson, MS 39202

Baptist Behavioral Health Services
1225 North State Street
Jackson, MS 39201

Center for Independent Learning, Inc.
Special Women's Program Transitional Services
4550 Manhattan Road
Jackson, MS 39286

Friends of Alcoholics
1422 Foa Road
Jackson, MS 39209

Harbor Houses of Jackson, Inc.
Men's Division Alcoholism Treatment
1019 West Capitol Street
Jackson, MS 39203

Women's Division
3588 Flowood Drive
Jackson, MS 39208

Metro Counseling Center, Inc.
927 Palmayra Street
Jackson, MS 39205

New Life for Women Inc
814 North Congress Street
Jackson, MS 39202

Veterans' Affairs Medical Center Chemical Dependence Treatment Program
1500 East Woodrow Wilson
Unit 116B1
Jackson, MS 39216

KESSLER AFB

Kessler Air Force Base Substance Abuse Program
81 MDOS/SGOMH
301 Fisher Street, Room 1A-132
Keesler AFB, MS 39534-2519

LAUREL

South Central Regional Medical Center
1220 Jefferson Street
Laurel, MS 39441

MCCOMB

Southwest Mississippi MH/MR Complex Regional Alcohol and Drug Services
1701 White Street
McComb, MS 39648

MENDENHALL

New Roads Residential Treatment Center
1060 Smith Road
Mendenhall, MS 39114

MERIDIAN

Adult Male Alcohol and Drug Services Unit
4555 Highland Park Drive
Meridian, MS 39302

Laurel Wood Center
5000 Highway 39 North
Meridian, MS 39303

Weems Mental Health Center
Alcohol and Drug Program
Weems Lifecare
1415 Junior College Road
Meridian, MS 39304

OCEAN SPRINGS

Home of Grace Men's Program
14200 Jericho Road
Ocean Springs, MS 39565

OLIVE BRANCH

Charter Parkwood
8135 Goodman Road
Olive Branch, MS 38654

OXFORD

Communicare Alcohol and Drug Program Haven House
152 Highway 7th Street
Oxford, MS 38655

PARCHMAN

Mississippi Department of Corrections Alcohol and Drug Abuse Program
Parchman, MS 38738

PHILADELPHIA

Choctaw Community Mental Health Mississippi Band of Choctaw Indians
Route 7
Choctaw Health Center
Philadelphia, MS 39350

TUPELO

North Mississippi Medical Center
830 South Gloster Street
Tupelo, MS 38801

Region III Community Mental Health Center
2434 South Eason Boulevard
Tupelo, MS 38801

VICKSBURG

Marian Hill Chemical Dependency Center
100 McAuley Drive
Vicksburg, MS 39180

Warren/Yazoo Mental Health Service
3444 Wisconsin Avenue
Vicksburg, MS 39180

WHITFIELD

Mississippi State Hospital Chemical Dependency Unit
Building 84
Whitfield, MS 39193

MISSOURI

ALBANY

Family Guidance Center for Behavioral Healthcare
302 North Smith Street
Albany, MO 64402

AVA

South Central Missouri Rehab Center, Inc.
Douglas County Courthouse
Ava, MO 65608

BELLE

Missouri Alcohol Assessment Consultants, Inc.
206 South Church Street
Belle, MO 65013

BELTON

Midwest ADP Center Belton Site CIP/Outpatient
17136 Bel Ray Place
Belton, MO 64012

BETHANY

North Central Missouri Mental Health Center
3405 Miller Street
Bethany, MO 64424

BOONVILLE

Boonville Valley Hope
1415 Ashley Road
Boonville, MO 65233

Family Counseling Center of Missouri, Inc. Outpatient Clinic
211 Main Street
Boonville, MO 65233

BRANSON

Tri-Lake Sigma House
360 Rinehart Road
Branson, MO 65616

BROOKFIELD

North Central Missouri Mental Health Center
1 Center Drive
Brookfield, MO 64628

Preferred Family Healthcare Inc Brookfield Office
1 Center Drive
Brookfield, MO 64628

CAMERON

Family Guidance Center for Behavioral Healthcare
502 Northland Plaza
Cameron, MO 64429

CANTON

Hannibal Council on Alcohol and Drug Abuse
413 College Street
Canton, MO 63435

CAPE GIRARDEAU

Family Counseling Center, Inc. Women's CSTAR
20 South Sprig Street
Suite 2
Cape Girardeau, MO 63701

Gibson Recovery Center, Inc.
1112 Linden Street
Cape Girardeau, MO 63703

CARUTHERSVILLE

Correctional Counseling, Inc.
1210 West Highway 84
Caruthersville, MO 63830

CHILLICOTHE

North Central Missouri Mental Health Center
705 Webster Street
Chillicothe, MO 64601

CLINTON

Pathways
1800 Community Drive
Clinton, MO 64735

COLUMBIA

Arthur Center
103-B Corporate Lake Drive
Columbia, MO 65203

Charter Behavioral Health System of Columbia
200 Portland Street
Columbia, MO 65201

DRD Columbia Medical Clinic
1415 Paris Road
Columbia, MO 65201

Family Counseling Center of Missouri, Inc.
Alcohol/Drug Treatment Services
117 North Garth Street
Columbia, MO 65203

CSTAR McCambridge Center
201 North Garth Street
Columbia, MO 65203

Harry S. Truman Memorial Veterans Hospital
800 Hospital Drive
Columbia, MO 65201

Mid-Missouri Mental Health Center Alcohol and Drug Abuse Unit
3 Hospital Drive
Columbia, MO 65201

Phoenix Programs, Inc. Residential Program
607 South 5th Street
Columbia, MO 65201

CRESTWOOD

Southeast Missouri Community Treatment Center Accredited Family Clinic
9264 Waston Road
Crestwood, MO 63126

CREVE COUER

Edgewood Program
615 South New Ballas Road
Creve Coeur, MO 63141

DESOTO

COMTREA, Inc.
3343 Armbruster Road
DeSoto, MO 63020

EL DORADO SPRINGS

Pathways Community Behavioral Healthcare, Inc. El Dorado Springs Outpatient
107 West Broadway Street
El Dorado Springs, MO 64744

EXCELSIOR SPRINGS

Northland Community Center
106 Elizabeth Street
Excelsior Springs, MO 64024

FARMINGTON

Southeastern Missouri Treatment Center
Aquinas Center
5336 Highway 32 East
Farmington, MO 63640

FESTUS

Community Treatment, Inc. (COMTREA)
227 Main Street
Festus, MO 63028

FLORISSANT

Christian Hospital Northwest
1225 Graham Road
Florissant, MO 63031

Eastern Missouri Alternative Sentencing Services EMA/ Flourissant CIP/Outpatient
19 Florissant Oaks Street
Florissant, MO 63031

FORDLAND

Ozark Correctional Center OTP Avalon Community Services, Inc.
Route 2
Fordland, MO 65652

FORT LEONARD WOOD

Alcohol and Drug Abuse Prevention and Control Program Community Counseling Center
MCXP-BM-AD Building 310
Fort Leonard Wood, MO 65473

FULTON

Fulton State Hospital
Alcohol and Drug Treatment Unit
600 East 5th Street
Fulton, MO 65251

Hannibal Council on Alcohol and Drug Abuse Recovery Center
502 North Nichols Street
Fulton, MO 65251

GAINESVILLE

South Central Missouri Rehab Center, Inc. Ozark County Health Center
304 West 3rd Street
Gainesville, MO 65655

GALLATIN

North Central Missouri Mental Health Center
109 East Jackson Street
Gallatin, MO 64640

GLADSTONE

Columbia Health Systems, Inc.
6910 North Holmes Street
Suite 148
Gladstone, MO 64118

HAMILTON

North Central Missouri Mental Health Center
1 Cross Street
Hamilton, MO 64644

HANNIBAL

Hannibal Council on Alcohol/ Drug Abuse, Inc.
146 Communications Drive
Hannibal, MO 63401

HARRISONVILLE

Community Mental Health Consultants, Inc.
Cass County Psychological Services
306 South Independence Street
Harrisonville, MO 64701

Pathways Community Behavioral Healthcare, Inc.
300 Galaxie Avenue
Harrisonville, MO 64701

HAYTI

Correctional Counseling, Inc.
806 East Washington Street
Hayti, MO 63851

Family Counseling Center, Inc.
Highway J North
Hayti, MO 63851

HOUSTON

Southeast Missouri Community Treatment Center
SEMO/Houston Office
Texas City Health
201 South First Street
Houston, MO 65483

INDEPENDENCE

Comprehensive Mental Health Services
10819 Winner Road
Independence, MO 64052

CSTAR Program
10901 Winner Road
Independence, MO 64052

Midwest Addiction, Inc.
4231 South Hocker Street
Building 13, Suite 250
Independence, MO 64055

JEFFERSON CITY

Capital Region Medical Center Chemical Dependency Recovery Program
1600 Southwest Boulevard
Capitol Region Medical Center
Jefferson City, MO 65102

Family Counseling Center of Missouri, Inc.
Jefferson City Outpatient
502 East McCarty Street
Jefferson City, MO 65101

Fulton State Hospital Capital City ADA Outpatient
211 Oscar Drive, Suite A
Jefferson City, MO 65101

Jefferson City Correctional Center Intensive Therapeutic Community
631 State Street
Jefferson City, MO 65101

JOPLIN

Family Self Help Center, Inc. DBA Lafayette House/CSTAR
1809 Connor Avenue
Joplin, MO 64804

Ozark Center New Directions
Acute Adult Substance Abuse Treatment Program
530 East 34th Street
Joplin, MO 64801

CSTAR
Substance Abuse Unit
2808 Picher Street
Joplin, MO 64803

Scott Greening Center for Youth Dependency, Inc.
1315 East 20 Street
Joplin, MO 64804

KANSAS CITY

Baptist Medical Center
6601 Rockhill Road
8th Floor
Kansas City, MO 64131

Benilde Hall Program
1600 Paseo Boulevard
Kansas City, MO 64108-1623

DRD Kansas City Medical Clinic
723 East 18 Street
Kansas City, MO 64108

Gateway Foundation, Inc. Intensive Outpatient Services
1734 East 63rd Street, Suite 301
Kansas City, MO 64110

Kansas City Community Center (KCCC)
1534 Campbell Street
Kansas City, MO 64108

Kansas City Free and Clean Gateway Foundation, Inc.
1734 East 63rd Street, Suite 301
Kansas City, MO 64110

Marillac Center
2826 Main Street
Kansas City, MO 64108

Midwest ADP Center Outpatient Program
1212 McGee Street
Kansas City, MO 64108

Missouri Dept of Labor/ Industrial Relations
North Clinic/Residential
Women's Place
5840 Swope Parkway
Kansas City, MO 64130

North Star Recovery Research Mental Health Services
2801 Wyandotte Street, 6th Floor
Kansas City, MO 64108

Residential Unit
3220 East 23 Street
Kansas City, MO 64127

Rodgers South
2701 East 31 Street
Kansas City, MO 64128

Salvation Army Missouri Shield of Service
5100 East 24th Street
Kansas City, MO 64127

Scott Greening Center, Inc. Western Region Unit
2750 Cherry Street
Kansas City, MO 64108-3140

Truman Medical Center East
Administrative Site
7900 Lees Summit Road
Kansas City, MO 64139

Behavioral Health/Relapse Program
221 Charlotte Street
Kansas City, MO 64108

Western Missouri Mental Health Center
Paseo Comprehensive Rehab Clinic
2211 Charlotte Street
Kansas City, MO 64108

KENNETT

Family Counseling Center, Inc.
1109 Jones Street
Kennett, MO 63857

KIRKSVILLE

Preferred Family Healthcare, Inc.
1101 South Jamison Street
Kirksville, MO 63501-0767

KIRKWOOD

Saint Louis Foundation for Alcohol and Related Dependencies
Exodus Program
135 West Adams Street, Suite 203
Kirkwood, MO 63122

LEES SUMMIT

Research Mental Health Services
901 NE Independence Avenue
Lees Summit, MO 64063

LINN CREEK

Family Counseling Center of Missouri, Inc. Cedar Ridge Treatment Center
Route 1
Linn Creek, MO 65052

LOCKWOOD

Community Mental Health Consultants, Inc. Dade County Psychological Services
1111 South Main Street
Lockwood, MO 65682

LOUISIANA

Hannibal Council on Alcohol
3516 Georgia Street
Louisiana, MO 63353

MACON

Preferred Family Healthcare
907 State Route
Macon, MO 63552

MALDEN

Correctional Counseling Inc.
110 East Main Street
Malden, MO 63863

MARSHALL

David R Rasse and Associates, Inc.
78 West Arrow Street
Marshall, MO 65340

Fulton State Hospital Marshall ADA Outpatient Clinic
Marshall Habilitation Center
700 East Slater
Marshall, MO 65340

MARYVILLE

Family Guidance Center
301 East Summit Drive
South Hills Medical Building
Maryville, MO 64468

MEXICO

Hannibal Council on Alcohol/ Drug Abuse, Inc.
Mexico Area Recovery Center
1130 South Elmwood Street
Mexico, MO 65265

MILAN

North Central Missouri Mental Health Center
217 East Second Street
Milan, MO 63556

MOBERLY

Better Choices
102 East Rollins Street
Moberly, MO 65270

Escape Outpatient Chemical Dependency Center
501 North Ault Street
Moberly, MO 65270

MONETT

Clark Community Mental Health Center
307 4th Street
Monett, MO 65708

MOUNTAIN GROVE

South Central Missouri Rehab Ctr Inc Grace and Glory Church
1104 North Main Street
Mountain Grove, MO 65711

MOUNTAIN VIEW

South Central Missouri Rehab Ctr Inc United Methodist Church
106 East 3rd Street
Mountain View, MO 65548

MOUNT VERNON

The University of MO Crossroads Prog DBA MO Rehabilitation Center
600 North Main Street
Mount Vernon, MO 65712

NEOSHO

Ozark Center New Directions
214 North Washington Street
Neosho, MO 64850

1011 West Hill Street
Neosho, MO 64850

NEVADA

Community Mental Health Consultants
815 South Ash Street
Nevada, MO 64772

North Complex
427 North Cedar Street
Nevada, MO 64772

Pathways Comm Behav Healthcare Inc Nevada Outpatient
2203 North Elm Street
Nevada, MO 64772

NEW MADRID

Correctional Counseling, Inc.
315 Main Street
New Madrid, MO 63869

ODESSA

Pathways Commmunity Behavioral Healthcare, Inc. Odessa Outpatient
301 North 2nd Street
Odessa, MO 64076

OVERLAND PARK

Wubbenhorst and Wubbenhorst, Inc. DBA Madison Avenue Psychologial Services
8826 Santa Fe Street, Suite 207
Overland Park, MO 66212

PERRYVILLE

Gibson Recovery Center, Inc.
300 Perry Plaza, Suite F
Perryville, MO 63775

POPLAR BLUFF

Diversified Treatment Services
New Era/Westwood Center
Weekend Intervention Program
Route 11
Poplar Bluff, MO 63901

Doctors Regional Medical Center
419 Oak Boulevard
Poplar Bluff, MO 63901

Family Counseling Center, Inc.
400 Vine Street
Poplar Bluff, MO 63901

POTOSI

Southeast Missouri Community Treatment Center
108 Thistle Street Austin Plaza
Potosi, MO 63664

ROCK PORT

Family Guidance Center for Behavioral Healthcare
201 East Highway 169
Rock Port, MO 64482

ROLLA

Southeast Missouri Community Treatment Center
1702 East 10th Street
Rolla, MO 65401

SAINT CHARLES

Bridgeway Counseling Services, Inc.
1601 Old South River Road
Saint Charles, MO 63303

SAINT JOSEPH

Saint Joseph Youth Center
702 Felix Street
Saint Joseph, MO 64501

SAINT LOUIS

Alexian Brothers Hospital
3800 South Broadway
Saint Louis, MO 63118

Archway Communities, Inc.
5652 Pershing Avenue
Saint Louis, MO 63112

Black Alcohol/Drug Service Info. Center (BASIC)
CSTAR
1221 Locust Street
Suite 800
Saint Louis, MO 63103

Christian Hospital Recovery Center
605 Old Ballas Road
Saint Louis, MO 63141

Dart, Inc.
Administrative Unit
Medication Unit
Outpatient Unit
1307 Lindbergh Plaza Center
Saint Louis, MO 63132

East Unit
1027 South Vandeventer Street
Saint Louis, MO 63110

Gateway Foundation Inc
1430 Olive Street Suite 300
Saint Louis, MO 63103-2303

Community Release Center
1621 North First Street
Saint Louis, MO 63102

DBA GFI Services
Gateway Free And Clean
1430 Olive Street Suite 305
Saint Louis, MO 63103-2303

Harris House Foundation
8327 South Broadway
Saint Louis, MO 63111

Hyland Center
10020 Kennerly Road
Saint Louis, MO 63128

Metropolitan Saint Louis Psychiatric Center
5351 Delmar Boulevard
Saint Louis, MO 63112

New Beginnings CSTAR Inc
Adolescent CSTAR
1408 North Kings Highway
Saint Louis, MO 63113

Alternative Care
625 North Euclid Street, 5th Floor
Saint Louis, MO 63108

Provident Counseling Family Care Program
9109 Watson Street
Saint Louis, MO 63126

Queen of Peace Center
325 North Newstead Street
Saint Louis, MO 63108

Saint Patrick Center
1200 North 6th Street
Saint Louis, MO 63106

Salvation Army
CSTAR Program
10740 Page Boulevard
Saint Louis, MO 63132

Harbor Light Center
3010 Washington Avenue
Saint Louis, MO 63103

SAINT PETERS

Missouri Valley Alcohol and Drug Program
1125 Cave Springs Estate Drive
Suite F
Saint Peters, MO 63376

SALEM

Southeast Missouri Community Treatment Center Salem Center
203 North Grand Street
Salem, MO 65560

SEDALIA

Pathways Community Behavioral Healthcare, Inc.
State Fair Shopping Center
Sedalia, MO 65301

SPRINGFIELD

Bridgeway Substance Abuse Program
2828 North National Avenue
Springfield, MO 65803

Burrell, Inc.
CSTAR Program
1300 Bradford Parkway
Springfield, MO 65804

Carol Jones Recovery Center for Women
2411 West Catalpa Street
Springfield, MO 65807

Center for Addictions Cox Health Systems
1423 North Jefferson Street
Springfield, MO 65802

DRD Springfield Medical Clinic
1046 West Sunshine Street
Springfield, MO 65807

Sigma House, Inc.
800 South Park Avenue
Springfield, MO 65802

TRENTON

North Central Missouri Mental Health Center
Administrative Unit
Substance Abuse Program
1601 East 28 Street
Trenton, MO 64683

Preferred Family Healthcare, Inc.
703 Main Street
Trenton, MO 64683

UNION

Meramec Recovery Center, Inc.
115 South Oak Street
Union, MO 63084

Missouri Alcohol Assessment Consultants, Inc.
206 South Church Street
Union, MO 63084

UNIONVILLE

North Central Missouri Mental Health Center
132 North 19th Street
Unionville, MO 63565

VANDALIA

Gateway Foundation, Inc.
Women's Eastern Reception/ Diagnostic
1101 East Highway 54
Vandalia, MO 63382

WARRENSBURG

Pathways Community Behavioral Healthcare, Inc. Warrensburg Recovery Center
703 North Devasher Street
Warrensburg, MO 64093

WASHINGTON

Clayton Concepts, Inc. New Hope
516 Jefferson Street
Washington, MO 63090

WAYNESVILLE

Piney Ridge Center, Inc.
1000 Hospital Road
Waynesville, MO 65583

WEST PLAINS

South Central Missouri Rehab Center
1015 Lanton Road
West Plains, MO 65775

WINDSOR

Royal Oaks Hospital
307 North Main Street
Windsor, MO 65360

WOODSON TERRACE

Saint Louis Metro Treatment Center, Inc.
4024 Woodson Road
Woodson Terrace, MO 63134

MONTANA

ANACONDA

Deer Lodge County Alcohol and Drug Services of Anaconda
100 West Park Street
Anaconda, MT 59711

BILLINGS

Journey Recovery Program
1245 North 29 Street
Billings, MT 59103

Rimrock Foundation
123l North 29 Street
Billings, MT 59101

BOX ELDER

Rocky Boys Chemical Dependency Center
Rural Route 1
Box Elder, MT 59521

BOZEMAN

Alcohol/Drug Services of Gallatin County
502 South 19 Street
Suite 302
Bozeman, MT 59715

BUTTE

Butte/Silver Bow Chemical Dependency Services
125 West Granite Street
Butte, MT 50701

Montana Chemical Dependency Center
2500 Continental Drive
Butte, MT 59701

North American Indian Alliance Chemical Dependency Program
100 East Galena Street
Butte, MT 59701

DEER LODGE

Chemical Dependency and Family Counseling, Inc.
304 Milwaukee Avenue
Deer Lodge, MT 59722

FORT BENTON

TLC Recovery, Inc.
1308 Frankin Street
Court House Annex
Fort Benton, MT 59442

FORT HARRISON

VAMAM/ROC
Fort Harrison, MT 59636-1500

GLENDIVE

District II Alcohol and Drug Program
119 South Kendrick Street
Glendive, MT 59330

GREAT FALLS

Benefits Health Care
500 15th Avenue South
Great Falls, MT 59405

Montana Deaconess Medical Center Chemical Dependency Unit
1101 26 Street South
Great Falls, MT 59405

Gateway Recovery Center
401 3 Avenue North
Great Falls, MT 59401

HAMILTON

Crossroads/Ravalli County Chemical Dependency Services
214 Pinckney Street
Hamilton, MT 59840

HARLEM

Fort Belknap Chemical Dependency Program
Fort Belknap Reservation
Route 1
Harlem, MT 59526

HAVRE

Northern Montana Chemical Dependency Program
1410 First Avenue
Havre, MT 59501

HELENA

Boyd Andrew Chemical Dependency Care Center
Arcade Building Unit 1-E
Helena, MT 59601

KALISPELL

Flathead Valley Chemical Dependency Clinic, Inc.
1312 North Meridian Road
Kalispell, MT 59901

Pathways Treatment Center Kalispell Regional Medical Center
200 Heritage Way
Kalispell, MT 59901

LEWISTOWN

Alcohol and Drug Services of Central Montana
505 West Main Street
Suite 418
Lewistown, MT 59457

LIBBY

Recovery Northwest/Lincoln County Main Office
418 Main Avenue
Libby, MT 59923

LIVINGSTON

Southwest Chemical Dependency Program
414 East Callendar Street
Livingston, MT 59047

MALSTROM AFB

Malmstrom Air Force Base Substance Abuse Program
341 MDG/SGOMH
468 74 Street North
Malmstrom AFB, MT 59402-6780

MALTA

High Plains Chemical Dependency Services
105 1/2 South 2nd Street East
Malta, MT 59538

MARION

Wilderness Treatment Center
200 Hubbart Dam Road
Marion, MT 59925

MILES CITY

Eastern Montana Mental Health Substance Abuse and Dependency Services
2200 Box Elder
Miles City, MT 59301

MISSOULA

Missoula Indian Center
2300 Regent Street
Missoula, MT 59801

Saint Patrick Hospital Addiction Treatment Program
500 West Broadway
Missoula, MT 59802

Western Montana Regional Mental Health Turning Point
500 North Higgins Street
Suite 101
Missoula, MT 59802

POLSON

Lake County Chemical Dependency Program
12 5th Avenue East
Polson, MT 59860

SAINT IGNATIUS

Confederated Salish/Kootenai Tribes Addiction Treatment Program
402 Mission Drive
Saint Ignatius, MT 59865

NEBRASKA

AINSWORTH

Sandhills Mental Health and Substance Abuse Services, Inc.
312 North Main Street
Ainsworth, NE 69210

ALLIANCE

Human Services, Inc.
419 West 25 Street
Alliance, NE 69301

AUBURN

Blue Valley Mental Health Center
1121 15th Street
Auburn, NE 68305

BEATRICE

Blue Valley Mental Health Center
1200 South 9 Street
Beatrice, NE 68310

BELLEVUE

Lutheran Family Services/ Bellevue
1318 Federal Square Drive
Bellevue, NE 68005

Rainbow Hope Counseling and Recovery Services
1103 Galvin Road South
Bellevue, NE 68005-3031

Renaissance Program
703 West 24th Avenue
Bellevue, NE 68005

COLUMBUS

Mid-East Nebraska Behavioral Health Care Services
3314 26th Street
Columbus, NE 68601

Sunrise Place
4432 Sunrise Place
Columbus, NE 68602

CRETE

Blue Valley Mental Health Center
225 East 9th Street, Suite 1
Crete, NE 68333

DAVID CITY

Blue Valley Mental Health Center
367 E Street
David City, NE 68632

FAIRBURY

Blue Valley Mental Health Center
521 E Street
Fairbury, NE 68352

FALLS CITY

Blue Valley Mental Health Center
116 West 19th Street
Falls City, NE 68355

FREMONT

Alegent Health Behavioral Services
2350 North Clarkson Street
Fremont, NE 68025

Pathfinder Alcohol/Drug Outpatient Clinic
658 North H Street
Fremont, NE 68025

GENEVA

Blue Valley Mental Health Center Alcohol and Drug Abuse Services
831 F Street
Geneva, NE 68361

GORDON

Northeast Panhandle Substance Abuse Center
305 Foch Street
Gordon, NE 69343

GRAND ISLAND

Friendship House, Inc.
406 West Koenig Street
Grand Island, NE 68801

Mid-Plains Center Behavioral Healthcare, Inc.
914 Bauman Street
Grand Island, NE 68801

Milne Detoxification Center
406 West Koenig Street
Grand Island, NE 68801

Saint Francis Alcoholism/Drug Treatment Center
2116 West Faidley Avenue
Grand Island, NE 68803

Veterans' Affairs Medical Center Substance Abuse Treatment Program
2201 North Broadwell Street
Grand Island, NE 68803

HASTINGS

Hastings Regional Center Chemical Dependency Unit
Hastings, NE 68901

South Central Counseling
Hastings Clinic
616 West 5th Street
Hastings, NE 68901

The Bridge, Inc.
922 North Denver Street
Hastings, NE 68901

HEBRON

Blue Valley Mental Health
Thayer County Courthouse
Hebron, NE 68370

HOLDREGE

South Central Behavioral Services Holdrege Clinic
701 4th Avenue
Johnson Center, Suite 7
Holdrege, NE 68949

IMPERIAL

Region II Alcoholism and Drug Abuse Center
East Highway 6
Weir Building
Imperial, NE 69033

KEARNEY

Richard Young Hospital Chemical Dependency Unit
4600 17th Avenue
Kearney, NE 68847

South Central Counseling Substance Abuse Treatment Program
3810 Central Avenue
Kearney, NE 68847

Christopher House
2521 Central Avenue
Kearney, NE 68847-4547

LEXINGTON

Heartland Counseling and Consulting Clinic
307 East 5th Street
Lexington, NE 68850

LINCOLN

Antlers
2501 South Street
Lincoln, NE 68502

Bryan LGH Medical Center West Independence Center
1650 Lake Street
Lincoln, NE 68502

Center Pointe
Administration/Outpatient Offices
1000 South 13th Street
Lincoln, NE 68502

Adult Residential Program
610 J Street
Lincoln, NE 68508

Community Mental Health of Lancaster County
2200 Saint Mary's Avenue
Lincoln, NE 68502

Cornhusker Place
Detoxification Program
721 K Street
Lincoln, NE 68508

First Step
2231 Winthrop Road
Lincoln, NE 68502

Houses of Hope of Nebraska, Inc.
2015 South 16th Street
Lincoln, NE 68502

Lincoln Medical Education Foundation School Community Intervention Program
4608 Valley Road
Lincoln, NE 68510

Lincoln Valley Hope Alcohol and Drug Counseling and Referral Center
3633 O Street
Lincoln, NE 68510

Lincoln/Lancaster County Child Guidance Adolescent Substance Abuse Program
215 Centennial Mall South
312 Lincoln Center Building
Lincoln, NE 68508

Lutheran Family Social Services Substance Abuse Program
4620 Randolph Street
Lincoln, NE 68510

Saint Monica's
Project Mother and Child
2109 South 24th Street
Lincoln, NE 68510

Residential and Outpatient Services
6420 Colby Street
Lincoln, NE 68505

Veterans' Affairs Medical Center Chemical Abuse Services
600 South 70 Street
Unit 116A
Lincoln, NE 68510

MACY

Macy Counseling Center
100 Main Street
Macy, NE 68039

Macy Youth and Family Services
Macy, NE 68039

MCCOOK

Heartland Counseling and Consulting Clinic
1012 West 3rd Street
McCook, NE 69001

NEBRASKA CITY

Blue Valley Mental Health Center
1903 4 Corso
Nebraska City, NE 68410

Oak Arbor Recovery Center
1314 3 Avenue
Nebraska City, NE 68410

NIOBRARA

Santee Sioux Tribe of Nebraska/Health Education Addictions Recovery Training (HEART)
Route 2
Niobrara, NE 68760

NORFOLK

Faith Regional Health Services
East Campus
1500 Koenigstein Avenue
Norfolk, NE 68701

Norfolk Regional Center
1700 North Victory Road
Norfolk, NE 68701

Odyssey III Counseling Services
401 South 17th Street
Norfolk, NE 68701-4724

Ponca Tribe of Nebraska
1310 Norfolk Avenue, Suite B
Norfolk, NE 68701

The Link, Inc.
1001 Norfolk Avenue
Norfolk, NE 68701

Well Link, Inc.
305 North 9 Street
Norfolk, NE 68702

NORTH PLATTE

Great Plains Regional Medical Center
601 West Leota Street
North Platte, NE 69101

Lutheran Family Services
1300 East 4th Street
North Platte, NE 69101

New Horizons Detoxification Unit
110 North Bailey Street
North Platte, NE 69101

Region II Human Services Heartland Counseling/ Consulting Clinic
110 North Bailey Street
North Platte, NE 69101

O'NEILL

Valley Hope Alcoholism Treatment Center
1421 North 10th Street
O'Neill, NE 68763

OGALLALA

Heartland Counseling and Consulting Clinic
103 East 10th Street
Ogallala, NE 69153

OMAHA

A and A Assessments
4780 South 130th Street
Omaha, NE 68137

Adlerian Center for Therapy Consultation and Education
11911 Arbor Street
Omaha, NE 68144-2970

Alcoholics Resocialization Conditioning Help (ARCH Inc)
604 South 37th Street
Omaha, NE 68105

Alegant Health Behavioral Services
6901 North 72nd Street
Omaha, NE 68122

Arbor Family Counseling Associates, Inc.
11605 Arbor Street, Suite 106
Omaha, NE 68144-2934

Catholic Charities
Outpatient
Saint Gabriel's Center
Sheehan Center
3300 North 60th Street
Omaha, NE 68104

Chicano Awareness Center
4821 South 24th Street
Omaha, NE 68107

Discovery Center
2937 South 120th Street
Omaha, NE 68144

Family Services/South Omaha Counseling
2900 O Street
Livestock Exchange Building
Suite 521
Omaha, NE

Greater Omaha Community Action Alcohol and Drug Outpatient Services
2406 Fowler Street
Omaha, NE 68111

Intertribal Treatment Center
2301 South 15th Street
Omaha, NE 68108

Lydia House
3030 North 21st Street East
Omaha, NE 68110

Methodist Richard Young Behavioral Health Unit
415 South 25th Avenue
Omaha, NE 68105

Nova Therapeutic Community
Partial Care Center
1915 South 38th Street
Omaha, NE 68105

Residential Center
3473 Larimore Avenue
Omaha, NE 68111

Omaha Psychiatric Associates
2132 South 42nd Street
Omaha, NE 68105

Pathway Counseling
5036 South 136th Street, Suite A
Omaha, NE 68137-1622

Santa Monica, Inc.
103 North 39th Street
Omaha, NE 68131

Sienna/Francis House
1702 Nicholas Street
Omaha, NE 68102

Stephens Center
2723 Q Street
Omaha, NE 68107

Therapy Resource Associates
10855 West Dodge Road
Suite 180
Omaha, NE 68154

United Behavioral Systems, Inc.
11717 Burt Street, Suite 104
Omaha, NE 68154

University Alcohol and Alcohol Program
2205 South 10th Street
Omaha, NE 68108

Veterans' Affairs Medical Center Substance Abuse Treatment Center
4101 Woolworth Avenue
Omaha, NE 68105

O'NEILL

Sandhills Mental Health and Substance Abuse Services, Inc.
204 East Everett Street
O'Neill, NE 68763

Valley Hope Alcoholism Treatment Center
1421 North 10 Street
O'Neill, NE 68763

PAPILLION

Lutheran Family Services Papillion Clinic
120 West 2nd Street
Papillion, NE 68046

PAWNEE CITY

Blue Valley Mental Health Center
701 I Street
Pawnee City, NE 68420

PLATTSMOUTH

Lutheran Family Services Cass Family Clinic
542 Main Street
Plattsmouth, NE 68048

SCHUYLER

Pathfinder Clinic
802 A Street
Schuyler, NE 68661

SCOTTSBLUFF

Human Services, Inc. Detoxification Center
15 West 16th Street
Scottsbluff, NE 69361

Panhandle Mental Health Center Substance Abuse Program
4110 Avenue D
Scottsbluff, NE 69361

Regional West Medical Center Behavioral Health Services
3700 Avenue B
Scottsbluff, NE 69361

SEWARD

Blue Valley Mental Health Center
729 Seward Street
Seward, NE 68434

SIDNEY

Memorial Health Center
835 15th Avenue
Sidney, NE 69162

SOUTH SIOUX CITY

Heartland Counseling Services, Inc.
917 West 21st Street
South Sioux City, NE 68776

SUPERIOR

Family Resource Center
344 North Dakota Street
Superior, NE 68978-1843

VALENTINE

Sandhills Mental Health and Substance Abuse Services, Inc.
325 North Victoria Street
Presbyterian Church
Valentine, NE 69201

WAHOO

Blue Valley Mental Health Center
543 North Linden Street
Wahoo, NE 68066

WEST POINT

Pathfinder Clinic Alcohol and Drug Outpatient Clinic
434 North Lincoln Street
West Point, NE 68788

WHITE CLAY

Hands of Faith Ministry
Whiteclay, NE 69365

WINNEBAGO

Chee Woy Na Zhee Halfway House
Highway 77
Winnebago, NE 68071

Indian Health Service Drug Dependency Unit
Highway 77-75
Winnebago, NE 68071

YORK

Blue Valley Mental Health Center
727 Lincoln Avenue
York, NE 68467

Family Counseling Center
1100 Lincoln Avenue, Suite C-3
York, NE 68467-1743

NEVADA

CARSON CITY

Carson City Community Counseling Center
625 Fairview Drive
Suite 111
Carson City, NV 89701

Carson Treatment Center
120 North Harbin Avenue
Carson City, NV 89701

ELKO

Ruby View Counseling Center Outpatient
401 Railroad Street, Suite 301
Elko, NV 89801

Vitality Center
Residential Treatment
3740 East Idaho Street
Elko, NV 89801

Teen Discovery
1297 Idaho Street
Elko, NV 89801

ELY

Bristlecone Counseling Service Outpatient
995 Campton Street
Ely, NV 89301

FALLON

Basic Recovery Associates, Inc.
141 Keddie Street
Fallon, NV 89406

Churchill Council Alcohol and Drug Treatment
165 North Carson Street
Fallon, NV 89406

LAS VEGAS

Bridge Counseling Associates Outpatient
1701 West Charleston Boulevard
Las Vegas, NV 89104

Center for Behavioral Health/ Nevada Methadone Outpatient Treatment Center
3050 East Desert Inn Road
Suite 117
Las Vegas, NV 89121

Clark County Health District Addiction Treatment Clinic/ Methadone
625 Shadow Lane
Las Vegas, NV 89127

Clark County Juvenile Court Services Family Based Drug Treatment Program
3401 East Bonanza Road
Las Vegas, NV 89101

Community Counseling Center
1120 Almond Tree Lane
Las Vegas, NV 89104

Community Health Centers of South Nevada
916 West Owens Avenue
Las Vegas, NV 89106

Economic Opportunity Board of Clark County
Treatment Center
522 West Washington Street
Las Vegas, NV 89106

Emotional Health Services
919 East Bonneville Street
Las Vegas, NV 89101-2305

Family Preservation Services
4220 South Maryland Street
Las Vegas, NV 89119

Healthy Families Project
2500 Apricot Lane
Las Vegas, NV 89108

Las Vegas Indian Center
2300 West Bonanza Road
Las Vegas, NV 89106

Mesa Family Counseling
1000 South 3rd Street
Las Vegas, NV 89101

Nevada Community Enrichment Program
2820 West Charleston Boulevard
Suite D-37
Las Vegas, NV 89102

Nevada Treatment Center
1721 East Charleston Boulevard
Las Vegas, NV 89104

New Life Medical Center
1750 Industrial Road
Las Vegas, NV 89102

Southwest Passage
1101 North Decatur Boulevard
Las Vegas, NV 89108-1220

Westcare, Inc.
Adult Detox
930 North 4th Street
Las Vegas, NV 89101

Community Involvement Center
401 South Martin Luther King Boulevard
Las Vegas, NV 89106

Harris Springs Ranch
Las Vegas, NV 89016

LOVELOCK

Lovelock Counseling Clinic
775 Cornell Street
Lovelock, NV 89419

MESQUITE

Mesquite Mental Health Center
416 Riverside Road
Mesquite, NV 89024

NIXON

Pyramid Lake Health Department Sumunumu Substance Abuse Program
705 Highway 446
Nixon, NV 89424

NORTH LAS VEGAS

Salvation Army Las Vegas Adult Rehabilitation Program
211 Judson Street
North Las Vegas, NV 89030

OWYHEE

Owyhee Community Health Facility Shoshone Paiute Substance Abuse Program
Nevada State Highway 225
Owyhee, NV 89832

PAHRUMP

Westcare, Inc. Pahrump Youth Outpatient
1670 East Heritage Street
Pahrump, NV 89048

RENO

Basic Recovery Associates, Inc. Psychotherapeutic and Educational Ctr
1085 South Virginia Street
Suite C and D
Reno, NV 89502

Center for Behavioral Health of Nevada
160 Hubbard Way, Suite A
Reno, NV 89502

Family Counseling Service of Northern Nevada
575 East Plumb Lane
Reno, NV 89501

Reno Treatment Center
750 Kuenzli Street
Reno, NV 89502

Ridge House
57 Vine Street
Reno, NV 89503

Sagewind
1725 South McCarran Boulevard
Reno, NV 89510-1491

Step Two
3220 Coronado Street
Suite 380
Reno, NV 89503

SPARKS

Family Counseling Service of Northern Nevada, Inc.
480 Gallette Way
Building 9 Room 40
Sparks, NV 89431

Northern Area Substance Abuse Council Chemical Dependency Unit/Detox
480 Galletti Way
Buildings 3 and 4 Second Floor
Sparks, NV 89431

TONOPAH

Tonopah Counseling Center
1100 Erie Main Street
Tonopah, NV 89049

WEST WENDOVER

Great Basin Counseling Service
915 Wells Street
West Wendover, NV 89883

WINNEMUCCA

Silver Sage Counseling Service Outpatient Services
530 Melarkey Street
Winnemucca, NV 89445

YERINGTON

Lyon Council Alcohol and Drugs Yerington Project
26 Nevin Way
Yerington, NV 89447

NEW HAMPSHIRE

BEDFORD

Bedford Counseling Associates
25 South River Road
Bedford Commons
Bedford, NH 03110

BERLIN

Founders Hall Androscoggin Valley MHC
13 Green Square
Berlin, NH 03570-3860

Tri-County Community Action Program, Inc.
361 School Street
Berlin, NH 03570

CANTERBURY

Odyssey Family Center
367 Shaker Road
Canterbury, NH 03224

CLAREMONT

Bailey House
18 Bailey Avenue
Claremont, NH 03743

COLEBROOK

Upper Connecticut Mental Health and Developmental Services
34 Colby Street
Colebrook, NH 03576

CONCORD

Community Services Council Merrimack County Alcohol and Drug Intervention
2 Industrial Park Drive
Suite 5
Concord, NH 03301

Concord Hospital Fresh Start Program
250 Pleasant Street
Concord, NH 03301

Summit Behind the Walls New Hampshire State Prison
281 North State Street
Concord, NH 03301

DOVER

Prospects Frisbie Strafford Guidance
130 Central Avenue
Dover, NH 03820

Southeastern New Hampshire Services
272 Country Farm Crossroad
Dover, NH 03820

DUBLIN

Beech Hill Hospital Substance Abuse Services
New Harrisville Road
Dublin, NH 03444

Marathon House
Adolescent Program
Long Term Residential
1 Pierce Road
Dublin, NH 03444

EXETER

Southeastern New Hampshire Services
24 Front Street
Exeter, NH 03833

HENNIKER

Contoocook Valley Counseling Center
9 Hall Avenue
Henniker, NH 03242

KEENE

Cheshire Medical Center Mental Health Unit
580 Court Street
Keene, NH 03431

Marathon Behavioral Treatment Center
106 Roxbury Street
Keene, NH 03431

Monadnock Region Substance Abuse Services, Inc.
310 Marlboro Street
Keene, NH 03431

LACONIA

Horizons Counseling Center
Village West, Building
Laconia, NH 03246

Lakes Region General Hospital Nathan Brody Chemical Dependency Program
80 Highland Street
Laconia, NH 03246

LEBANON

Brill, Jacqueline
106 Hanover Street
Lebanon, NH 03766

Community Support Services Horizon House
85 Mechanic Street, Suite 360
Lebanon, NH 03766

Headrest
14 Church Street
Lebanon, NH 03766

West Central Services Counseling Center
2 Whipple Place
Suite 202
Lebanon, NH 03766

20 West Park
Lebanon, NH 03766

LITTLETON

White Mountain Mental Health Center Substance Abuse Services
29 Maple Street
Littleton, NH 03561

MANCHESTER

Farnum Center
235 Hanover Street
Manchester, NH 03104

Manchester Office of Youth Services
50 Bridge Street, Suite 308
Manchester, NH 03101

Mental Health Center of Greater Manchester Co-Occurring Disorders Treatment Program
43 Walnut Street
Manchester, NH 03104

Riverway Center for Recovery
100 McGregor Street
Manchester, NH 03102

Tirrell House
15–17 Brook Street
Manchester, NH 03104

Veterans Affairs Medical Center Substance Abuse Treatment Program (SATP)
718 Smyth Road Building 5
Manchester, NH 03104

NASHUA

Charter Brookside Behavioral Health Systems
29 Northwest Boulevard
Nashua, NH 03063

Gateway Family Health Center
268 Main Street
Nashua, NH 03060

Greater Nashua Council on Alcoholism Pine Street Extension
Keystone Hall
Nashua, NH 03060

Nashua Youth Council
112 West Pearl Street
Nashua, NH 03060

Saint Joseph's Hospital New Start
172 Kinsley Street
Nashua, NH 03061

NEW LONDON

Kearsarge Counseling Center
Seamans Road
New London, NH 03257-1101

NEWPORT

West Central Services Counseling Center of Newport
167 Summer Street
Newport, NH 03773

PORTSMOUTH

Child and Family Services
1 Junkins Avenue
Portsmouth, NH 03801

Southeastern New Hampshire Services at Portsmouth
151 Court Street
Portsmouth, NH 03801

ROCHESTER

Southeastern New Hampshire Services
32 Wakefield Street
Rochester, NH 03867

WOLFEBORO

Carroll County Mental Health
Wolfeboro, NH 03894

NEW JERSEY

ABSECON

Family Service Association
312 East Whitehorse Pike
Absecon, NJ 08201

Thomas E. Hand Professional Associates
283 East Jimmie Leeds Road
Absecon, NJ 08201

ASBURY PARK

Jersey Shore Addiction Services, Inc. T/A Asbury Park Drug Treatment Center
1200 Memorial Drive
Asbury Park, NJ 07712

ATLANTIC CITY

Archway Associates for Life Enhancement
26 South New York Avenue
Atlantic City, NJ 08401

Atlanticare Behavioral Health
210-B Maryland Avenue
Atlantic City, NJ 08401

Institute for Human Development (IHD)
1315 Pacific Avenue
Atlantic City, NJ 08401

ATLANTIC HIGHLANDS

Matonti, Alane E., BSW CADC NCAC
64 7th Avenue
Atlantic Highlands, NJ 07716

BASKING RIDGE

Bresnahan, Jeremiah, ACSW CAC, and Maureen Bresnahan, MS CADC
36 Manchester Drive
Basking Ridge, NJ 07920

BAYONNE

Community Psychotherapy Associates
479 Avenue C
Bayonne, NJ 07002

New Pathway Counseling Services, Inc.
995 Broadway Street
Bayonne, NJ 07002

Private Counseling Service
510 Broadway Street
Bayonne, NJ 07002

BELLE MEAD

Carrier Foundation Addiction Unit
Belle Mead, NJ 08502

BELLEVILLE

Community Healthcare Network of Belleville, Bloomfield, Nutley
570 Belleville Avenue
Belleville, NJ 07109

Marriage and Family Counseling Center
387 Union Avenue
Belleville, NJ 07109

BLAIRSTOWN

Little Hill/Alina Lodge
Paulinskill River and Squires Road
Blairstown, NJ 07825

BOONTON

Saint Clare's Health Services
130Powerville Road
Boonton, NJ 07005

BOUND BROOK

Family Counseling Service of Somerset County Addiction Services
339 West 2nd Street
Bound Brook, NJ 08805-1833

BRANT BEACH

Saint Francis Community Center
4700 Long Beach Boulevard
Brant Beach, NJ 08008

BRICK

Ocean Counseling and Referral Services
35 Beaverson Boulevard
Lion's Head Office Park
Building 9B
Brick, NJ 08723

BRIDGETON

Cumberland County Alcoholism and Drug Treatment
72 North Pearl
Bridgeton, NJ 08302

Faith Farm, Inc.
21 Stretch Road
Bridgeton, NJ 08302

South Jersey Drug Treatment Center
Cumberland Drive
Bridgeton, NJ 08302

BRIDGEWATER

Catholic Charities Comprehensive Family Addiction Treatment
540–550 Route 22 East
Bridgewater, NJ 08807

Cedar House
520 North Bridge Street
Bridgewater, NJ 08807

Richard Hall CMHC Outpatient Substance Abuse Services
500 North Bridge Street
Bridgewater, NJ 08807

BURLINGTON

Catholic Charities of Burlington
206 High Street
Burlington, NJ 08016

Amity House for Men
1004 High Street
Burlington, NJ 08016

Family Enrichment Institute, Inc.
415 Keim Boulevard, Suite 1-B
Burlington, NJ 08016

CALDWELL

The Bridge, Inc.
14 Park Avenue
Caldwell, NJ 07006

CAMDEN

Camden County Division of Alcohol and Substance Abuse Step-Up Program
2600 Mount Ephraim Avenue
Camden, NJ 08104

Cooper House
225 South 6th Street
Camden, NJ 08103

Hispanic Family Center of Southern New Jersey La Esperanza
35 Church
Camden, NJ 08103

Sikora Center, Inc.
613-615 Clinton Street
Camden, NJ 08103

Substance Abuse Center of Southern Jersey, Inc.
413 Broadway
Segaloff Treatment Center
Camden, NJ 08103

CAPE MAY

Employee Care
Bank Street Commons
Suite 130
Cape May, NJ 08204

CAPE MAY COURT HOUSE

Burdette Tomlin Hospital Outpatient Counseling
Stone Harbor Boulevard
Route 9
Cape May Court House, NJ 08210

Cape May County Youth Shelter Substance Abuse Services
151 Crest Haven Road
Cape May Court House, NJ 08210

CEDAR GROVE

Turning Point, Inc.
125 Fairview Avenue
Cedar Grove, NJ 07009

CHERRY HILL

Kennedy Memorial Hospital/ Cherry Hill Division Substance Abuse Services/ Detox and Outpatient
Chapel Avenue and Cooperlanding Road
Cherry Hill, NJ 08034

UMDNJ/University Behavioral Health Care
498 Marlboro Avenue
Cherry Hill, NJ 08002

CLIFTON

Clifton Counseling Services
60 Hadley Avenue
Suite A
Clifton, NJ 07011

COLLINGSWOOD

Genesis Counseling Center Alcoholism Outpatient Services
636 Haddon Avenue
Collingswood, NJ 08108

CRANFORD

Catholic Community Services Mount Carmel Guild
505 South Avenue
Cranford, NJ 07016

DENVILLE

OPT Counseling Services
61 Broadway
Denville, NJ 07834

DOVER

Hope House Outpatient Services
19–21 Belmont Avenue
Dover, NJ 07802

EAST ORANGE

East Orange General Hospital Alcohol Rehab/Family Treatment
300 Central Avenue
East Orange, NJ 07018-2819

East Orange Substance Abuse Treatment Program
160 Halsted Street
East Orange, NJ 07018

Veterans' Affairs Medical Center
Drug Dependency Treatment Program
385 Tremont Avenue
East Orange, NJ 07019

EDISON

Edison Catholic Charities Substance Abuse Program
26 Safran Avenue
Edison, NJ 08837

JFK Center for Behavioral Health
65 James Street
Edison, NJ 08818

EGG HARBOR TOWNSHIP

Atlanticare Behavioral Health
6010 Black Horse Pike
Egg Harbor Township, NJ 08234

ELIZABETH

Bridgeway, Inc.
615 North Broad Street
Elizabeth, NJ 07208-3409

Elizabeth General Medical Center Substance Abuse Services
655 East Jersey Street
3rd Floor
Elizabeth, NJ 07201

Essex Substance Abuse Treatment Center Elizabeth Clinic
850 Woodruff Lane
Elizabeth, NJ 07201

Flynn Christian Fellowship
1089–1091 East Jersey Street
Elizabeth, NJ 07201

Proceed, Inc. Addiction Services
815 Elizabeth Avenue
Elizabeth, NJ 07201

Seton Center for Chemical Dependency
225 Williamson Street
Saint Elizabeth Hospital
Elizabeth, NJ 07207

ENGLEWOOD

Community Centers for Mental Health, Inc. Substance Abuse Services
93 West Palisade Avenue
Englewood, NJ 07631

The Van Ost Institute for Family Living, Inc.
150 East Palisade Avenue
Englewood, NJ 07631

Contini, Richard A.
26-07 Route 4
Fair Lawn, NJ 07410

FLEMINGTON

Catholic Charities Substance Abuse Services Care Program
6 Park Avenue
Flemington, NJ 08822

Good News Home for Women
33 Bartles Corner Road
Flemington, NJ 08822

Hunterdon Drug Awareness Program
8 Main Street
Suite 7
Flemington, NJ 08822

Hunterdon Medical Center Addictions Treatment Services
2100 Wescott Drive
Mental Health Center
Flemington, NJ 08822

Hunterdon Youth Services Inside Out Program
Rural Route 2
322 Highway 12
Flemington, NJ 08822

National Council on Alcoholism and Drug Dependence/ Hunterdon County
153 Broad Street
Flemington, NJ 08822

FORT DIX

McGuire Air Force Base Substance Abuse Program
305 MDOS/SGOMH
5250 New Jersey Avenue
Fort Dix, NJ 08640

FORT LEE

Behavioral Counseling Associates
1580 Lemoine Avenue
Suite 8
Fort Lee, NJ 07024

FORT MONMOUTH

Community Counseling Center
Building 864 Selfm-Ad
Fort Monmouth, NJ 07703

FREEHOLD

Freehold Community Counseling Service
30 Jackson Mills Road
Freehold, NJ 07728

Monmouth County Division of Social Services
Project Transition, Unit 505
Freehold, NJ 07728

New Hope Foundation, Inc. Outpatient
51 Throckmorton Street
Freehold, NJ 07728

GLASSBORO

Together, Inc. Drug Treatment Program
7 State Street
Glassboro, NJ 08028

HACKENSACK

Alternatives to Domestic Violence Substance Abuse Unit
21 Main Street
Room 111W
Hackensack, NJ 07601

Bergen County Div of Family Guidance Adolescent Substance Abuse Program
21 Main Street, Room 110
Hackensack, NJ 07602

Department of Health Services Addiction Recovery Program
151 Hudson Street
Hackensack, NJ 07601

Hackensack Medical Center Addiction Treatment Center/ Outpatient
60 2nd Street, First Floor
Hackensack, NJ 07601-1271

Monsignor Wall Social Service Center
149 Hudson Street
Hackensack, NJ 07601

HACKETTSTOWN

Hackettstown Community
Hospital Substance Abuse Department
651 Willow Grove Street
Hackettstown, NJ 07840

HADDONFIELD

Addiction Recovery Trt and Service
118 North Haddon Avenue
Haddonfield, NJ 08033-2306

HAZLET

Bradley, Carolyn A., LCSW CADC CPS
1 Bethany Road
Suite 30-A, Building 2
Hazlet, NJ 07730-1663

Women's Center of Monmouth
County, Inc. Outpatient Alcohol Counseling
1 Bethany Road
Building 3 Suite 42
Hazlet, NJ 07730

HOBOKEN

Saint Mary's CMHC Substance Abuse Unit
314 Clinton Street
Hoboken, NJ 07030

Saint Mary's Hospital Giant
Steps/Adolescent Substance Abuse Program
527 Clinton Street
Hoboken, NJ 07030

HOLMDEL

Bayshore Counseling Center
719 North Beers Street
Holmdel, NJ 07733

HOWELL

Howell Township Youth and Family Counseling Services
425 Adelphia Street
Howell, NJ 07731

IRVINGTON

L and L Clinics, Inc. Methadone Maintenance and Detox
57–59 New Street
Irvington, NJ 07111

JERSEY CITY

Catholic Community Services
249 Virginia Avenue
Jersey City, NJ 07304

Counseling Resources Center
176 Palisade Avenue
Jersey City, NJ 07306

Hogar Crea
79 Cornelison Avenue
Jersey City, NJ 07302

Jersey City Medical Center Dept of Psychiatry Addiction Services
50 Baldwin Avenue, 11 Center
Jersey City, NJ 07304-3154

Salvation Army Adult Rehab
Center/Inpatient and Outpatient
248 Erie Street
Jersey City, NJ 07302

Spectrum Health Care, Inc.
74–80 Pacific Avenue
Jersey City, NJ 07304

KEANSBURG

Awareness Counseling Drug and Alcohol Rehabilitation Center, Inc.
23 Church Street
Keansburg, NJ 07734

KEARNY

Inter County Council on Drug/
Alcohol Abuse Administration/
Drug Free Counseling
416 Kearny Avenue
Kearny, NJ 07032

KEYPORT

Endeavor House
6 Broadway
Keyport, NJ 07735

LAFAYETTE

Sunrise House Foundation, Inc. Alcohol Residential Program
Sunset Inn Road
Intersection of Routes 15 and 94
Lafayette, NJ 07848

LAKEWOOD

Counseling Center for Self Discovery
222 River Avenue
Route 9 South
Lakewood, NJ 08701

Preferred Behavioral Health of New Jersey
700 Airport Road
Lakewood, NJ 08701

LEONARDO

Middletown Office of Substance Abuse Services
900 Leonardville Road
Croydon Hall
Leonardo, NJ 07737

LINWOOD

Recovery Counseling Services
Office 1 Central Square
Linwood, NJ 08221

LIVINGSTON

Saint Barnabas Behavioral Health Network
5 Regent Street, Suite 522
Livingston, NJ 07039

LYNDHURST

Comprehensive Behavioral Health Center
516 Valley Brook Avenue
Lyndhurst, NJ 07071

MANALAPAN

Manalapan Community and Family Services
120 Route 522
Manalapan, NJ 07726

MARGATE CITY

Gegner, Murray, LCSW
210 North Rumson Avenue
Margate City, NJ 08402

MARLBORO

Discovery Institute for Addictive Disorders
Route 5, Cottage 15
Marlboro, NJ 07746

New Hope Foundation, Inc. Substance Abuse Services
Route 520
Marlboro, NJ 07746

MATAWAN

Community YMCA
166 Main Street
Matawan, NJ 07747

MAYS LANDING

Lighthouse/Recovery Services of New Jersey
5034 Atlantic Avenue
Mays Landing, NJ 08330

MEDFORD

Elm Lifelines
23 South Main Street, Suite 1
Medford, NJ 08055

Fox Counseling Associates
1 North Main Street
Suite 3B
Medford, NJ 08055

MENDHAM

Daytop Village
80 West Main Street
Mendham, NJ 07945

MILLTOWN

Trautz Associates
134 North Main Street
Milltown, NJ 08850

MONTCLAIR

Mountainside Hospital Alcoholism Treatment Unit
Bay and Highland Avenues
Montclair, NJ 07042

Cope Center, Inc.
104 Bloomfield Avenue
Montclair, NJ 07042

MORRIS PLAINS

New Views Treatment Program, Inc.
Central Avenue
Morris Plains, NJ 07950-9068

MORRISTOWN

Atlantic Behavioral Health Morristown Memorial Hospital Outpatient Addictive Service
95 Mount Kemble Avenue
Morristown, NJ 07962

Morris County Addictions Recovery Center
30 Schuyler Place, 2nd Floor
Morristown, NJ 07963-0900

Morristown Memorial Hospital Juvenile Evaluation and Treatment Services
100 Madison Avenue
Morristown, NJ 07960

Mrs. Wilson's Halfway House
56 Mount Kemble Avenue
Morristown, NJ 07960

MOUNT HOLLY

Amity House, Inc.
211 Garden Street
Mount Holly, NJ 08060

Burlington Comp. Counseling, Inc.
75 Washington Street
Mount Holly, NJ 08060

NEPTUNE

Jersey Shore Medical Center
1945 Highway 33
Neptune, NJ 07753

NEWARK

American Habitare
687 Frelinghuysen Avenue
Newark, NJ 07114

Choices, Inc.
169 Roseville Avenue
Newark, NJ 07107

Community United for Rehabilitation of Addiction, Inc. (CURA)
35 Lincoln Park
Newark, NJ 07102

Essex Substance Abuse Treatment Center, Inc.
164 Blanchard Street
Newark, NJ 07105

461 Frelinghuysen Avenue
Newark, NJ 07144

Integrity House, Inc.
103 Lincoln Park
Newark, NJ 07102

Mount Carmel Guild
Halfway House
56 Freeman Street
Newark, NJ 07105

Addiction Treatment Services
1160 Raymond Boulevard
Newark, NJ 07102

Newark Renaissance House, Inc. Youth and Family Treatment Center
62-80 Norfolk Street
Newark, NJ 07103

NEW BRUNSWICK

Damon House, Inc. Residential and Outpatient
105 Joyce Kilmer Avenue
New Brunswick, NJ 08901

New Brunswick Counseling Center
84 New Street
New Brunswick, NJ 08901

Open Door Alcoholism Treatment Program
2–4 Kirkpatrick and New Street
New Brunswick, NJ 08901

Program for Addictions Consultation and Treatment (PACT)
254 Easton Avenue
New Brunswick, NJ 08901

Saint Peter's Medical Center
Center for Treatment of Pregnancy and Addiction
288 Livingstone Avenue
New Brunswick, NJ 08903

NEW LISBON

Burlington County Health Department Post House
610 Pemberton/Browns Mills Road
New Lisbon, NJ 08064

NEWTON

Center for Mental Health
175 High Street
Newton, NJ 07860

Decide Program
35 High Street
Newton, NJ 07860

Newton Memorial Hospital
Alcohol and Substance Abuse Program
175 High Street
Newton, NJ 07860

Professional Counseling Associates
35 High Street
Newton, NJ 07860

Riser Sommer Tolliver Corp.
40 Park Place
Newton, NJ 07860

NORTH BERGEN

Palisades General Hospital Counseling Center
7101 Kennedy Boulevard
North Bergen, NJ 07047

NORTHVALE

Bergen County Community Action Program Ladder Project
35 Piermont Road, Building N
Northvale, NJ 07647

OLD BRIDGE

Extracare Health Services
201 Route 34
Old Bridge, NJ 08857

ORADELL

Professional Counseling Associates
370 Kinderkamack Road
Oradell, NJ 07649

ORANGE

City of Orange Drug/Alcohol Abuse Program
439 Main Street
Orange, NJ 07050

Family Connections
395 South Center Street
Orange, NJ 07050

PARAMUS

Bergen Pines County Hospital
Evergreen Treatment Center
Monsignor Wall Social Service Center
230 East Ridgewood Avenue
Paramus, NJ 07652

Mid-Bergen Mental Health Center
610 Industrial Avenue
Paramus, NJ 07652

PASSAIC

Hispanic Information Center Alcohol Outreach Program for Minorities
186 Gregory Street
Passaic, NJ 07055

PATERSON

Eva's Shelter and Kitchen
393 Main Street
Paterson, NJ 07505

Paterson Counseling Center, Inc.
321 Main Street
Paterson, NJ 07505

Straight and Narrow
508 Straight Street
Paterson, NJ 07501

PHILLIPSBURG

Catholic Charities/Warren ADAPT
700 Sayre Avenue
Phillipsburg, NJ 08865

Warren Hospital Alcohol/Drug Recovery Center/Detox
185 Roseberry Street
Phillipsburg, NJ 08865

Warren Hospital MICA Program/ Inpatient
185 Roseberry Street
Mental Health Unit 2 South
Phillipsburg, NJ 08865

PICATINNY ARSENAL

US Army Armament Resource Development Center Employee Assistance Office
Amsta AR MWR
Building 120
Picatinny Arsenal, NJ 07806-5001

PISCATAWAY

Specialized Addiction Services
667 Hoes Lane
Piscataway, NJ 08854

PLAINFIELD

Organization for Recovery
519 North Avenue
Plainfield, NJ 07060

Project Alert
930 Putnam Avenue
Plainfield, NJ 07060

Union County Psychiatric Clinic Adolescent Alcohol Program
117–119 Roosevelt Avenue
Plainfield, NJ 07062

Steps Recovery Center Muhlenberg Regional Medical Center
Park Avenue and Randolph Road
Plainfield, NJ 07060

POMPTON LAKES

Matthew E. Collins CDC CRPS
Counseling and Relapse Prevention Services
109 Beech Avenue
Pompton Lakes, NJ 07442

POMPTON PLAINS

New Bridge Service, Inc.
21 Evans Place
Pompton Plains, NJ 07444-1428

PRINCETON

Cornerhouse
369 Witherspoon Street
Valley Road Building
Princeton, NJ 08540

Family/Children Service of Central New Jersey Outpatient Alcoholism Counseling and Education
120 John Street
Princeton, NJ 08542

RANCOCAS

Hampton Behavioral Health Center
650 Rancocas Road
Rancocas, NJ 08073

RANDOLPH

Morris County Aftercare Center
Outpatient/Drug Free and Methadone
1574 Sussex Turnpike
Randolph, NJ 07869

RED BANK

CPC Behavioral Health Care
270 Highway 35
Red Bank, NJ 07701

Ruane, Mary Anne, MSW CAC
30 Linden Place
Red Bank, NJ 07701

Barbetta, Philip
30 Linden Place, Suite A-1
Red Bank, NJ 07701-1817

Riverview Medical Center Addiction Recovery Services
48 East Front Street
Red Bank, NJ 07701

RIDGEWOOD

Seligson, Henry, Ph.D. and Bryan Granelli, Ph.D.
112 Prospect Street
Ridgewood, NJ 07452

RINGWOOD

Sandra A. Carlson Counseling
11 Sunset Road
Ringwood, NJ 07456

RIVERSIDE

Zurbrugg Memorial Health Facility
Hospital Plaza
Riverside Division
Riverside, NJ 08075

RIVERTON

Healthmark Counseling
101 Route 130
Madison Building, Suite 321
Riverton, NJ 08077

SADDLE BROOK

High Focus Centers
299 Market Street, Suite 110
Saddle Brook, NJ 07663

SALEM

Maryville, Inc. Outpatient Services
567 Salem Quinton Road
Salem, NJ 08079

SEABROOK

Seabrook House
Polk Lane
Seabrook, NJ 08302

SECAUCUS

Integrity, Inc.
575 County Avenue, Building C-3
Secaucus, NJ 07096

SKILLMAN

Crawford House, Inc. Halfway House for Women Alcoholics
362 Sunset Road
Skillman, NJ 08558

SOMERS POINT

Amethyst Addictions Services
1409 Roberts Avenue
Somers Point, NJ 08244

SOMERVILLE

Samaritan Homeless Interim Program
67 West High Street
Somerville, NJ 08876

Somerset Medical Center
Specialized Treatment for Addictions Recovery Program (STAR)
111 Courtyard Drive
Somerville, NJ 08876

Somerset Treatment Services
256 East Main Street
Somerville, NJ 08876

SOUTH AMBOY

Hynes, Jack MA
South Amboy, NJ 08879

Stevens, Inc.
169 North Stevens Avenue
South Amboy, NJ 08879

Strathmore Treatment Associates
1 Lower Main Street, Route 35
South Amboy, NJ 08879

SOUTH RIVER

Memorial Medical Center
77 Water Street
South River, NJ 08882

SPRINGFIELD

Overlook Hospital Addictive Health System
530 Morris Avenue
Springfield, NJ 07081

SUMMIT

Charter Behavioral Health Systems of New Jersey
19 Prospect Street
Summit, NJ 07901

TOM'S RIVER

Alternatives Counseling Center, Inc.
96 East Water Street
Tom's River, NJ 08754

Counseling and Referral Services of Ocean County, Inc.
247 Main Street
Toms River, NJ 08753

Easter Seal Substance Abuse Treatment Services
1595 Route 9
Toms River, NJ 08755

Healy Counseling Associates
1108 Hooper Avenue
Tom's River, NJ 08753

TRENTON

Catholic Charities Alcoholism/ Addictions Program
47 North Clinton Avenue
Trenton, NJ 08607

Family Guidance Center of
Mercer County Substance Abuse Recovery Program
2300 Hamilton Avenue
Trenton, NJ 08619

Fort Dix Community Counseling Center
Building 5203
Maryland Avenue
Trenton, NJ 08640-5140

Greater Trenton CMHC Outpatient MICA Services
132 North Warren Street
Trenton, NJ 08607

Mercer Street Friends Center Outpatient Drug and Alcohol Treatment Service
1201 West State Street
Trenton, NJ 08618

New Horizon Treatment Services, Inc.
132 Perry Street
2nd Floor
Trenton, NJ 08618

Rescue Mission of Trenton
98 Carroll Street
Trenton, NJ 08604

United Progress, Inc. Detoxification Center
541 East State Street
Trenton, NJ 08609

VENTNOR CITY

Jewish Family Services Addiction Services
3 South Weymouth Avenue
Ventnor City, NJ 08406

VINELAND

Hendricks House for Men
542 Northwest Boulevard
Vineland, NJ 08360

Lloyd Reynolds Associates
733 Elmer Street
Vineland, NJ 08360

VOORHEES

Reality House, Inc.
1 Alpha Avenue
Suite 43
Voorhees, NJ 08043

WALL

Wall Youth Center and Community Services
1824 South M Street
Wall, NJ 07719

WASHINGTON

Family Guidance Center of
Warren Outpatient Substance Abuse Treatment Program
492 Route 57 West
Washington, NJ 07882

WAYNE

Wayne Counseling Center
475 Valley Road
Wayne, NJ 07470

WEST NEW YORK

Mental Health and Addictive Services
5301 Broadway Street
West New York, NJ 07093

WESTVILLE

Maryville Alcoholism Rehab Center Outpatient Program
156 Broadway
Westville, NJ 08093

WHITING

America's Keswick Keswick Colony Division
601 Route 530
Whiting, NJ 08759

WILDWOOD

Cape Counseling Services, Inc. Drug and Alcohol Unit
2604 Pacific Avenue
Wildwood, NJ 08260

WILLIAMSTOWN

Maryville, Inc.
1403 Grant Avenue
Williamstown, NJ 08094

WOODBURY

Services to Overcome Drug
Abuse Among Teenagers of New Jersey, Inc. (SODAT, Inc.)
124 North Broad Street
Woodbury, NJ 08096

NEW MEXICO

ALAMOGORDO

Counseling Center, Inc.
1900 East 10th Street
Alamogordo, NM 88310

Otero County Council on Alcohol Abuse and Alcoholism
850 Wright Road
Alamogordo, NM 88310

ALBUQUERQUE

Albuquerque Health Care for the Homeless
805 Tijenas Street
Albuquerque, NM 87102

Aliviar Counseling Service
1121 Kent NW
Albuquerque, NM 87102

All Indian Pueblo Council, Inc. Two Worlds Project
3939 San Pedro Street NE
Suite D
Albuquerque, NM 87190

Charter Heights Behavioral Health Services Substance Abuse Services
103 Hospital Loop NE
Albuquerque, NM 87108

Citizens' Council on Alcoholism and Drug Abuse
7711 Zuni Road SE
Albuquerque, NM 87108

Conflict Management, Inc.
3900 Georgia Street NE
Albuquerque, NM 87110

Counseling and Psychotherapy Institute
803 Tijeras Street NW
Albuquerque, NM 87194

Hogares, Inc.
1218 Griegos Road NW
Albuquerque, NM 87107

Kaseman Presbyterian
8300 Constitution Street NE
Albuquerque, NM 87110

Lifestyle Recovery
3306 4th Street NW
Albuquerque, NM 87107

Lovelace Park Center Substance Abuse Services
5655 Jefferson Street NE
Albuquerque, NM 87109

Memorial Hospital Addictive Disease Program
806 Central Street SE
Albuquerque, NM 87102

New Mexico Monitored Treatment Program
9204 Menaul Boulevard NE
Suite 6
Albuquerque, NM 87112

Saint Martin's Hospitality Center
1201 3 Street
Albuquerque, NM 87125

Turquoise Lodge
6000 Isleta Boulevard SW
Albuquerque, NM 87105

University of New Mexico
Milagro Program
1007 Stanford Road NE
Albuquerque, NM 87131

Center on Alcoholism Substance Abuse and Addictions
2350 Alamo Drive SE
Albuquerque, NM 87106-3202

Veterans' Affairs Medical Center
Substance Abuse Treatment Program
2100 Ridgecrest Drive SE
Albuquerque, NM 87108

Western Clinical Health Services of New Mexico
Silver Street Clinic
4105 Silver Street SE
Albuquerque, NM 87108

ANTHONY

BERNALILLO

Five Sandoval Indian Pueblos, Inc.
1043 Highway 313
Bernalillo, NM 87004

La Buena Vida, Inc.
872 Camino Del Pueblo
Bernalillo, NM 87004

CARLSBAD

Carlsbad Mental Health Association Villa de Esperanza
914 North Canal Street
Carlsbad, NM 88220

CLAYTON

Golden Spread Rural Frontier Coalition
200 Aspen Street
Clayton, NM 88415

CLEVELAND

Rio Grande Treatment Center
Cleveland, NM 87715

CLOVIS

Mental Health Resources, Inc.
919 Rencher Street
Clovis, NM 88101

CROWNPOINT

Navajo Nation Behavioral Health Services]
Crownpoint, NM 87313

CUBA

Presbyterian Medical Services Cuba Health Center
State Road 44
Cuba, NM 87013

DEMING

Border Area Mental Health Services
901 West Hickory Street
Deming, NM 88030

DULCE

Jicarilla Apache Tribe Multi Service Center
Jicarilla Reservation, Building 23
Dulce, NM 87528

EMBUDO

Rio Grande Alcoholism Treatment Program, Inc.
Embudo, NM 87531

ESPANOLA

Ayudantes, Inc. Espanola Northern Clinic
810-F Riverside Drive
Espanola, NM 87533

Hoy Alcoholism Program
1102-A North Paseo De Onate
Espanola, NM 87532

FARMINGTON

Presbyterian Medical Services
Community Counseling Center
1001 West Broadway
Farmington, NM 87410

San Juan Detoxification Services
Four Winds Addiction Recovery Center
1313 Mission Avenue
Farmington, NM 87401

FORT BAYARD

Fort Bayard Medical Center
Yucca Lodge
Fort Bayard, NM 88036

GALLUP

Na Nihzhoozhi Center, Inc. (NCI)
2205 East Boyd Street
Gallup, NM 87301

Rehobeth McKinley Christian Health Care Services
650 Vanden Bosch Parkway
Gallup, NM 87301

GRANTS

Valencia Counseling Services, Inc. Cibola Counseling
210 East Santa Fe Street
Grants, NM 87020

HOBBS

Guidance Center of Lea County Treatment Center
920 West Broadway
Hobbs, NM 88240

Palmer Drug Abuse Program of Lea County
200 East Snyder Street
Hobbs, NM 88241

HOLLOMAN AFB

Holloman Air Force Base Substance Abuse Program
49 MDOS/SGOMH
1022 Fifth Street
Holloman AFB, NM 88330-8039

ISLETA

Pueblo of Isleta Alcoholism and Drug Program
Isleta, NM 87022

JEEZ PUEBLO

Behavioral Health Program
Jemez Pueblo, NM 87024

LAGUNA

Pueblo of Laguna Service Center
Laguna, NM 87026

LAS CRUCES

DWI Drug Court Treatment Program
642 South Alameda Street
Las Cruces, NM 88005

Families and Youth, Inc.
221 North Downtown Mall
Las Cruces, NM 88004

Mesilla Valley Hospital Residential Unit
3751 Del Rey Boulevard
Las Cruces, NM 88005

Southwest Counseling Center, Inc.
2401 South Espina Street
Las Cruces, NM 88005

Serenity House
1050 Monte Vista Avenue
Las Cruces, NM 88001

LAS VEGAS

Ayudantes, Inc.
803 Grand Avenue
Las Vegas, NM 87701-4252

LORDSBURG

Border Area Mental Health Services Lordsburg Counseling Center
500 East 13th Street
Medical Complex
Lordsburg, NM 88045

LOS ALAMOS

Los Alamos Family Council
1505 15th Street, Suite A
Los Alamos, NM 87544

LOS LUNAS

Valencia Counseling Services
735 Don Pasqual Road
Los Lunas, NM 87301

LOVINGTON

Guidance Center of Lea County, Inc.
1115 West Avenue, Suite D
Lovington, NM 88260

MAGDALENA

Alamo Alcoholism Program Outpatient and Prevention
Alamo Navajo Reservation
Magdalena, NM 87825

MESCALERO

Mescalero Tribal Human Services
107 Sunset Loop
Mescalero, NM 88340

MORA

Helping Hands, Inc.
North 15 Highway
Mora, NM 87732

PORTALES

Mental Health Resources, Inc. Substance Abuse Services/ Outpatient
300 East First Street
Portales, NM 88130

QUESTA

Presbyterian Medical Services Questa Health Center
Questa, NM 87556

RAMAH

Ramah Navajo Behavioral Health Services
Southside of Pinehill Street School Campus
Ramah, NM 87321

ROSWELL

Counseling Associates, Inc.
109 West Bland Street
Roswell, NM 88201

New Mexico Rehabilitation Center Chemical Dependency Unit
31 Gail Harris Avenue
Roswell, NM 88201

RUIDISO DOWNS

The Counseling Center, Inc. Substance Abuse Services
206 Sudderth Drive
Ruidoso Downs, NM 88346

SAN FELIPE PUEBLO

San Felipe Behavioral Health Substance Abuse and Prevention Program
San Felipe Pueblo Street
San Felipe Pueblo, NM 87001

SAN FIDEL

Acoma Canoncito Laguna Hospital New Sunrise Regional Treatment Center
San Fidel, NM 87049

SAN JUAN PUEBLO

Delancey Street/New Mexico, Inc.
40 Old Alcalde Road
San Juan Pueblo, NM 87566

Eight Northern Indian Pueblos Behavioral Health Program
Lower Alcada Road
San Juan Pueblo, NM 87566

SANTA FE

Ayudantes, Inc. Santa Fe Northern Clinic
1316 Apache Street
Santa Fe, NM 87504

Life Link
2325 Cerrillos Road
Santa Fe, NM 87505

Pinon Hills Hospital Substance Abuse Services
313 Camino Alire
Santa Fe, NM 87501

Recovery of Alcoholics Program, Inc.
4100 Lucia Lane
Santa Fe, NM 87505

Saint Vincent Hospital Substance Abuse Services
455 Saint Michael's Drive
Santa Fe, NM 87501

SANTA ROSA

Greater Santa Rosa Council on Alcohol
The Sure House
130 South 4th Street
Santa Rosa, NM 88435

SANTA TERESA

Alliance Hospital of Santa Teresa Rio Valle
100 Laurel Court
Santa Teresa, NM 88008

SANTO DOMINGO PUEBLO

Santo Domingo Substance Abuse Program
San Ildefonso Street
Santo Domingo Pueblo, NM 8705

SHIPROCK

Four Corners Regional Adolescent Treatment Center
Yucca Street Dorm 2
Shiprock, NM 87420

Shiprock Outpatient Treatment Center
Old PHS Hospital Building
Shiprock, NM 87420

SILVER CITY

Border Area Mental Health Substance Abuse Services
315 South Hudson Street
Silver City, NM 88061

SOCORRO

Socorro Mental Health Foundation
204-B Heel Avenue
Socorro, NM 87801

TAOS

Taos Alcohol and Drug Program
413 Sipapu Road
Taos, NM 87571

TRUTH OR CONSEQUENCES

Southwest Counseling Center, Inc.
118 Broadway
Truth Or Consequences, NM 87901-2830

TUCUMCARI

Mental Health Resources, Inc.
300 South 2nd Street
Tucumcari, NM 88401

ZUNI

Teambuilders Counseling Services, Inc.
Tucumcari, NM 88401

NEW YORK

ALBANY

Albany Citizens Council on
Alcohol and Other Chemical Dependence, Inc.
Alcohol Crisis Center
75 New Scotland Avenue Unit G
Capital District Psychiatric Center
Albany, NY 12208

90 McCarty Avenue
Albany, NY 12202

Albany County Substance Abuse Prevention Clinic
845 Central Avenue East 1
Albany, NY 12206

Altamont Program Inc
575 Broadway Street
Albany, NY 12204

Arbor Hill Alcoholism Program
(AHAP) Supportive Living Facility
250 Clinton Avenue
Albany, NY 12206

Eight Twenty River Street, Inc. Eleanor Young Clinic
134 Franklin Street
Albany, NY 12202

Equinox Counseling Center
306 Central Avenue
Albany, NY 12210

Hospitality House Therapeutic Community, Inc./Residential
271 Central Avenue
Albany, NY 12206

La Salle School, Inc.
391 Western Avenue
Albany, NY 12203

Pearl Street Counseling Center, Inc. Drug Free Clinic
42 South Pearl Street
Albany, NY 12207

Saint John's Project Lift, Inc.
Alcoholism Community Residence
37 South Ferry Street
Albany, NY 12202

Drug Abuse Services
45 South Ferry Street
Albany, NY 12202

Saint Peter's Addiction Recovery Center (SPARC)
Acute Care Unit
315 South Manning Boulevard
Cusack Pavilion
Albany, NY 12208

64 2nd Avenue
Albany, NY 12202

The Next Step, Inc. Recovery Home for Women
276 Sherman Street
Albany, NY 12206

Trinity Institution Homer Perkins Center, Inc.
76-82 2nd Street
Albany, NY 12210

Visiting Nurse Assoc. of Albany, Inc. Geriatric Alcohol Program
35 Colvin Avenue
Albany, NY 12206

Whitney M. Young, Jr. Health Center, Inc.
Family Alcoholism/Chemical Dependency Treatment Services
900 Lark Drive
Albany, NY 12207

Rehabilitation Clinic Methadone Maintenance Treatment Program
10 Dewitt Street
Albany, NY 12207-1306

ALBION

Unity Behavioral Health Chemical Dependency Services
168 South Main Street
Medical Arts Center
Albion, NY 14411

ALDEN

Brylin Hospitals Addiction Medical Services
11438 Genesee Street
Alden, NY 14004

ALTAMONT

Eight Twenty River Street, Inc. The Altamount House/Alcohol Inpatient Rehab
1180 Berne Altamont Road Route 156
Altamont, NY 12009

AMHERST

Sisters of Charity Hospital Star Outpatient Services
4512 Main Street
Amherst, NY 14226

AMITYVILLE

Long Island Home at South Oaks Hospital
Alcoholism Outpatient and Drug Clinic
Bailey House Alcohol Inpatient Detox Unit
Robbins Inpatient Alcoholism Rehab Center
400 Sunrise Highway
Amityville, NY 11701

Town of Babylon Division of Drug And Alcohol Services
400 Broadway
Amityville, NY 11701

AMSTERDAM

Saint Mary's Hospital
Alcoholism Inpatient Rehab Program
427 Guy Park Avenue
Amsterdam, NY 12010

Comprehensive Alcohol Outpatient Clinic
76 Guy Park Avenue
Amsterdam, NY 12010

APPLETON

Fellowship House, Inc. Somerset House/Alcohol Halfway House
7397 Lake Road
Appleton, NY 14008

ASTORIA

Hanac Substance Abuse Program
31-14 30 Avenue
Astoria, NY 11102

AUBURN

Recovery Counseling Services Alcoholism Outpatient Clinic
188 Genesee Street
Auburn, NY 13021

Unity House of Cayuga County, Inc. Grace House
56 Osborne Street
Auburn, NY 13021

Confidential Help for Alcohol Drugs (CHAD)
Alcoholism Outpatient Clinic
75 Genesee Street
Piccolo Building
Auburn, NY 13021

BABYLON

Crossings Recovery Program, Inc.
Crossings Alcoholism Outpatient Clinic
133 East Main Street
Berger Professional Plaza Suite 1B
Babylon, NY 11702

BALDWIN

Baldwin Council Against Drug Abuse (BCADA) Outpatient Drug Free
950 Church Street
Baldwin, NY 11510

BALDWINSVILLE

Confidential Counseling and Evaluation Services
2115 Downer Street
Baldwinsville, NY 13027

BALLSTON SPA

Clinical Services and Consulting, Inc. Ballston SPA Alcohol Clinic
433 Geyser Road
Ballston Spa, NY 12020

Hedgerow House
994 Route 67
Ballston Spa, NY 12020

BARRYVILLE

New Hope Manor, Inc. Residential Unit
35 Hillside Road
Barryville, NY 12719

Veritas Therapeutic Community, Inc. Lucy Rudd House
375 Route 55
Barryville, NY 12719

BATAVIA

Genesee Council on Alcohol and Substance Abuse, Inc.
Drug Abuse Services
Substance Abuse Outpatient
30 Bank Street
Batavia, NY 14020

Mercy Hall Chemical Dependency Treatment Program
16 Bank Street
Batavia, NY 14020

BATH

Kinship Community Residence
130 Rumsey Street
Bath, NY 14810

Steuben County Alcoholism and Substance Abuse Services
115 Liberty Street
Bath, NY 14810

BAY SHORE

Family Consultation Service, Inc. Family Alcoholism Treatment Center
38 Park Avenue
Bay Shore, NY 11706

Southside Hospital Substance Abuse Detoxification Services
Montauk Highway
Bay Shore, NY 11706

BAYSIDE

Long Island Jewish Hillside
Medical Center Family
Treatment Program Outpatient Alcohol Clinic
212-02 41st Avenue
Bayside, NY 11364

BEACON

Saint Francis Hospital
Alcohol Outpatient Clinic
Turning Point/Acute Care
Turning Point/Inpatient Rehabilitation
60 Delavan Avenue
Beacon, NY 12058

Beacon Counseling Center
223 Main Street
Beacon, NY 12508

BEDFORD HILLS

Renaissance Project, Inc. Bedford Hills Unit
524-26 North Bedford Road
Bedford Hills, NY 10507

BELLPORT

Outreach Development Corporation Outreach Project
11 Farber Drive, Unit D
Bellport, NY 11713

BETHPAGE

Bethpage Adolescent Development Associates (BADA)
936 Stewart Avenue
Bethpage, NY 11714

Bridge Back to Life Center, Inc. Drug Abuse Outpatient Clinic
4271 Hempstead Turnpike
Bethpage, NY 11714

BINGHAMTON

Addictions Center of Broome County, Inc.
455 State Street
Binghamton, NY 13901

Alternatives Counseling Center, Inc.
37 Mill Street
Binghamton, NY 13903

Broome County Chemical Dependency Services
168 Water Street
Binghamton, NY 13901

Fairview Recovery Services
Alcohol Crisis Center
247 Court Street
Binghamton, NY 13904

Fairview Halfway House
110 Fairview Avenue
Binghamton, NY 13904

Merrick Halfway House
1 Merrick Street
Binghamton, NY 13904

United Health Services, Inc.
New Horizons Alcohol Inpatient Rehab Unit
New Horizons Detox Program
New Horizons Chemical Dependency
Mitchell Avenue
Binghamton General Hospital
Binghamton, NY 13903

YWCA Clear Visions for Women Halfway House
80 Hawley Street
Binghamton, NY 13901

BLAUVELT

Daytop Village, Inc. Rockland Outreach Center
620 Route 303
Blauvelt, NY 10913

BOHEMIA

Catholic Charities (Talbot House) Alcohol Crisis Center
30-C Carlough Road
Bohemia, NY 11716

BOICEVILLE

Catskill Mountain Counseling
4080 Route 28
Boiceville, NY 12412

BRADFORD

Kinship House
3261 State Route 226
Bradford, NY 14815

BRENTWOOD

A Program Planned For Life Enrichment, Inc. (APPLE)
600 Suffolk Avenue, Suite A
Brentwood, NY 11717

Charles K Post Addiction Treatment Center
Pilgrim Psychiatric Center
Building 1
Brentwood, NY 11717

Outreach Development Corporation Outreach II
400 Crooked Hill Road
Brentwood, NY 11717

Town of Islip Dept. of Human Services Access
Division of Drugs and Alcohol
452 Suffolk Avenue
Brentwood, NY 11717

BRIDGEHAMPTON

Catholic Charities of Rockville Centre Outpatient Alcohol Clinic
2442 Main Street
Bridgehampton, NY 11932

BRONX

Albert Einstein College of Medicine Division of Substance Abuse
Melrose Unit
1764 Randall Avenue
Bronx, NY 10473

HUB 1/2/3
368 East 149 Street
Bronx, NY 10455

Yeshiva University/Melrose
260 East 161 Street
Bronx, NY 10451

Trailer 1
1500 Waters Place
Bronx, NY 10461

Van Etten Hospital Clinic
Morris Park and Seminole Avenue
Wing A
Bronx, NY 10461

Alternatives Youth Programs Chemical Dependency for Youth Clinic
324 East 149th Street
1st and 2nd Floor
Bronx, NY 10455

Argus Community Inc.
Harbor House
402 East 156th Street
Bronx, NY 10456

Basics/Franklin House
1064 Franklin Avenue
Bronx, NY 10456

Bronx Alcoholism Treatment
Center Alcoholism Rehabilitation Unit
1500 Waters Place
Building 13
Bronx, NY 10461

Bronx Citizens Committee, Inc.
1668 Webster Avenue
Bronx, NY 10457

City Probation Programs
480 East 185th Street
Bronx, NY 10458

Bronx/Lebanon Hospital Center
Alcoholism Halfway House
Alcoholism Outpatient Clinic
321 East Tremont Street
Bronx, NY 10457

Alcoholism Halfway House
742-44 Kelly Street
Bronx, NY 10456

Alcoholism Inpatient Rehab
Dept. of Psychiatry Detox Unit
1276 Fulton Avenue
Bronx, NY 10456

Methadone Maintenance
Treatment Program/KEEP
3100 3rd Avenue
Bronx, NY 10451

City Probation Programs
480 East 185th Street
Bronx, NY 10458

Concourse Medical Methadone Treatment Clinic
880 Morris Avenue
Bronx, NY 10451

Cosmetic Executive Women Residence at Casa Rita
284 East 151st Street
Bronx, NY 10451

Daytop Village, Inc.
Medically Supervised Drug Clinic
Bronx, NY 10461

Dr. Martin Luther King, Jr. Health Center Alcoholism Outpatient Clinic
3565 3rd Avenue, Suite B-1
Bronx, NY 10456

Hunt's Point Multi-Service
Substance Abuse Treatment Program
785 Westchester Avenue
Bronx, NY 10455

Alcoholism Outpatient Clinic
Chemical Dependency Probation Program
630 Jackson Avenue
Bronx, NY 10455

Jacobi Medical Center Comprehensive Alcoholism Treatment Center
Morris Park Avenue and Seminole Avenue
Bronx, NY 10461

La Casita
834 East 156th Street
Bronx, NY 10455

Learning for the Living Center
760 East 160th Street
Bronx, NY 10456

Lincoln Medical and Mental
Health Center Alcoholism Outpatient Clinic
349 East 140 Street
Bronx, NY 10454

Montefiore Medical Center
SATP Unit I
3550 Jerome Avenue
Bronx, NY 10467

SATP Unit II
SATP Unit III
2005 Jerome Avenue
Bronx, NY 10453

Mrs A's Day Program
966 Prospect Avenue
Bronx, NY 10459

Narco Freedom, Inc.
Children and Families Together
391 East 149th Street
Bronx, NY 10455

Key Extended Entry Program
487 Willis Avenue
Bronx, NY 10455

Alternatives Drug/Free Treatment Prog
Independence Alcohol Treatment Program
477–479 Willis Avenue
Bronx, NY 10455

Methadone Maintenance
Treatment Program
477–479 Willis Avenue
Bronx, NY 10455

250 Grand Concourse
1st Floor
Bronx, NY

Regeneration Women and Children Residential Treatment Program
2640-2652 3rd Avenue
2nd Floor
Bronx, NY 10454

Neighborhood Youth and Family Services
4137 3 Avenue
Bronx, NY 10457

Osborne Association Treatment Services
807-09 Westchester Avenue
Bronx, NY 10455

Our Lady of Mercy Medical Center
4401 Bronx Boulevard
Bronx, NY 10470

Phoenix House
Phelan Place
1851 Phelan Place
Bronx, NY 10453

Police Athletic League, Inc. Youhlink Program
2255 Webster Avenue, 3rd Floor
Bronx, NY 10457

Project Return Foundation, Inc.
Discovery Program
Exdous House Homeless Unit
1600 Macombs Road
Bronx, NY 10452

Womens Day Treatment Program
1484 Inwood Avenue 1st Floor
Bronx, NY 10452

Promesa, Inc.
Drug Treatment
1776 Clay Avenue
Bronx, NY 10457

Riverdale Mental Health Association
5676 Riverdale Avenue
Bronx, NY 10471

Saint Barnabas Hospital
Alcohol Detox Program
Alcoholism Outpatient Rehab Program
3rd Avenue and East 183rd Street
Bronx, NY 10457

Methadone Maintenance Treatment Program
4535-39 3rd Avenue
Bronx, NY 10457

Samaritan Village, Inc. Residential Drug Free Program
1381 University Avenue
Bronx, NY 10452

Scan New York Volunteer Parent
Aides Assoc. Family Renewal Center Drug Abuse Treatment
1075 Grand Concourse
Bronx, NY 10452

Soundview Throgs Neck CMHC
1967 Turnbull Avenue
Bronx, NY 10473

South Bronx Mental Health
Council, Inc. CMHC Alcoholism Outpatient Clinic
1241 Lafayette Street
Bronx, NY 10474

Sports Foundation, Inc.
391 East 149 Street
Room 317
Bronx, NY 10455

Tri-Center, Inc. Drug Abuse Treatment
2488 Grand Concourse
Bronx, NY 10458

Veterans Affairs Medical Center Substance Abuse Program
130 West Kingsbridge Road
Bronx, NY 10468

VIP Community Services Drug Free Day Treatment
770 East 176th Street
Bronx, NY 10460

Methadone Maintenance Treatment Program
1910 Arthur Avenue, 7th Floor
Bronx, NY 10457

VIP Women's Residence
1946 Bathgate Avenue, 4th Floor
Bronx, NY 10457

Vocational Instruction Project Community Services Alcoholism Halfway House
671 East 231st Street
Bronx, NY 10466

Willow Shelter Program
781 East 135th Street
Bronx, NY 10454

BROOKLYN

ARTC Brooklyn
Medically Supervised Outpatient/ Probation
937 Fulton Street
Brooklyn, NY 11238

Brooklyn Clinic 11/Fort Greene
937 Fulton Street
Brooklyn, NY 11238

Brooklyn Clinic 13/Bushwick
1149-55 Myrtle Avenue
Brooklyn, NY 11206

Brooklyn Clinic 14/Brownsville
494 Dumont Avenue
Brooklyn, NY 11207

Bedford Stuyvesant Comprehensive Alcoholism Treatment Center
1121 Bedford Avenue
Brooklyn, NY 11216

Bensonhurst Mental Health Clinic, Inc.
Drug Abuse Services
Outpatient/Prevention
86-20 18 Avenue
Brooklyn, NY 11214

Beth Israel Medical Center MMTP
Cumberland Clinic
98 Flatbush Avenue
Brooklyn, NY 11217

Break Free Russian Adolescent Project Midwood Adolescent Project
2020 Coney Island Avenue
Brooklyn, NY 11223

Bridge Back to Life Center, Inc.
6823 5th Avenue
Brooklyn, NY 11220

Builders for Family and Youth Flatbush Addiction Treatment Center
1463 Flatbush Avenue
Brooklyn, NY 11210

Canarsie Aware, Inc.
Day Service
Outpatient/Prevention
1310 Rockaway Parkway
Brooklyn, NY 11236

Church Avenue Merchants' Block
Assoc., Inc. Drug Abuse Prevention Services
2211 Church Avenue
Brooklyn, NY 11226

Coney Island Hospital
Alcoholism and Drug Treatment Program
2601 Ocean Parkway
Brooklyn, NY 11235

Counseling Service of Eastern District New York, Inc.
186 Montague Street
Brooklyn, NY 11201

Cumberland Diagnostic and
Treatment Center Alcoholism Treatment Program
100 North Portland Avenue
Brooklyn, NY 11205

CSEDNY REDI Program
185 Montague Street, 4th Floor
Brooklyn, NY 11201

Damon House New York, Inc.
Bushwick Homeless Drug Abuse Residential Center
1154-1156 Dekalb Avenue
Brooklyn, NY 11221

Williamsburg Homeless Program
310 South First Street
Brooklyn, NY 11211

Daytop Village, Inc. Brooklyn Outreach Center
401 State Street
Brooklyn, NY 11201

Discipleship Outreach Ministries
Exodus Treatment Center
5220 4th Avenue
Brooklyn, NY 11220

District 3 Youth and Adult, Inc. Outpatient Drug Free
271 Melrose Street
Brooklyn, NY 11206

EL Regreso Foundation, Inc.
Drug Abuse Treatment
189–191 South 2 Street
Brooklyn, NY 11211

232 Metropolitan Avenue
Brooklyn, NY 11211

Health Science Center Brooklyn/Kings County
Polydrug Unit 1
600 Albany Avenue
Building K Box 9 Code 26
Brooklyn, NY 11203

HHC New York City Kings County Hospital
600 Albany Avenue
Brooklyn, NY 11204

HHC/Woodhill Medical CMH
Center Chemical Dependency Services Drug Detox Unit
760 Broadway
Brooklyn, NY 11206

Interfaith Medical Center
1545 Atlantic Avenue
Brooklyn, NY 11213

Bushwick Clinic
Methadone Maintenance Treatment Program
555 Prospect Place
Ambulatory Building
Brooklyn, NY 11238

Kings County Hospital Center
Acute Detox
Alcohol Outpatient
600 Albany Avenue
Brooklyn, NY 11203

Comprehensive Alcoholism Outpatient Clinic
591 Kingston Avenue
Brooklyn, NY 11203

Kingsboro Addiction Treatment Center
754 Lexington Avenue
Brooklyn, NY 11215

Long Island College Hospital
Outpatient Clinic
255 Duffield Street
Brooklyn, NY 11201

Lutheran Medical Center
Alcoholism Outpatient Clinic
Drug Abuse Treatment
514 49 Street
Brooklyn, NY 11220

Acute Care Addiction Program
150 55th Street
Brooklyn, NY 11220

Mid-Brooklyn Health Society, Inc. Alcohol Crisis Center
599 Ralph Avenue
Brooklyn, NY 11233

Narco Freedom/Court Street Clinic
217 Court Street
Brooklyn, NY 11201

New Directions Alcohol and Substance Abuse Treatment Program
202-206 Flatbush Avenue
Brooklyn, NY 11217

NYC Department of Probation Tri Center Unit III
175 Remsen Street
Brooklyn, NY 11201

Paul J. Cooper Center for Human
Services Outpatient Alcoholism Clinic
106 New Lots Avenue
Brooklyn, NY 11212

Saint Martin de Porres Alabama Avenue Clinic
480 Alabama Avenue
Brooklyn, NY 11207

Saint Mary's Hospital
Substance Abuse Treatment
635 Classon Avenue
Brooklyn, NY 11238

1480 Prospect Place
Brooklyn, NY 11213

229 Powell Street
Brooklyn, NY 11212

Saint Vincent's Services, Inc. Alcoholism Outpatient Clinic
333 Atlantic Avenue, 1st Floor
Brooklyn, NY 11201

Serendipity
977 Bedford Avenue
Brooklyn, NY 11205

South Brooklyn Medical Administrative Services Methadone Maintenance Program
685 3 Avenue
Brooklyn, NY 11232

SUNY Health Science Center of Brooklyn Family Youth Center
604 Winthrop Street
Building F, 5th Floor
Brooklyn, NY 11203

Villa II, Inc. Alcoholism and Drug Abuse Outpatient Clinic
175 Remsen Street, 10th Floor
Brooklyn, NY 11201

Tri-Center, Inc. Drug Abuse Treatment
175 Remsen Street
Brooklyn, NY 11201

Urban Resource Institute Marguerite Saunders Urban Center for Alcohol Services
937 Fulton Street
Brooklyn, NY 11238

Victim Services Agency Outpatient Drug Abuse Clinic
3021 Atlantic Avenue
Brooklyn, NY 11208

Woodhull Medical and Mental Center
Chemical Dependency Services
Alcohol and Drug Detox Unit
760 Broadway
Brooklyn, NY 11206

BUFFALO

Alcohol and Drug Dependency Services Inc.
Alcohol Crisis Program
Inpatient Rehabilitation Services
291 Elm Street
Buffalo, NY 14203

Casa de Vita Halfway House/ Women
200 Albany Street
Buffalo, NY 14213

Chemical Dependency Program for Youth/LT
920 Harlem Road
Buffalo, NY 14224

Outpatient Clinic
210 Franklin Street
Buffalo, NY 14202

Men's Halfway House
2025 Broadway
Buffalo, NY 14213

Beacon Center Alcoholism and Drug Outpatient Clinic
695 Ellicot Square
Buffalo, NY 14150

Brylin Hospitals, Inc.
Drug Abuse Treatment Unit
Outpatient Unit
2625 Delaware Avenue
Buffalo, NY 14216

Williamsville Outpatient Clinic
5225 Sheridan Drive
Buffalo, NY 14221

Buffalo General Health Care System
Addiction Services
80 Goodrich Street
Buffalo, NY 14203

Deconess Center Alcoholism Clinic
1001 Humboldt Parkway
Buffalo, NY 14208

Buffalo General Hospital
Alcohol Outpatient
1001 Humboldt Parkway
Buffalo, NY 14201

CAO/Dart Drug Abuse Research and Treatment Program
1237 Main Street
Buffalo, NY 14209

Cazenovia Recovery Systems, Inc.
Cazenovia Manor
486 North Legion Drive
Buffalo, NY 14210

New Beginnings Community Residence
376 Dewitt Street
Buffalo, NY 14213

Supportive Living Program
923 Sycamore Street
Buffalo, NY 14212

City of Buffalo DSAS Fillmore/ Leroy Counseling Center
2255 Fillmore Avenue
Buffalo, NY 14214

Ellicott/Masten Counseling Clinic
425 Michigan Avenue
Sheehan Memorial Hospital
Buffalo, NY 14203

Elmwood Counseling Clinic
656 Elmwood Avenue
Suite 201
Buffalo, NY 14222

Genesee/Moselle Clinic
1532 Genesee Street
Buffalo, NY 14211

Lakeshore Behavioral Health
El Comienzo Hispanic Alcoholism Outpatient Clinic
508 Niagara Street
Buffalo, NY 14201

Erie County Medical Center
Alcoholism Acute Care Program
Detoxification Unit
Chemical Dependency Program
462 Grider Street
Buffalo, NY 14215

West Eagle Clinical Services
134 West Eagle Street, Room 500
Buffalo, NY 14202

Erie Niagara Counseling
Associates Alcoholism Outpatient Clinic
6245 Sheridan Drive
Buffalo, NY 14221

Health Care Plan Inc
899 Main Street
Buffalo, NY 14203

Horizon Health Services
Addictions Outpatient/Bailey
3297 Bailey Avenue
Buffalo, NY 14215

Addictions Outpatient/Black Rock
699 Hertel Avenue
Buffalo, NY 14207

Addictions Outpatient/Central Park
60 East Amherst Street
Buffalo, NY 14214

Mid-Erie Mental Health Services, Chemical Dependency Program
1520 Walden Avenue
Buffalo, NY 14225

Alcoholism Outpatient Clinic
1131 Broadway Street
Buffalo, NY 14212

Monsignor Carr Institute Ambulatory Substance Abuse Services
76 West Humboldt Parkway
Buffalo, NY 14214

Northwest Community Mental Health Center
Elmwood Avenue Unit
2495 Elmwood Avenue
Buffalo, NY 14217

Niagara Street Unit
1300 Niagara Street
Buffalo, NY 14213

Research Institute on Addictions Clinical Research Center
1021 Main Street
Buffalo, NY 14203

Sheeham Memorial Hospital
Chemical Dependency Treatment
425 Michigan Avenue
Buffalo, NY 14203

Sisters of Charity Hospital Star Alcoholism Outpatient Clinic
1500 Union Road
Buffalo, NY 14224

Spectrum Human Services
New Alternatives
1235 Main Street
Buffalo, NY 14201

South Buffalo Counseling Center
2040 Seneca Street
Buffalo, NY 14210

Stutzman Alcoholism Treatment
Center Alcoholism Inpatient Rehab Unit
360 Forest Avenue
Buffalo, NY 14213

Veterans' Affairs Medical Center Substance Abuse Program
3495 Bailey Avenue
Unit 116G
Buffalo, NY 14215

CAMILLUS

Professional Counseling Services Alcoholism Outpatient Clinic
5099 West Genesee Street
Camillus, NY 13031

CANAAN

Berkshire Alcoholism Outpatient Clinic
Route 22
Canaan, NY 12029

CANADAIGUA

Kim, Chong, M.D. VAMC Substance Abuse Services
400 Fort Hill Avenue
Canandaigua, NY 14424

Clifton Springs Hospital Alcoholism Outpatient Clinic
11 North Street
Canandaigua, NY 14424

Ontario County Division of Substance Abuse Services
3019 County Complex Drive
Canandaigua, NY 14424

CANTON

North Country Freedom Homes
The Canton House/Halfway House
25 Dies Street
Canton, NY 13617

Saint Lawrence County Alcohol and Substance Abuse Services Alcoholism Outpatient Clinic
University Shopping Plaza
Canton, NY 13617

CARMEL

Arms Acres, Inc.
Alcoholism Inpatient/Outpatient
Carmel, NY 10512

Putnam Family and Community Services
47 Brewster Avenue
Carmel, NY 10512

CARTHAGE

Carthage Clinic of Community Center for Alcoholism
410 State Street
Carthage, NY 13619

CASSADAGA

Tri-County Chemical Dependency
33 North Main Street
Cassadaga, NY 14718

CATSKILL

Twin County Alcohol and Substance Abuse Services, Inc.
66 William Street
Catskill, NY 12414

CENTER MORICHES

Greater Hamptons Interfaith Outpatient Drug Abuse Clinic
529 Main Street
School Special Education Admin. Bldg.
Center Moriches, NY 11934

Transitions Counseling Center, Inc. Alcoholism Outpatient Clinic
408 Main Street
Center Moriches, NY 11934

COBLESKILL

New Directions Schoharie County Substance Abuse Program
150 East Main Street
Cobleskill, NY 12043

Schoharie County Community
Services Program for Alcoholism Recovery
150 East Main Street
Cobleskill, NY 12043

COLD SPRING HARBOR

Huntington Youth Bureau/Drug
and Alcohol Cold Spring Harbor YDA
82 Turkey Lane
Cold Spring Harbor High School
Cold Spring Harbor, NY 11724

COMMACK

Huntington Youth Bureau/Drug
and Alcohol Commack YDA/ Long Acre School
Sarina Drive and Betty Lane
Commack, NY 11725

Catholic Charities of Rockville Centre Outpatient Alcohol Clinic/Commack
155 Indian Head Road
Commack, NY 11725

CORAM

Passages Counseling Center
Alcoholism Outpatient Clinic/
Montauk
3680 Route 112
Coram, NY 11727

YMCA Family Services
6 Middle Country Road
Coram, NY 11727

CORNING

Catholic Charities of the Southern Tier Transitions Counsel for Healthy Living
65 East First Street
Corning, NY 14830

Steuben Council Alcohol and Substance Abuse Services
114 Chestnut Street
Corning, NY 14830

CORTLAND

Alcohol Services Inc. Cortland Alcoholism Outpatient Clinic
17 Main Street
Cortland, NY 13045

Catholic Charities of Cortland
County The Charles Street
Halfway House
29 Charles Street
Cortland, NY 13045

Family Counseling Services of Cortland County
Alcoholism Outpatient Clinic
10 North Main Street
Cortland, NY 13045

CORTLAND MANOR

Hudson Valley Hospital Center Methadone Maintenance Treatment Program
1980 Crompond Road
Cortlandt Manor, NY 10567

DANSVILLE

Livingston County Council/
Alcoholism Alcoholism Outpatient
Clinic
Red Jacket Street
Dansville, NY 14437

DELMAR

Addiction Counseling Center of Bethlehem Crossroads
4 Normanskill Boulevard
Delmar, NY 12054

DOUGLASTON

Jewish Board of Family/ Children Services Pride of Judea Mental Health Center
243-02 Northern Boulevard
Douglaston, NY 11362

DOVER PLAINS

Saint Francis Hospital Eastern Dutchess Counseling Center
Reimer and Mill Streets Chemical
Dependency Clinic
Dover Plains, NY 12522

DUNKIRK

Chautauqua County Dept. Of
Mental Health Alcohol and
Substance Abuse Clinic
319 Central Avenue, 2nd floor
Dunkirk, NY 14048

EAST GREENBUSH

Seton Addiction Services
743 Columbia Turnpike
East Greenbush, NY 12061

EAST HAMPTON

A Program Planned for Life
Enrichment, Inc. (APPLE)/East
95 Industrial Road
East Hampton, NY 11937

Hampton Outpatient
43 Main Street
East Hampton, NY 11937

EAST MEADOW

Family Service Association East Meadow Substance Abuse Outpatient
1975 Hempstead Turnpike, Suite
405
East Meadow, NY 11554

Nassau County Dept of Drug and Alcohol Addiction Drug Counseling Program
2201 Hempstead Turnpike
Nassau County Med Center
Bldg K, 2nd Floor
East Meadow, NY 11554

Nassau County Medical Center
Alcoholism Outpatient Unit
Detox Unit
2201 Hempstead Turnpike
Nassau County Medical Center
Building K
East Meadow, NY 11554

Nassau County Substance Alternative Clinic Methadone Maintenance Treatment Program
2201 Hempstead Turnpike
Nassau CO Medical Center
Building Z
East Meadow, NY 11554

EAST NORTHPORT

Huntington Youth Bureau/Drug and Alcohol Northport/East Northport YDA
7 Diane Court
East Northport, NY 11731

EDEN

Turning Point House Recovery Home
9136 Sandrock Road
Eden, NY 14057

ELIZABETHTOWN

Saint Joseph's Rehabilitation
Center Inc. Alcoholism and
Substance Abuse Outpatient
Cinic
Maple Avenue
Elizabethtown, NY 12932

ELLENVILLE

Ellenville Community Hospital
Acute Care Program
Outpatient Services
Route 209
Ellenville, NY 12428

Renaissance Project, Inc. Ellenville Residential Facility
767 Cape Road
Ellenville, NY 12428

Samaritan Village, Inc.
751 Briggs Highway
Ellenville, NY 12428

Ulster County Mental Health
Services Ellenville Alcohol Abuse Outpatient Clinic
50 Center Street
Trudy Resnick Farber Center
Ellenville, NY 12428

ELMIRA

Economic Opportunity Program, Inc.
Alcoholism and Drug Rehab Clinic
310 West 3 Street
Elmira, NY 14901

Our House Community Residence
401 Division Street
Elmira, NY 14901

Saint Joseph's Hospital
Southern Tier Alcoholism Rehab Services (STARS)
555 East Market Street
Elmira, NY 14902

Schuyler/Chemung/TIOGA
Boces Workplace Intervention/ Alcoholism EAP
495 Philo Road
Elmira, NY 14903

ELMONT

Long Island Counseling Center ACT Chemical Dependency Program
570 Elmont Road, 3rd Floor
Elmont, NY 11003

Long Island Jewish Medical Center
Elmont Treatment Center
40 Elmont Road
Elmont, NY 11003

EVANS MILLS

Credo Foundation, Inc.
24180 County Road 16
Evans Mills, NY 13637

FARMINGVILLE

Suffolk County Division of Alcohol and Substance Abuse Services Farmingville Alcoholism Outpatient Clinic
15 Horse Block Place
Farmingville, NY 11738

FAR ROCKAWAY

South Shore Alcoholism Outpatient Program
718-720 Beach 20th Street
Far Rockaway, NY 11691

Saint John's Episcopal Hospital
South Shore Alcohol Detox Program
327 Beach 19th Street
Far Rockaway, NY 11691

Task Force on Integrated Projects/ Mica
718-720 Beach 20th Street
Far Rockaway, NY 11691

FISHKILL

Mid-Hudson Alcoholism Recovery Center Florence Manor Community Residence
2977 Route 9
Fishkill, NY 12524

FLUSHING

Aurora Concept, Inc.
160-40 78 Road
Flushing, NY 11366

ELMCOR Youth and Adult Activities, Inc.
Day Service Drug Treatment Program
107-20 Northern Boulevard
Flushing, NY 11368

Homeward Bound Program
107-10 Northern Boulevard
Flushing, NY 11368

Elmhurst Halfway House
81-30 Baxter Avenue
Flushing, NY 11373

Elmhurst Hospital Center
Methadone Maintenance Treatment Program
79-01 Broadway
Flushing, NY 11373

Human Service Centers, Inc. Alcoholism Outpatient Clinic
87-08 Justice Avenue, Suite 1-G
Flushing, NY 11373

Jewish Board of Family/Child Services Living Free Drug Program
97-45 Queens Boulevard
Flushing, NY 11374

Mental Health Providers of
Western Queens, Inc. Alcoholism Services
62-07 Woodside Avenue
Flushing, NY 11377

New York Hospital/Medical Center Queens Alcoholism Outpatient Clinic
174-11 Horace Harding Expressway
Flushing, NY 11365

Saint Barnabas Hospital Correctional Health Affiliate Keep
18-18 Hazen Street
Flushing, NY 11370

FRANKLIN SQUARE

Community Counseling Services of West Nassau
Alcoholism Outpatient Clinic
Outpatient Drug Free
1200-A Hempstead Turnpike
Franklin Square, NY 11010

FREEPORT

Operation Pride Outpatient Drug Free
33 Guy Lombardo Avenue
Freeport, NY 11520

Mercy Medical Center
Mercy Hill HWH/Women
95 Pine Street
Freeport, NY 11520

Women's Day Rehabilitation Services
90 Mill Road
Freeport, NY 11520

South Shore Child Guidance
Center Care Alcoholism Program
87 Church Street
Freeport, NY 11520

FULTON

Alcoholism Services in Oswego County
153 North 2nd Street
Fulton, NY 13069

Farnham, Inc. Drug Abuse Outpatient Clinic
120 Cayuga Street, Suite B
Fulton, NY 13069

GARDEN CITY

Medical Arts Samaritan, Inc. Cornerstone Continuous Care
233 7th Street
Garden City, NY 11530

Mercy Hospital Association
Family Counseling Alcoholism Outpatient Clinic
385 Oak Street
Garden City, NY 11530

Mercy Hospital New Hope Primary Care Program
8 Street Avenue P
Mitchel Field Complex
Garden City, NY 11530

GENEVA

Geneva General Hospital 3 South Detox Unit
196 North Street
Geneva, NY 14456

GLEN COVE

Angelo J. Melillo Center for
Mental Health, Inc. Alcoholism Counseling Services
30A Glen Street
Glen Cove, NY 11542

North Shore University Hospital at Glen Cove
Adolescent Substance Abuse Program
Women's/Children's Program
Substance Abuse Program
Saint Andrew's Lane
Glen Cove, NY 11542

GLENS FALLS

Human Resource Center
46 Elm Street
Glens Falls, NY 12801

GLOVERSVILLE

Fulton County Comm. Services
Board Fulton County Alcoholism Services
34 West Fulton Street
Gloversville, NY 12078

Fulton Friendship House, Inc. Victorian Manor
8-10 First Avenue
Gloversville, NY 12078

Saint Mary's Hospital Alcoholism Services
73 North Main Street
Gloversville, NY 12078

GOSHEN

New York State Office of Children/Family Services Drug Abuse Residential Treatment Program
Goshen Secusre Center
Cross Road
Goshen, NY 10924

Pius XII Chemical Dependency Program Substance Abuse Clinic
224 Main Street
Goshen, NY 10924

GOWANDA

Tri-County Memorial Hospital
Alcoholism Inpatient/Outpatient Program
Chemical Dependency Programs
100 Memorial Drive
Gowanda, NY 14070

GREAT NECK

Great Neck Community
Organization for Parents and Youth (COPAY)/Outpatient DE
21 North Station Plaza
2nd Floor
Great Neck, NY 11021

GREENLAWN

Huntington Youth Bureau/Drug and Alcohol Harbor Fields Elwood YDA
8 Gates Street
Greenlawn, NY 11740

GREENPORT

Eastern Long Island Hospital
Quannacut Alcoholism Inpatient Rehab Program
201 Manor Place
Greenport, NY 11944

GROTON

Ithaca Alpha House Center, Inc. Outpatient Drug Abuse Clinic
101 Cayuga Street
Groton, NY 13073

GUILDERLAND

Saint Peter's Addiction Recovery
Center (SPARC) Inpatient Rehabilitation Program
2232 Western Avenue
Guilderland, NY 12084

HAMDEN

Delaware County Alcohol and Drug Abuse Services
Route 10
Hamden, NY 13782

Delaware County Comm. Services
Board De County Alcohol Drug Abuse Services/Hamden
Road 1
Hamden, NY 13782

HAMPTON BAYS

Greater Hamptons Interfaith Council Outpatient Drug Abuse Clinic
154-5 West Montauk Highway
Hampton Bays, NY 11946

Long Island Center for Recovery, Inc.
Alcohol Primary Care
Alcoholism and Drug Abuse Inpatient Rehabilitation
320 Montauk Highway
Hampton Bays, NY 11946

HARRIS

Comm. General Hospital of
Sullivan County Biochemical Dependency Unit
Bushville Road
Harris, NY 12742

HARRISON

Saint Vincent's Westchester Alcoholism Treatment and Outpatient Program
275 North Street
Harrison, NY 10528

HAUPPAUGE

A Program Planned for Life Enrichment, Inc. (APPLE)
220 Veterans Highway
Hauppauge, NY 11788

1373-40 Veterans Highway
Hauppauge, NY 11788

North County Complex Methadone Maintenance Treatment Program
Building 151
415 Oser Avenue
Hauppauge, NY 11788-3620

Suffolk County Dept of Alcohol/ Substance Abuse
Methadone Maintenance and Keep Program
1330 Motor Parkway
Hauppauge, NY 11788

Outpatient Drug Abuse Clinic
Veterans Memorial Highway
North County Complex
Bldg 16, 1st Floor
Hauppauge, NY 11788

HAVERSTRAW

Open Arms, Inc. Halfway House
57-59 Sharp Street
Haverstraw, NY 10927

Village of Haverstraw Counseling Center/Reachout
40 New Main Street
Haverstraw, NY 10927

HAWTHORNE

Cortland Treatment Center of Saint Vincent's
4 Skyline Drive
Hawthorne, NY 10532

HEMPSTEAD

Counseling Service of Eastern
District New York, Inc. Drug Abuse Treatment
175 Fulton Avenue
Suite 301C
Hempstead, NY 11501

EAC, Inc. Outpatient Clinic
250 Fulton Avenue 2nd Floor
Hempstead, NY 11550

Family Services Association of Nassau County
Alcohol Treatment Center
Drug Treatment Center
126 North Franklin Street
Hempstead, NY 11550

Hempstead General Hospital Medical Center Acute Care Alcoholism Program
800 Front Street
Hempstead, NY 11550

Hispanic Counseling Center
Outpatient Drug Free Unit
250 Fulton Avenue
Hempstead, NY 11500

Alcoholism Outpatient Clinic
175 Fulton Avenue, Suite 500
Hempstead, NY 11550

HERKIMER

Herkimer County Alcoholism Services
301 North Walsh Street
Herkimer, NY 13350

HICKSVILLE

Central Nassau Guidance and Counseling Services, Inc.
950 South Oyster Bay Road
Hicksville, NY 11801

Family Service Association
Drug Program
Hicksville Alcoholism Outpatient Clinic
385 West John Street
Hicksville, NY 11801

HIGHLAND

Step One
106 Vineyard Avenue
Highland, NY 12528

Ulster County Mental Health Services Highland/New Paltz Alcohol Abuse Outpatient
560 Route 299 East
Highland, NY 12528

HOGANSBURG

Saint Regis Mohawk Tribe Health Services
St. Regis Road
Hogansburg, NY 13655

HOLTSVILLE

Transitions Counseling Center, Inc. Alcoholism Outpatient Clinic
1150 Portion Road
Holtsville, NY 11742

HORNELL

Saint James Mercy Hospital
Mercycare Alcoholism Treatment Center
1 Bethesda Drive
Hornell, NY 14843

HUDSON

Twin County Alcohol and Substance Abuse Services, Inc. Alcoholism Outpatient Clinic
419 Warren Street
Hudson, NY 12534

Catholic Charities of Columbia County/Supervised Outpatient
431 East Allen Street
Hudson, NY 12534

Columbia County Schools Community Services Project
71 North 3 Street
Hudson, NY 12534

HUDSON FALLS

Family Treatment Center for Alcoholism of
Glen Falls Hospital Alcohol Outpatient Clinic
418 Lower Main Street
Hudson Falls, NY 12839

HUNTINGTON

Huntington Youth Bureau/Drug and Alcohol
Counseling Center
423 Park Avenue
Huntington, NY 11743

HUNTINGTON STATION

Daytop Village, Inc.
Suffolk Outreach
2075 New York Avenue
Huntington Station, NY 11746

Huntington Youth Bureau/Drug and Alcohol
Huntington Station YDA
4 Railroad Street
Huntington Station, NY 11746

South Huntington YDA
300 West Hills Road
Huntington Station, NY 11746

Half Hollow Hills YDA
525 Half Hollow Road
Huntington Station, NY 11746

Long Island Center, Inc. Alcoholism Outpatient Clinic
11 Dawson Street
Huntington Station, NY 11746

Saint Christopher Ottilie Morning Star Community
151 Burrs Lane
Huntington Station, NY 11746

Suffolk County Dept. of
Alcoholism and Substance Abuse Services Huntington Station MMTP Clinic
689 East Jericho Turnpike
Huntington Station, NY 11746

HURLEY

Never Alone, Inc.
20 Crofts Road
Hurley, NY 12443

INDIAN LAKE

Hamilton County Community
Services Alcoholism
Counseling and Prevention Services
83 White Birch Lane
Indian Lake, NY 12842

IRVING

Cattaraugus Indian Reservation Health Center Human Services Unit
1510 Route 438
Irving, NY 14081

ISLIP

Town of Islip Dept. of Human Services
Drug Counseling Services
Outpatient Drug Free
401 Main Street
Islip, NY 11751

ITHACA

Alcoholism and Substance Abuse Council of Tompkins County
201 East Green Street
Suite 500
Ithaca, NY 14850

Ithaca Alpha House Center, Inc. Outpatient Center
102 The Commons
Ithaca, NY 14850

JAMAICA

Beth Israel Medical Center MMTP Queens Clinic
82-68 164 Street
3C1 Bldg. T, Dept. Medicine
Jamaica, NY 11432

Counseling Services of EDNY Heights Recovery Center
89-31 161st Street, Suite 708
Jamaica, NY 11432

Creedmoor Addiction Treatment Center Alcoholism Inpatient Rehab Program
80-45 Winchester Boulevard
Building 19(D)
Jamaica, NY 11427

Daytop Village, Inc. Queens
91-01 Merrick Boulevard
Jamaica, NY 11432

Interline Employee Assistance Program, Inc. Alcoholism Outpatient Clinic
89-00 Sutphin Boulevard
Suite 409
Jamaica, NY 11435

J/CAP Day Services
162-04 South Road
Jamaica, NY 11433

Mary Immaculate Hospital MMTP Clinic
147-18 Archer Avenue
Jamaica, NY 11435

New Spirit Outpatient Alcoholism Clinic
162-04 South Road
Jamaica, NY 11433

Phoenix House, Inc. Portal
175-15 Rockaway Avenue
Jamaica, NY 11434

Queens Child Guidance Center, Inc. Jamaica Family Center
89-56 162nd Street, 3rd Floor
Jamaica, NY 11432

Queens Hospital Center
Alcoholism Clinic
Alcoholism Consultation Team
Alcoholism Inpatient Detox Unit
82-68 164 Street
Jamaica, NY 11432

Stop DWI Program
114-2 Guy Brewer Boulevard
Suite 216
Jamaica, NY 11434

Queens Village Commission for Mental Health
J CAP, Inc./Safe Kids
146-15 Rockaway Boulevard
Jamaica, NY 11433

J CAP Residential Unit
177-33 Baisley Boulevard
Jamaica, NY 11434

Samaritan Village, Inc.
MTA Ambulatory/Residential
130-15 89 Road
Jamaica, NY 11419

MTA Residential Drug Free Outpatient
Program
88-83 Van Wyck Expressway
Jamaica, NY 11435

JAMESTOWN

Chautauqua County Dept. of
Mental Health Alcohol and Substance Abuse Clinic
73 Forest Avenue
Jamestown, NY 14901

WCA Hospital
Alcoholism Rehab Program
51 Glasgow Avenue
Jamestown, NY 14701

JOHNSON CITY

Southern Tier Drug Abuse Treatment Center
Outpatient Methadone Treatment Clinic
40 Arch Street
Johnson City, NY 13790

JOHNSTOWN

New York State Office of Children/Family Services Drug Abuse Residential Treatment Program
Tryon Residential Center
881 County Highway 107
Johnstown, NY 12095

KATONAH

Four Winds Hospital, Inc. Choices Alcoholism Outpatient Clinic
800 Cross River Road
Katonah, NY 10536

KENMORE

Northern Erie Clinical Services
2282 Elmwood Avenue
Kenmore, NY 14217

KERHONKSON

Veritas Villa, Inc.
Alcoholism and Drug Abuse Inpatient
Rehabilitation
5 Ridgeview Road
Kerhonkson, NY 12446

KINGSTON

The Bridge Back of FRMH
30 Broadway Street, Suite 205
Kingston, NY 12401

Kingston Hospital Alcohol Acute Care Program
396 Broadway Street
Kingston, NY 12401

Ulster County Mental Health Services
Alcohol Day Rehab/Evening
Intensive Program
Drug Free Clinic
Drug Free Jail Program
Kingston Alcohol Abuse
Outpatient Clinic
Methadone Maintenance and
Rehab Program/Outpatient
239 Golden Hill Drive
Kingston, NY 12401

LAKE GROVE

Lake Grove Treatment Centers of New York Alcoholism Outpatient Clinic
921 Hawkins Avenue
Lake Grove, NY 11755-1306

Chemical Dependency for Youth Clinic
111 Moriches Road
Lake Grove, NY 11755-1306

LATHAM

Clinical Services and Consulting Inc. Latham Alcoholism Clinic
636 New London Road
Latham, NY 12110

LAWRENCE

Committee on Drug Abuse (CODA) Outpatient Drug Free
270 Lawrence Avenue
Lawrence, NY 11559

Peninsula Counseling Center Alcoholism Counseling Service
270 Lawrence Avenue
5 Towns Community Center
Lawrence, NY 11559

LEVITTOWN

Yours Ours Mine Community Center, Inc.
Adolescent and Family Alcohol Program
Outpatient Ambulatory Drug Free Unit
152 Center Lane Village Green
Levittown, NY 11756

LEWISTON

Mount Saint Mary's Hospital Clearview Treatment Services
5300 Military Road
Lewiston, NY 14092

LIBERTY

Inward House Substance Abuse Treatment
Upper Ferndale Road
Liberty, NY 12754

Sullivan County Alcohol and Drug Abuse Services
Outpatient Clinic
710 Infirmary Road 2nd Floor
Liberty, NY 12754

LIVERPOOL

Conifer Park, Inc. Outpatient Alcoholism Clinic
526 Old Liverpool Road, Suite 4
Liverpool, NY 13088

Family Services Associates Alcohol Outpatient Clinic
7445 Morgan Road
Suite 100
Liverpool, NY 13090

LIVONIA

Livingston County Council on Alcohol and Substance Abuse
30 Commerical Street
Livonia, NY 14487

LOCKPORT

Alcoholism Council in Niagara
County Alcoholism Outpatient Clinic
41 Main Street
Lockview Plaza
Lockport, NY 14094

Reflections Recovery Center
521 East Avenue
Suite 4S
Lockport, NY 14094

LONG BEACH

Long Beach Medical Center
FACTS Alcoholism Outpatient Clinic
455 East Bay Drive
Long Beach, NY 11561

Long Beach Reach
Drug Abuse Clinic
26 West Park Avenue
Long Beach, NY 11561

LONG ISLAND CITY

A Way Out, Inc. II Day Service
10–34 44 Drive
Long Island City, NY 11101

Bridge Plaza Treatment and
Rehab Clinic Education and Methadone Treatment Unit
41-15 27 Street
Long Island City, NY 11101

Phoenix House
Vernon Boulevard Unit
34-25 Vernon Boulevard
Long Island City, NY 10023

Marcy II Unit
2900 Northern Boulevard
Long Island City, NY 11101

LOWVILLE

Lewis County Alcoholism and
Substance Abuse Treatment Center Alcoholism Outpatient Clinic
7514 South State Street
Lowville, NY 13367

LYNBROOK

Link Counseling Center, Inc. Outpatient Drug Free
21 Langdon Place
Lynbrook, NY 11563

LYONS

Clifton Springs Outpatient Alcoholism Clinic
122 Broad Street
Lyons, NY 14489

Wayne County Substance Abuse Services
1519 Nye Road
Lyons, NY 14489

MADRID

North Country Freedom Homes John E. Murphy Community Residence
3702 Circle 14
Madrid, NY 13660

MALONE

Citizen Advocates, Inc. North Star Substance Abuse Services
16 4th Street
Malone, NY 12953

Saint Joseph Rehabilitation
Center, Inc. Alcoholism Outpatient Clinic/Malone
214 East Main Street
Malone, NY 12953

MANHASSET

LIJ/HMC Manhasset Clinic
Daycare Unit
Outpatient Drug Free Unit
1355 Northern Boulevard
Manhasset, NY 11030

North Shore University Hospital
Drug Treatment Center
400 Community Drive
Manhasset, NY 11030

MASSAPEQUA

Yes Community Counseling Center Outpatient Medically Supervised
30 Broadway
Massapequa, NY 11758

MASSENA

Canadian/American Youth
Services, Inc. Rose Hill Treatment Center
2 Elizabeth Drive
Massena, NY 13662

MATTITUCK

Eastern Long Island Hospital Quannacut Outpatient Services
7555 Main Road
Mattituck, NY 11952

MELVILLE

Seafield Services, Inc. Alcohol and Drug Abuse Treatment Unit
900 Walt Whitman Road
Suite 102
Melville, NY 11747

MERRICK

Tempo Group Outpatient Clinic
1260 Meadowbrook Road
Merrick, NY 11566

MEXICO

Harbor Lights Chemical Dependency Services Alcoholism Outpatient Clinic
3358 Main Street
Mexico, NY 13114

MIDDLE ISLAND

Family Recovery Center Alcoholism Outpatient Clinic
514 Middle Country Road
Middle Island, NY 11953

MIDDLETOWN

Emergency Housing Group, Inc.
Middletown Alcohol Crisis Center
Middletown Psychiatric Center
Building 8
Middletown, NY 10940

Horton Family Program Outpatient Clinic for Youth
406 East Main Street
Middletown, NY 10940

Pius XII Youth and Family Services
10 Orchard Street
Middletown, NY 10940

Regional Economic Community Action Recap Alcoholism Outpatient Rehab Program
40 Smith Street
Middletown, NY 10940

Restorative Management Corporation Outpatient Drug Clinic
15 King Street
Middletown, NY 10940

Richard C. Ward Addiction Treatment Center
141 Monhagen Avenue
Middletown, NY 10940

MILLBROOK

Saint Francis Hospital Millbrook Counseling Center
Oak Summit Road
Millbrook, NY 12545

MINEOLA

Long Island Jewish Hillside Medical Center
Family Consultation Center Alcoholism Outpatient
366 Jericho Turnpike
Mineola, NY 11501

Nassau Counseling, Inc.
Outpatient Program
450 Jericho Turnpike, Suite 206
Mineola, NY 11501

Seafield Center, Inc. Mineola Alcoholism Outpatient Clinic
110 Main Street
Mineola, NY 11501

MONROE

Pius XII Chemical Dependency Program
Monroe Clinic/Outpatient Drug Free
Monroe Alcoholism Outpatient Clinic
520 Route 17-M
Monroe, NY 10950

MONTICELLO

Sullivan County Council on Alcohol/Drug Abuse, Inc.
17 Hamilton Avenue
Monticello, NY 12701

MORICHES

Passages Counseling Center Alcoholism Outpatient Clinic
Montauk Highway Monarch Center Suite 109
Moriches, NY 11955

MOUNT KISCO

The Weekend Center, Inc. Alcoholism Outpatient Clinic
24 Smith Avenue
Mount Kisco, NY 10549

MOUNT VERNON

Mount Vernon Hospital Methadone Maintenance Treatment Clinic
3 South 6th Avenue
Mount Vernon, NY 10550

Renaissance Project, Inc. Mount Vernon Unit
3 South 6 Street
Mount Vernon, NY 10550

Westchester Community
Opportunity Program Mount Vernon Open Door Program
34 South 6 Avenue
Mount Vernon, NY 10550

Yonkers General Hospital Archway Alcoholism Outpatient Clinic
100 East First Street, 6th Floor
Mount Vernon, NY 10550

NEWARK

Finger Lakes Alcoholism
Counseling Referral Agency Alcoholism Outpatient Clinic
301 West Union Street
Newark, NY 14513

NEWBURGH

Pius XII Chemical Dependency
Program Newburgh Clinic/ Outpatient Drug Free
62 Grand Street
Newburgh, NY 12550

Saint Luke's Hospital of Newburgh
479 Broadway
Newburgh, NY 12550

Alcohol Outpatient
Methadone Maintenance Treatment Program
3 Commercial Place
Newburgh, NY 12550

NEW HARTFORD

Center for Addiction Recovery,
Inc. Alcoholism Outpatient Clinic
4299 Middle Settlement Road
New Hartford, NY 13413

NEW HYDE PARK

Long Island Jewish Medical Center
Daehrs Outpatient Drug Free
270-05 76 Avenue
Building 5
New Hyde Park, NY 11042

NEW ROCHELLE

Guidance Center, Inc.
Chemical Dependency Treatment Center
403-5 North Avenue
New Rochelle, NY 10801

Renaissance Project, Inc.
New Rochelle Unit
Re-Entry Unit/Storefront
350 North Avenue
New Rochelle, NY 10801

United Hospital Alcoholism Outpatient Clinic
3 The Boulevard
New Rochelle, NY 10801

Volunteers of America
395 Webster Avenue
New Rochelle, NY 10801

Westchester Community
Opportunity Program New Rochelle Outreach Center
33 Lincoln Avenue
Suite 2
New Rochelle, NY 10801

NEW YORK

Adolescent Health Center of The Mount Sinai Medical Center
312 East 94th Street
New York, NY 10128

Alcoholism Outpatient Clinic
19 Union Square West, 7th Floor
New York, NY 10003

Alianza Dominicana, Inc.
2410 Amsterdam Avenue
New York, NY 10033

American Indian Community Substance Abuse Services
708 Broadway
New York, NY 10003

Areba Casriel, Inc.
Alcoholism Inpatient Rehab Program
Inpatient Drug Detox Program
Inpatient Primary Alcohol Program
500 West 57th Street, 2nd Floor
New York, NY 10019

Drug Outpatient Program
Substance Abuse Outpatient Clinic
145 West 45th Street
New York, NY 10019

ARMS Acres, Inc. Alcoholism
Outpatient Services of Manhattan
1841 Broadway
3rd floor
New York, NY 10023

ARTC

Manhattan Clinic 21
Starting Point
136 West 125th Street
6th Floor
New York, NY 10027

Manhattan Clinic 22
Kaleidoscope
136 West 125th Street
New York, NY 10027

Manhattan Clinic 23
Third Horizon
2195 3rd Avenue
New York, NY 10035

Beth Israel Medical Center

Alcoholism Acute Care Program
10 Nathan D Perlman Place
Bernstein Pavilion
New York, NY 10003

Drug Detoxification Program
1-9 Nathan D Perlman Place
New York, NY 10003

Stuyvesant Square Chemical Dependency Program
380 2nd Avenue, 10th Floor
New York, NY 10010

Stuyvesant Square Chemical Dependency Program
1st Avenue at 16th Street
New York, NY 10003

Beth Israel Medical Center Methadone Maintenance Treatment Program
Avenue A Clinic
26 Avenue A
New York, NY 10009

Clinics 1/2/3/6/7
Interium Clinic
103 East 125th Street
New York, NY 10035

Clinics IE/2F/3G
429 2nd Avenue
New York, NY 10003

Clinic 2C
435 2nd Avenue
New York, NY 10010

Clinic 3C
435 2nd Avenue
New York, NY 10010

Clinic 4, Units 1/2
21 Old Broadway
Basement
New York, NY 10027

Clinics 8/8D
140 West 125th Street
New York, NY 10027

Gouverneur Clinic
109 Delancy Street
New York, NY 10002

Coney Island Clinic
215 Park Avenue South, 15th Floor
New York, NY 10003

Saint Vincent's Clinic
201 West 13th Street
New York, 10011

Bliss Poston the Second Wind, Inc.
152 Madison Avenue
Suite 505
New York, NY 10016

Bowery Residents Committee, Inc. Alcoholism Outpatient Clinic
191 Chrystie Street
New York, NY 10002

Boys Harbor, Inc. Alcohol Outpatient Unit
1 East 104th Street
New York, NY 10029

BRC Human Services Corporation Alcohol Crisis Center
324 Lafayette Street
New York, NY 10012

Cabrini Medical Center Start
137 2nd Avenue
New York, NY 10003

Carnegie Hill Institute Methadone Treatment Center
116 East 92nd Street
New York, NY 10028

Center for Comp. Health Practice, Inc.
1900 2nd Avenue
12th Floor
New York, NY 10029

163 East 97th Street
New York, NY 10029

Central Harlem Emergency Care Services Alcohol Crisis Center
419 West 126th Street
New York, NY 10027

Chinatown Alcoholism Services
253 South Street 2nd Floor
New York, NY 10002-7827

CIS Counseling Center, Inc. CIS Addiction Services
150 Nassau Street, Room 1100
New York, NY 10038

Cornell University Medical
College Midtown Center for Treatment and Research
55 West 44th Street
New York, NY 10036

Create, Inc.
121 West 111th Street
New York, NY 10026

Daytop Village, Inc.
Federal Parole
132 West 83rd Street
New York, NY 10024

Education Alliance
25 Avenue D
New York, NY 10009

Project Contact
Outpatient Program
315 East 10th Street
New York, NY 10009

Enter, Inc.
Alcoholism Community Residence
2009 3rd Avenue
New York, NY 10029

Alcoholism Outpatient Clinic
302–306 East 111 Street
2nd Floor
New York, NY 10029

Exponents Treatment Exchange
151 West 26th Street, 3rd Floor
New York, NY 10001

First Step to Recovery
330 West 58th Street, Suite 609
New York, NY 10019

Freedom Institute, Inc. Alcoholism Outpatient Clinic
515 Madison Avenue
35th Floor
New York, NY 10022

Gracie Square Hospital
416 East 76th Street
New York, NY 10021

Gramercy Park Medical Group
253-55 3rd Avenue
New York, NY 10010

Greenwich House Counseling Center
80 5th Avenue
10th Floor
New York, NY 10011

Greenwich House, Inc.
Alcohol and Drug Treatment Program
55 5th Avenue
New York, NY 10003

Alcohol Treatment Program
312 Bowery
New York, NY 10012

Greenwich House MMTP
Cooper Square
50 Cooper Square
New York, NY 10003

Greenwich House West MMTP
24 West 20th Street
New York, NY 10011

Harlem Hospital
Alcohol Detoxification Unit
Harlem Hospital Center
K Building Mezzanine
136 Street and 5th Avenue
New York, NY 10037

Alcoholism Treatment Center
22-44 West 137th Street 4th Floor
New York, NY 10037

Methadone Treatment Clinic
15 West 136th Street K Building
New York, NY 10037

Methadone Maintenance Treatment Program
264 West 118th Street
New York, NY 10026-1620

Harold L. Trigg Clinic
543 Cathedral Parkway
New York, NY 10025

Hazelden New York
Inpatient Drug Abuse Rehab Program
Outpatient Drug Abuse Clinic
233 East 17th Street
New York, NY 10003

HHC Bellevue Hospital
Methadone Maintenance Treatment Program
27th Street and 1st Avenue
Buildings C and D
New York, NY 10016

Alcoholism Outpatient Clinic
462 1st Avenue at 27th Street
New York, NY 10016

HHC/Metropolitan Hospital Center
Drug Detoxification Program
Methadone Treatment Program
1900 2nd Avenue, 2nd Floor
New York, NY 10029

Immigrant Social Services, Inc.
137 Henry Street
New York, NY 10002

Inter Care, Ltd.
51 East 25th Street
Suite 400
New York, NY 10010

International Center for the Disabled (TCD) Chemical Dependency Services/ Outpatient Clinic
340 East 24 Street
New York, NY 10010

Inwood Community Services, Inc.
Comprehensive Outpatient Alcoholism Program
Get Centered/Outpatient
651 Academy Street, 2nd Floor
New York, NY 10034

Koeppel, Richard, M.D. Methadone Maintenance Treatment
311 West 35th Street
New York, NY 10001

Lesbian and Gay Community Services Center
Project Connect
208 West 13th Street
New York, NY 10011

Lower Eastside Service Center
Drug Abuse Prevention Services
127 West 22nd Street
New York, NY 10011

Methadone Maintenance Treatment Program Unit 1
Outpatient Day Clinic
46 East Broadway
New York, NY 10002

Methadone Maintenance Treatment Program Unit 2
7 Governeur Slip East
New York, NY 10002

Methadone Maintenance Treatment Program Unit 3
62 East Broadway
New York, NY 10002

Su Casa Methadone Maintenance Treatment Program
157 Chambers Street 8th Floor
New York, NY 10007

Manhattan Addiction Treatment Center
600 East 125th Street
Wards Island
New York, NY 10035

Medical Arts Center Hospital
57 West 57th Street
New York, NY 10019

Medically Supervised Ambulatory Substance Abuse Clinic
19 Union Square West, 7th Floor
New York, NY 10003

Metropolitan Hospital Center
Drug Addiction Clinic
Methadone Maintenance Treatment Program
1900 2nd Avenue
Psychiatric Pavilion
New York, NY 10029

Mount Sinai Hospital
Narcotics Rehab Center
17 East 102nd Street
New York, NY 10029

Narco Freedom Program
337 West 51st Street
New York, NY 10019

458 West 50th Street
New York, NY 10019-6501

National Recovery Institute
458 West 50th Street
New York, NY 10019

New York City Department of Probation Tri-Center Unit
Alcoholism Outpatient Clinic
Drug Abuse Treatment
575 8th Avenue, 7th Floor
New York, NY 10018

New York Foundling
3280 Broadway Street
New York, NY 10027

New York Hospital Methadone Maintenance Treatment Clinic
401 East 71st Street
New York, NY 10021

New York Presbyterian Hospital Adolescent Development Program
411 East 69th Street
New York, NY 10021

New York Society for The Deaf Substance Abuse Clinic
817 Broadway Street, 7th Floor
New York, NY 10003

New York State Association for Retarded Children Sobriety Services Clinic
200 Park Avenue South, 3rd Floor
New York, NY 10003

New York University Downtown Hospital Methadone Maintenance Treatment Program
74 Trinity Place
New York, NY 10006

North General Hospital
Alcoholism Detoxification Unit
1879 Madison Avenue
New York, NY 10035

Alcoholism
Treatment Center
1824 Madison Avenue
New York, NY 10035

NRL Resources, Inc.
450 South Park Avenue
Suite 402
New York, NY 10016

Odyssey House, Inc. of New York
Odyssey House Adult Program
Wards Island
Mabon Building 13
New York, NY 10035

Phase Piggy Back, Inc.
Adult Resocialization Unit
507 West 145th Street
New York, NY 10031

Project Okhute
1780-1784 Amsterdam Avenue
New York, NY 10031

Striver House
202-204 Edgecomb Avenue
New York, NY 10030

Parallax Center, Inc.
145 East 32nd Street, 6th Floor
New York, NY 10016

Phoenix House
164 West 74th Street
New York, NY 10023

Pride Site I Male Only Site
371 East 10th Street
New York, NY 10009

Project Green Hope Services for Women
Drug Abuse Services
448 East 119th Street
New York, NY 10035

Project Renewal Alcohol Crisis Center
8 East 3rd Street, 4th Floor
New York, NY 10003

Project Return Foundation, Inc.
814-816 Amsterdam Avenue
New York, NY 10025

Chelsea Tribeca Institute
Continuing Care Treatment
Program
740 Broadway, 6th Floor
New York, NY 10003

Dreitzer Residence for Women and Children
315-317 East 115th Street
New York, NY 10029

Project Return Parole
814 Amsterdam Avenue
New York, NY 10025

Transitional Treatment Program
2112 2nd Avenue
New York, NY 10029

Reality House, Inc
Drug Free Outpatient
MTA Day Service
637 West 125th Street
New York, NY 10027

Saint Clare's Hospital Health Center Methadone Treatment Services
426 West 52nd Street
New York, NY 10019

Saint Luke's/Roosevelt Hospital Center
Alcoholism Halfway House
306 West 102nd Street
New York, NY 10025

Alcoholism Inpatient Detox Unit
Amsterdam Avenue at 114th
Street
New York, NY 10025

Alcoholism Outpatient Clinic
411 West 114th Street
New York, NY 10025

Smithers Alcoholism Outpatient Clinic
Substance Abuse Program/
Narcotics
1000 10th Avenue
New York, NY 10019

Smithers Inpatient Rehabilitation Unit
56 East 93rd Street
New York, NY 10028

Saint Marks Place Institute Outpatient Alcoholism Program
57 Saint Marks Place
New York, NY 10003

Samaritan Village, Inc. Residential Drug Free
225-27 East 53rd Street
New York, NY 10022

Drug Residential Treatment
Program
327 West 43rd Street
New York, NY 10036

Settlement Health Association, Inc. Rush Program
1775 3rd Avenue
New York, NY 10029

Silbermann, Eugene. M.D., Outpatient Methadone Clinic
2369 2nd Avenue
New York, NY 10035

22 East 110th Street
New York, NY 10029

Upper Manhattan Mental Health Center Alcoholism Program
1727 Amsterdam Avenue
New York, NY 10031

Veritas Therapeutic Community, Inc.
Residential Drug Treatment Program
912 Amsterdam Avenue
New York, NY 10025

Infants and Toddlers Program
119 West 106th Street
New York, NY 10025

Veterans' Affairs Medical Center
Alcohol/Drug Dependence Treatment Program
523 East 22nd Street
New York, NY 10010

Villa OPC II, Inc.
Alcoholism Outpatient Clinic
290 Madison Avenue 6th Floor
New York, NY 10017

Women in Need Alcoholism and Drug Abuse Services
115 West 31st Street
New York, NY 10001

NIAGARA FALLS

Alcohol Council in Niagara County
First Step Chemical Crisis Center
1560 Buffalo Avenue
Niagara Falls, NY 14303

Fellowship House, Inc.
Alcoholism Halfway House
431 Memorial Parkway
Niagara Falls, NY 14303

Fellowship House Supportive Living
625 Buffalo Avenue
Niagara Falls, NY 14303

Horizon Health Services
Addictions Outpatient Drug Clinic
Alcoholism Outpatient Clinic
6560 Niagara Falls Boulevard, 2nd Floor
Niagara Falls, NY 14304

Milestones Alcoholism Services Alcoholism Outpatient Clinic
501 10 Street
Niagara Falls, NY 14301

Niagara County Mental Health Department Alcoholism Outpatient Clinic
1001 11th Street
Niagara Falls, NY 14301

Niagara Drug Abuse Program
Methadone Maintenance Treatment Program
Outpatient Services
1001 11th Street
Trott Access Center
Niagara Falls, NY 14301

NORTH BABYLON

Suffolk County Dept of Health Services Division of Alcohol and Substance Abuse Services
1121 Deer Park Avenue
North Babylon, NY 11703

NORTH MERRICK

Tempo Group, Inc. Drug Abuse Outpatient Clinic
1260 Meadowbrook Road
North Merrick, NY 11566

NORTHPORT

Concepts For Narcotics
Prevention, Inc. The Place/ Outpatient Drug Free
324 Main Street
Northport, NY 11768

NORTH TONAWANDA

Bry-Lin Hospitals Alcoholism Services
3571 Niagara Falls Boulevard
North Tonawanda, NY 14120

Mount Saint Mary's Hospital Clearview Alcoholism Outpatient Services
66 Mead Street
North Tonawanda, NY 14120

NORWICH

Chenango County Alcohol and Drug Services
Alcoholism Outpatient
105 Leilanis Way
Norwich, NY 13815

NYACK

Nyack Hospital
Alcoholism Acute Care Program
Alcoholism Inpatient Rehab Program
160 North Midland Avenue
Nyack, NY 10960

Nyack/Orangeburg Outreach to Youth
42 Burd Street
Nyack, NY 10960

OAKDALE

Sanctuary East, Ltd. Outpatient Drug Abuse Clinic
One Berard Boulevard
Oakdale, NY 11769

OCEANSIDE

Oceanside Counseling Center, Inc.
Alcoholism Services
Medically Supervised Outpatient Drug Free
71 Homecrest Court
Oceanside, NY 11572

OGDENSBURG

Saint Lawrence Addiction Treatment Center
1 Chimney Point Drive
Hamilton Hall
Ogdensburg, NY 13669

Saint Lawrence County Comm.
Services Board Alcoholism Outpatient Clinic/Ogdensburg
1 Chimney Point Drive
Pritchard Pavilion
Ogdensburg, NY 13669

OLEAN

Cattaraugus County Council on
Alcoholism and Substance Abuse, Inc.
201 South Union Street
Olean, NY 14760

ONEIDA

Mancusco Counseling Services
123 Phelps Street
Oneida, NY 13421

Maxwell House
Next Step Apartments
312 Main Street
Oneida, NY 13421

ONEONTA

Otsego County Community
Services Otsego Chemical Dependencies Clinic
31 Main Street
Oneonta, NY 13820

ORANGEBURG

Blaisdell Alcoholism Treatment Center Inpatient Rehabilitation Unit
Rockland Psychiatric Center Campus
Building 28
Orangeburg, NY 10962

ORCHARD PARK

Spectrum Human Services
Southtowns Counseling
227 Thorn Avenue
Orchard Park, NY 14127

OSSINING

Phelps Alcohol Treatment Services
22 Rockledge Avenue
Ossining, NY 10562

OSWEGO

Farnham, Inc. Outpatient Services
33 East First Street
Oswego, NY 13126

Oswego County Council on Alcoholism
Alcoholism Outpatient Clinic
53 East 3rd Street
Oswego, NY 13126

Tioga County Alcohol and Drug Services
175 Front Street
Owego, NY 13827

Substance Abuse Outpatient Clinic
1277 Taylor Road
Wash Glad Building
Owego, NY 13827

OVID

Dick Van Dyke Addiction Treatment Center Alcoholism Inpatient Rehab Unit
1330 County Road, Suite 132
Ovid, NY 14521-9716

OYSTER BAY

Youth and Family Counseling
Agency of Oyster Bay/East Norwich, Inc.
193A South Street
Oyster Bay, NY 11771

PATCHOGUE

Crossings of Long Island, Inc. Alcohol and Drug Treatment Programs
450 Waverly Avenue, Suite 5
Patchogue, NY 11772

Brookhaven Health Center
365 East Main Street
Patchogue, NY 11772

PEARL RIVER

Nyack Hospital Alcoholism Outpatient Clinic
2 Blue Hill Plaza
Pearl River, NY 10965

PEEKSKILL

Peekskill Area Health Center, Inc.
Alcoholism Outpatient Clinic
Peekskill Pathways
1037 Main Street
Peekskill, NY 10566

PLAINVIEW

Nassau County Dept. of Drugs and Alcohol Addiction
1425 Old Country Road
Plainview, NY 11803

Plainview/Old Bethpage CSD
Youth Activities Council/ Reflection/Prevention
777 Old Country Road
Plainview, NY 11803

PLATTSBURGH

Champlain Valley Family Center Drug Treatment Youth Services Inc.
20 Ampersand Drive
Plattsburgh, NY 12901

Clinton County Alcoholism Program
16 Ampersand Drive
Plattsburgh, NY 12901

Clinton County Mental Health Association
Twin Oaks Alcoholism Halfway House
79 Oak Street
Plattsburgh, NY 12901

Conifer Park, Inc. Alcoholism Outpatient Clinic
13 Latour Avenue
Plattsburgh, NY 12901

POMONA

Rockland County Dept of Mental Health Alcoholism Detoxification Unit
Dr Robert L Yeager Health Center
Building C
Pomona, NY 10970

PORT CHESTER

Renaissance Project, Inc. Port Chester Center
4 Poningo Street
Port Chester, NY 10573

United Hospital Substance Abuse Detoxification Unit
406 Boston Post Road
Port Chester, NY 10573

WCMHB Saint Vincent's Hospital
Methadone Maintenance Treatment Program/Outpatient
350 North Main Street
Port Chester, NY 10573

PORT JEFFERSON

Saint Charles Hospital and Rehab Center Alcoholism Inpatient Rehab Program
200 Belle Terre Road
Port Jefferson, NY 11777

PORT JEFFERSON STATION

Crossings Recover Center
5225 Route 347
Davis Professional Park, Suite 40
Port Jefferson Station, NY 11776

John T. Mather Memorial Hospital Mather Outpatient Alcoholism Clinic
208 Route 112
Port Jefferson Station, NY 11776

PORT JERVIS

Crossroads At Mercy Community
Hospital Crossroads Acute Care Alcoholism Program
160 East Main Street
Port Jervis, NY 12771

Support Center, Inc. Substance Abuse Treatment Program
181 Route 209
Port Jervis, NY 12771

PORT WASHINGTON

Port Counseling Center, Inc.
225 Main Street
Port Washington, NY 11050

POTSDAM

Canton/Potsdam Hospital
Alcoholism Detoxification Unit
50 Leroy Street
Potsdam, NY 13676

Saint Lawrence County Alcohol and Substance Abuse Services Outpatient Clinic
State University of New York at Potsdam
Van Housen Hall
Potsdam, NY 13676

POUGHKEEPSIE

Astor School Based Clinic Alcoholism Youth Clinic
350 Dutchess Turnpike
Poughkeepsie, NY 12603

Dutchess County Dept. of Mental Hygiene
Alcohol Abuse Clinic
20 Manchester Road
Poughkeepsie, NY 12603

Dutchess County Methadone Clinic Outpatient
230 North Road
Poughkeepsie, NY 12601

Dutchess County Substance Abuse Clinic
20 Manchester Road
Poughkeepsie, NY 12603

Josephs House Alcoholism Supportive Living Facility
4 Fallkill Place
Poughkeepsie, NY 12601

Mid-Hudson Alcoholism Recovery Center
Alcoholism Primary Care Program
Branch B. Ryon Hall
Poughkeepsie, NY 12601

Bolger House Community Residence
260 Church Street
Poughkeepsie, NY 12601

New Hope Manor, Inc. Re-Entry House
141 South Avenue
Poughkeepsie, NY 12601

Waryas House Rehab Programs, Inc.
101 Inwood Avenue
Poughkeepsie, NY 12603

QUEENSBURY

Baywood Center
551 Bay Road
Queensbury, NY 12804

REGO PARK

Long Island Consultation Center, Inc. Alcoholism Outpatient Clinic
97-29 64th Road
Rego Park, NY 11374

Psychiatric and Addictions Recovery Services (PARS)
92-29 Queens Boulevard
Suite 2-E
Rego Park, NY 11374

RHINEBECK

Cornerstone of Rhinebeck
500 Milan Hollow Road
Rhinebeck, NY 12572

Daytop Village, Inc.
Fox Hollow Road
Rhinebeck, NY 12572

Saint Francis Hospital Rhinebeck Counseling Center
14 Springbrook Avenue
Rhinebeck, NY 12572

RICHMOND HILL

New York City Department of Probation Outreach Family Services
117-11 Myrtle Avenue
Richmond Hill, NY 11418-1751

RICHVILLE

Canton Potsdam Hospital Alcoholism Outpatient Clinic
The Richville Clinic
Richville, NY 13681

RIDGEWOOD

Outreach House I
16-14 Weirfield Street
Ridgewood, NY 11385

RIVERHEAD

Seafield Services
Alcoholism Outpatient Program
Drug Abuse Treatment Unit
212 West Main Street
Riverhead, NY 11091

Alternatives East Counseling Center
540 East Main Street
Riverhead, NY 11901

Suffolk County Dept of Alcohol and Substance Abuse Services
300 Center Drive County Center
Riverhead, NY 11901

ROCHESTER

Anthony L. Jordan Health Center Alcoholism Outpatient Clinic
30 Hart Street
Rochester, NY 14603

Bry-Lin Hospitals
Outpatient Alcoholism Program
2741 Ridge Road West
Rochester, NY 14626

Catholic Charities/Rochester
Catholic Family Center
Outpatient/Intensive Outpatient
Restart Alcoholism Outpatient Clinic
55 Troup Street
Plymouth Park West
Rochester, NY 14608

CFC/Restart Substance Abuse Services
Liberty Manor
1111 Joseph Avenue
Rochester, NY 14621

81 Barberry Terrace
Rochester, NY 14621

Community Alcoholism Services Clinic
150 North Clinton Avenue
Rochester, NY 14604

Conifer Counseling Services
1150 University Avenue
Rochester, NY 14607

Crossroads Apartment Program
758 South Avenue
Rochester, NY 14620

East House Corporation
269 Alexander Street
Rochester, NY 14607

239 Alphonse Street
Rochester, NY 14621

50 Browncroft boulevard
Rochester, NY 14609

Crossroads III/Cody House
407 Frederick Douglas Street
Rochester, NY 14608

Family Services of Rochester, Inc.
Administrative Unit
Alcoholism Outpatient Clinic
Avon Drug Abuse Prevention Unit
30 North Clinton Avenue
Rochester, NY 14604

Genesee Hospital Dept. of
Psychiatry Genesee Alcohol Treatment Center
580 South Avenue
Rochester, NY 14607

Huther/Doyle Memorial Institute
Alcoholism Outpatient Clinic
Drug Abuse Treatment Unit
360 East Avenue
Rochester, NY 14604

John L. Norris Alcoholism Treatment Center
Alcoholism Inpatient Rehab Unit
1732 South Avenue
Rochester Psychiatric Center, Howard I
Rochester, NY 14620

Main Quest Treatment Center
184 Alexander Street
Rochester, NY 14607

287 Wellington Avenue
Rochester, NY 14611

Alcoholism Inpatient Rehab Unit
Comprehensive OP Alcoholism Clinic
Supportive Living
774 West Main Street
Rochester, NY 14611

Burlington Community Residence
380 Barrington Street
Rochester, NY 14620

West Avenue Community Residence
383 West Avenue
Rochester, NY 14611

Park Ridge Chemical Dependency, Inc.
Brighton Alcoholism Outpatient Clinic
Drug Abuse Treatment
2000 South Winton Road
Building 2
Rochester, NY 14618

Adolescent Community Residence
2654 Ridgeway Avenue
Rochester, NY 14626

Unity Health System
1565 Long Pond Road
Rochester, NY 14626

Women's Community Residence
2650 Ridgeway Avenue
Rochester, NY 14626

Park Ridge Hospital
Chemical Dependency Unit
Short Term Rehab Unit
1565 Long Pond Road
Rochester, NY 14626

Outpatient Substance Abuse Clinic
81 Lake Avenue
Rochester, NY 14608

Pathway Houses of Rochester
Alcoholism Supportive Living Facility
353 University Avenue
Rochester, NY 14607

Sisters of Charity Hospital
435 East Henrietta Road
Rochester, NY 14620

Supportive Living Facility
440 Fredrick Douglas Street
Rochester, NY 14608

Rochester Mental Health Center
Alcoholism Outpatient Clinic
Drug Treatment Services/ MSASATP
490 East Ridge Road
Rochester, NY 14621

Saint Joseph's Villa of Rochester, Inc.
Life Program/Residential Chemical Dependency Services/Youth/ Long Term
3300 Dewey Avenue
Rochester, NY 14616

University of Rochester/Strong Memorial Hospital
Methadone Maintenance Treatment Clinic
Drug Dependency Outpatient
300 Crittenden Boulevard
Rochester, NY 14642

Veterans' Affairs Medical Center
465 Westfall Road, Suite 116-6
Rochester, NY 14614

Volunteers of America of Western New York Alcoholism Halfway House
175 Ward Street
Rochester, NY 14606

Westfall Associates, Inc.
919 Westfall Road
Suite C-120
Rochester, NY 14618

YWCA of Rochester/Monroe County
Alcohol Clinic
Steppingstone Drug Program
Supported Living Program
175 North Clinton Avenue
Rochester, NY 14604

ROCKVILLE CENTRE

Mercy Medical Center
Hospital Intervention Services
1000 North Village Avenue
Rockville Centre, NY 11570

Rockville Center Narcotics/Drug Abuse Confide/Outpatient Drug Free
30 Hempstead Avenue
Suite H-6
Rockville Centre, NY 11570

ROME

Rome Memorial Hospital
Community Recovery Center Alcoholism Outpatient
264 West Dominick Street
Rome, NY 13440

RONKONKOMA

A Program Planned for Life Enrichment, Inc. (APPLE)
161 Lake Shore Road
Ronkonkoma, NY 11779

153 Lake Shore Drive
Ronkonkoma, NY 11779

Community Counseling Services of Ronkonkoma Alcoholism Outpatient Clinic
3275 Veterans Memorial Highway
Suite B-1
Ronkonkoma, NY 11779

Passages Counseling Center
650 Hawkins Avenue
Ronkonkoma, NY 11779

Professional Addiction Counseling and Education
3555 Veterans Highway
Suite E
Ronkonkoma, NY 11779

ROOSEVELT

Nassau County Department of Drug and Alcohol Addiction
42 East Fulton Avenue
Roosevelt, NY 11575

Roosevelt Education Alcoholism Counseling Treatment Center
React Alcoholism Outpatient Clinic
27A Washington Place
Roosevelt, NY 11575

SAINT ALBANS

Queens Village Commission for Mental Health
JCAP Inc.,
177-33 Baisley Boulevard
Saint Albans, NY 11434

SALAMANCA

Lionel R John Health Center Human Services Unit
987 R C Hoag Drive
Salamanca, NY 14779

SANBORN

Horizon Village Drug Free Residential Treatment
6301 Inducon Drive East
Sanborn, NY 14132

SARANAC LAKE

Saint Joseph's Rehabilitation Center, Inc.
Alcoholism Inpatient Rehabilitation Program
Glenwood Estates
Saranac Lake, NY 12983

Alcoholism and Drug Outpatient Clinic
50 Woodruff Street
Saranac Lake, NY 12983

SARATOGA SPRINGS

Saratoga County Alcoholism Services Alcoholic Outpatient Clinic
254 Church Street
Saratoga Springs, NY 12866

Saratoga Springs Office of Abused Substances and Intervention Services, Inc.
517 Broadway
Saratoga Springs, NY 12866

SCHENECTADY

Alcoholism and Substance Abuse Council of Schenectady County, Inc.
834 Emmett Street
Schenectady, NY 12307

575 Lansing Street
Schenectady, NY 12303

406-408 Summit Avenue
Schenectady, NY 12307

302 State Street
Schenectady, NY 12305

Bridge Center of Schenectady, Inc.
Residential Drug Treatment
70–72 Union Street
Schenectady, NY 12308

Carver Community Counseling Services Medically Supervised Outpatient
949 State Street
Schenectady, NY 12307

Lifestart Ambulatory Substance Abuse Program
1356 Union Street
Schenectady, NY 12308

Seton Addiction Services
1594 State Street
Schenectady, NY 12304

SCOTIA

Conifer Park, Inc.
Alcoholism and Drug Abuse Services
79 Glenridge Road
Scotia, NY 12302

SEAFORD

Seaford Union Free School District Drug Abuse Program
1575 Seamans Neck Road
Seaford, NY 11783

SHRUB OAK

Phoenix Academy
Stoney Street
Shrub Oak, NY 10588

SMITHTOWN

Employee Assistance Resource Services, Inc. (EARS)
278 East Main Street
Smithtown, NY 11787

Saint John's Episcopal Hospital
Smithtown Alcohol Detoxification Unit
498 Smithtown Bypass
Smithtown, NY 11787

Town of Smithtown/Horizons Counseling and Education Center
124 West Main Street
Smithtown, NY 11787

SOUTHAMPTON

Alternatives Counseling Center
291 Hampton Road
Southampton, NY 11968

SOUTH KORTRIGHT

Phoenix House
County Road 513
Old Route 10 Belle Terre
South Kortright, NY 13842

SOUTH OZONE PARK

Faith Mission Alcohol Crisis Center, Inc.
114-40 Van Wyck Expressway
South Ozone Park, NY 11420

SPRING VALLEY

Rockland County Dept of Mental Health Alcoholism Outpatient Clinic
50A South Main Street
Spring Valley, NY 10977

Town of Ramapo Youth
Counseling Services Outpatient Drug Free
296 North Main Street
Spring Valley, NY 10977

STATEN ISLAND

Amethyst House, Inc. Alcoholism Halfway House
75 Vanderbilt Avenue
Staten Island, NY 10304

Bayley Seton Hospital, Inc.
Alcoholism Acute Care Unit
75 Vanderbilt Avenue
Staten Island, NY 10304

Bridge Back to Life Center, Inc. Staten Island Drug Abuse Treatment
1688 Victory Boulevard
Staten Island, NY 10314

Camelot of Staten Island, Inc.
Adolescent Drug Abuse Prog.
Outpatient Adult Program
263 Port Richmond Avenue
Staten Island, NY 10302

Drug Free Residential
273 Heberton Avenue
Staten Island, NY 10302

Tier 2
1111 Front Capadanno Boulevard
Staten Island, NY 10306

Chemical Dependency North/ SIUH
450 Seaview Avenue
Staten Island, NY 10305

Project Hospitality, Inc.
Women's Recovery Program
100 Central Avenue
Staten Island, NY 10301

Saint Vincent's Hospital Medical Center
Alcoholism Outpatient Clinic/DWI Program
1794 Richmond Road
Staten Island, NY 10306

Seamens Society for Children and Families Substance Abuse Treatment Services
25 Hyatt Street
Staten Island, NY 10301

Sisters of Charity Healthcare, Inc.
Bayley Seton Campus Outpatient
75 Vanderbilt Avenue
Staten Island, NY 10304

Saint Vincents Campus Richmond
427 Forest Avenue
Staten Island, NY 10301

South Beach Alcoholism Treatment Center
777 Seaview Avenue
South Beach Psychiatric Center
Building A
Staten Island, NY 10305

Staten Island Children's Council, Inc. Drug Free Outpatient
420 Target Street
Staten Island, NY 10304

Staten Island University Hospital
Drug Free Services
Key Extended Entry Program
Methadone Maintenance
Treatment Program
392 Seguine Avenue
Staten Island, NY 10309

Chemical Dependancy Rehab
Alcohol and Drug Detox
375 Seguine Avenue
Staten Island, NY 10309

Alcoholism Outpatient Clinic
376 Seguine Avenue
Staten Island, NY 10309

YMCA of Greater New York
Staten Island YMCA Counseling Services
3902 Richmond Avenue
Staten Island, NY 10312

SUFFERN

Good Samaritan Hospital of Suffern
Alcoholism Clinic
Drug Abuse Treatment Unit
255 Lafayette Avenue
Suffern, NY 10901

SWAN LAKE

Daytop Village, Inc.
Route 55
Swan Lake, NY 12783

SYOSSET

North Shore University Hospital at Plainview Alcoholism Acute Care Program
221 Jericho Turnpike
Syosset, NY 11791

Syosset Central School District Drug Abuse Program
South Woods Road
Syosset High School
Syosset, NY 11791

Syosset Counseling Center, Inc. Neighborhood SCAN/Drug Free Outpatient
23 Willis Avenue, Suite 300
Syosset, NY 11791

Kenneth Peters Center for Recovery Outpatient Alcoholism Clinic
6800 Jericho Turnpike, Suite 122-W
Syosset, NY 11791

SYRACUSE

Alcohol Services, Inc. Alcoholism Outpatient Clinic
247 West Fayette Street
Syracuse, NY 13202

Bright Path Counseling Center
7266 Buckley Road
Syracuse, NY 13212

Clinical Counseling Services Alcoholism Outpatient Clinic
70 James Street, Suite 215
Syracuse, NY 13202

Crouse Hospital
Intervention Services
Alcoholism Outpatient Clinic
Drug Free Outpatient Unit
Methadone Maintenance
Treatment Program
410 South Crouse Avenue
Syracuse, NY 13210

Crouse Health, Inc.
Commonwealth Place
6010 East Molloy Road
Syracuse, NY 13211

Alcoholism Acute Care Unit
Hospital Intervention Services
736 Irving Avenue
Syracuse, NY 13210

Forensic Consultants, Ltd. Alcoholism Outpatient Clinic
State Tower Building, Suite 700
Syracuse, NY 13202

Pelion of Central New York, Inc.
Alcoholism Outpatient Clinic
Chronic Disorders Outpatient
500 South Salina Street
Suite 218
Syracuse, NY 13202

Recovery Counseling Services
508 State Tower Building
Syracuse, NY 13202

Syracuse Behavioral Healthcare
Outpatient Services
518 James Street
Syracuse, NY 13203

The Willows Alcoholism Inpatient Program
Si Van Duyn Street
Onondaga Hill
Syracuse, NY 13215

Syracuse Brick House, Inc.
Men's Halfway House
121 Green Street
Syracuse, NY 13203

Women's Halfway House
3606 James Street
Syracuse, NY 13206

Syracuse Community Health Center
Alcoholism Outpatient Clinic
Ambulatory Substance Abuse Services
819 South Salina Street
Syracuse, NY 13202

Veterans' Affairs Medical Center
800 Irving Avenue
Syracuse, NY 13210

Yost, Inc. Center for Individual and Family Development
205 South Salina Street, 2nd Floor
Syracuse, NY 13202

TARRYTOWN

Phelps Memorial Hospital Center Alcoholism Inpatient Rehabilitation Program
701 North Broadway Street
Tarrytown, NY 10591

Phelps Mental Health Center Threshold Program/Alcohol Outpatient Clinic
38 Beekman Avenue
Tarrytown, NY 10591

TICONDEROGA

Saint Josephs Rehabilitation Center Alcoholism Halfway House
Moses Ludington Hospital Pavilion
Wicker Street
Ticonderoga, NY 12883

TONAWANDA

Beacon Center
Alcoholism Outpatient Clinic
Drug Abuse Outpatient Clinic
2440 Sheridan Drive
Tonawanda, NY 14150

Horizon Health Services, Inc.
Addictions Outpatient
1370 Niagara Falls Boulevard
Tonawanda, NY 14150

TROY

Hudson Mohawk Recovery Center Alcoholism and Drug Outpatient Clinic
16 First Street
Troy, NY 12180

Pahl, Inc. Drug Abuse Treatment Services
106–108 9th Street
Troy, NY 12180

Pahl Transitional Apartments
2239–2243 5th Avenue
Troy, NY 12180

Rensselaer County Mental Health Unified Services
Outpatient Drug Free Program
7 Avenue and State Street
County Office Building
Troy, NY 12180

Samaritan Hospital Detoxification Service
2215 Burdett Avenue
Troy, NY 12180

Seton Addiction Services at Saint Marys Hospital
1300 Massachusetts Avenue
Troy, NY 12180

TRUMANSBURG

Ithaca Alpha House Center, Inc. Residential
6625 Route 227
Trumansburg, NY 14886

TUCKAHOE

The Maxwell Institute of St. Vincent's Hospital
92 Yonkers Road
Tuckahoe, NY 10707

TUPPER LAKE

Saint Josephs Rehabilitation Center Alcoholism Outpatient Clinic
114 Wawbeek Avenue
Tupper Lake, NY 12986

UTICA

Dam Counseling Services Drug Abuse Clinic
250 Genesee Street, Suite 306
Utica, NY 13502

Insight House Chemical Dependency Services
500 Whitesboro Street
Utica, NY 13501

McPike Alcoholism Treatment
Center Alcoholism Inpatient Rehab Unit
1213 Court Street
Mohawk Valley Psychiatric Center
Utica, NY 13502

Rescue Mission of Utica, Inc. Alcohol Crisis Center
210 Lansing Street
Utica, NY 13501

VALHALLA

Weekend Center, Inc. Generations Alcoholism Outpatient Clinic
7-11 Legion Drive
Valhalla, NY 10595

Westchester County Medical Center
Behavioral Health Clinic
Valhalla Campus
Valhalla, NY 10595

VALLEY STREAM

Friends of Bridge, Inc. Drug Abuse Treatment Program
5–11 Pflug Place
Valley Stream, NY 11580

WALTON

Delaware Valley Hospital
Alcoholism Inpatient Rehabilitation
1 Titus Place
Walton, NY 13856

WAMPSVILLE

Madison County Alcohol and Drug Abuse Program
North Court Street
Veterans Memorial Building
Wampsville, NY 13163

WANTAGH

Southeast Nassau Guidance Center (SNG)
Alcoholism Counseling and Treatment
3401 Merrick Road
Wantagh, NY 11793

WARSAW

Allegany Rehab Associates, Inc.
Wyoming County Chemical Abuse Treatment Program
422 North Main Street
Warsaw, NY 14569

WARWICK

Sleepy Valley Center
Alcoholism Inpatient/Outpatient Rehabilitation Unit
117 Sleepy Valley Road
Warwick, NY 10990

WATERLOO

Seneca County Community Counseling Center Alcoholism Outpatient Clinic
31 Thurber Drive
Waterloo, NY 13165

WATERTOWN

Community Center for Alcoholism of Jefferson County
Alcoholism Outpatient Clinic
595 West Main Street
Watertown, NY 13601

Men's Halfway House
417 Washington Street
Watertown, NY 13601

Women's Halfway House
1130 State Street
Watertown, NY 13601

Credo Foundation, Inc. Drug Treatment Program
138 Winthrop Street
Watertown, NY 13601

WAVERLY

Tioga County Alcohol and Drug Services Satellite
284 Route 17-C
Waverly, NY 14892

WEBSTER

Delphi Drug and Alcohol Council
Drug Free Outpatient
55 East Main Street
Webster, NY 14580

WELLSVILLE

Allegany Area Council on Alcoholism
Trapping Brook House
3084 Trapping Brook Road
Wellsville, NY 14895

Drug Abuse Outpatient Clinic
76 Park Avenue
Wellsville, NY 14895

WEST BABYLON

Nepenthe, Inc.
1 Farmingdale Road, Route 109
West Babylon, NY 11704

WESTBURY

North Shore Child/Family Guidance Association
Chemical Dependency for Youth
50 Sylvester Street
Westbury, NY 11590

WESTHAMPTON BEACH

Greater Hamptons Interfaith Council Outpatient Drug Abuse Clinic
Main Street
Beinecke Building
Westhampton Beach, NY 11978

Seafield Center, Inc. Alcoholism Inpatient Rehabilitation Unit
7 Seafield Lane
Westhampton Beach, NY 11978

WEST HEMPSTEAD

Long Island Jewish Hillside Hospital Medical Center
Project Outreach
600 Hempstead Turnpike
West Hempstead, NY 11552

WESTON MILLS

Cattaraugus County Council on
Alcohol and Substance Abuse Program/Weston's Manor
Route 417
Weston Mills, NY 14788

WEST POINT

US Army MEDDAC Department of U.S. Army
Building 684
West Point, NY 10996-1197

WEST SENECA

Health Care Plan, Inc. Alcoholism Outpatient Clinic
130 Empire Drive
West Seneca, NY 14224

WHITE PLAINS

Greenburgh Open Door
5 Prospect Avenue, 2nd Floor
White Plains, NY 10607

Halfway Houses of Westchester,
Inc. Hawthorne House Alcoholism Halfway House
14 Longview Avenue
White Plains, NY 10605

Innovative Health Systems Inc (IHS) Drug Abuse Treatment Unit
7 Holland Avenue
White Plains, NY 10603

New York and Presbyterian Hospital
Alcoholism Inpatient/Outpatient Rehabilitation Unit
21 Bloomingdale Road
White Plains, NY 10605

Saint Agnes Hospital Inpatient Substance Abuse Detox
305 North Street/Two Gaisman
White Plains, NY 10605

Treatment Center of Westchester Alcoholism Outpatient Clinic
10 Mitchell Place
White Plains, NY 10601

West Help Greenburgh
1 West Help Drive
White Plains, NY 10603

White Plains Hospital Medical Center Methadone Maintenance Treatment Program
Davis Avenue East Post Road
White Plains, NY 10601

Yonkers General Hospital Greenburgh Alcohol Treatment Services
30 Manhattan Avenue
White Plains, NY 10607

WILLARD

New York Services Department of Correctional Services
Willard Drug Treatment Campus
7116 County Route 132
Willard, NY 14588

WOODMERE

Tempo Group, Inc.
Drug Abuse Treatment/Intensive Program
Outpatient Drug Free Unit
Prevention Unit
112 Franklin Place
Woodmere, NY 11598

YONKERS

Renaissance Project, Inc.
Chemical Dependency Treatment Facility
42 Warburton Avenue
Yonkers, NY 10701

Saint Joseph's Hospital
Drug Free Counseling
107 South Broadway
Yonkers, NY 10701

Methadone Maintenance Treatment Program
8 Guion Street
Yonkers, NY 10701

Weekend Center, Inc. Generations Alcoholism Outpatient Clinic
70 Ashburton Avenue
Yonkers, NY 10701

Yonkers General Hospital
Alcoholism Acute Care Program
Substance Detox
2 Park Avenue
Yonkers, NY 10703

Methadone Maintenance Treatment Program
70 Ashburton Avenue
Yonkers, NY 10701

Yonkers Residential Center
Breakaway Alcoholism Outpatient Clinic
317 South Broadway
Yonkers, NY 10705

Residential Treatment Program for Youth
100 North Broadway Street
Yonkers, NY 10705

NORTH CAROLINA

AHOSKIE

Roanoke/Chowan Human Services Center
Route 3, Box 22A
Ahoskie, NC 27910

ALBEMARLE

Albemarle House, Inc.
242 North 2nd Street
Albemarle, NC 28001

Piedmont Behavioral Healthcare
1000 North 1st Street, Suite 1
Albemarle, NC 28001-2833

ASHEBORO

Alpha House, Inc.
1006 Sunset Avenue
Asheboro, NC 27203

Randolph County Mental Health/
DD and Substance Abuse Services Program
110 West Walker Road
Asheboro, NC 27203

ASHEVILLE

ARP/Phoenix LLP
129 Biltmore Avenue
Asheville, NC 28801

Blue Ridge Center Adult Substance Abuse Program
283 Biltmore Avenue
Asheville, NC 28801

Horizon Recovery
31 College Place, Suite 304-D
Asheville, NC 28801-2483

Mountain Treatment Center
260 Merrimon Avenue
Asheville, NC 28801

Neil Dobbins Center
277 Biltmore Avenue
Asheville, NC 28801

Veterans' Affairs Medical Center Substance Abuse Treatment Program
1100 Tunnel Road
Asheville, NC 28805

BELMONT

Carolinas Counseling Consulting
35 North Main Street
Belmont, NC 28012-3155

BLACK MOUNTAIN

Alcohol and Drug Abuse Treatment Center
301 Tabernacle Road
Black Mountain, NC 28711

Robert Swain Recovery Center
1280 Old U.S. 70
Black Mountain, NC 28711

BOONE

New River Behavioral Health Services
132 Poplar Grove Connector
Boone, NC 28607

Yadkin Valley Extended Services
252 East King Street
Boone, NC 28607-4042

BREVARD

Bridgeway/A Division of Transylvania Community Hospital, Inc.
Hospital Drive
Brevard, NC 28712

BRYSON CITY

Smoky Mountain Counseling Center
80 Academy Street
Bryson City, NC 28713-0181

BUIES CREEK

Lee/Harnett Mental Health Center
5841 U.S. 421 South
Buies Creek, NC 27506

BURLINGTON

Alamance Caswell Area MH/DD and Substance Abuse Program
319 North Graham Hopedale Road
Suite A
Burlington, NC 27217

Alamance Regional Medical Center
1240 Huffman Mill Road
Burlington, NC 27215

Residential Treatment Services of Alamance
136 Hall Avenue
Burlington, NC 27215

BURNSVILLE

New Hope Counseling
525 West Main Street, Suite 1
Burnsville, NC 28714-2834

BUTNER

Alcohol and Drug Abuse Treatment Center
101 North Broad Street
Butner, NC 27509

CAMP LEJEUNE

Naval Hospital Alcohol Rehabilitation Department
Building 326
Camp Lejeune, NC 28542

CANDLER

First Step Farm of Western North Carolina, Inc.
214 Black Oak Cove Road
Candler, NC 28715

CARRBORO

Orange/Person/Chatham Mental Health Center Substance Abuse Services
101 East Weaver Street Suite 300
Carrboro, NC 27510

CARTHAGE

Sandhills Teen Challenge
444 Farm Life School Road
Carthage, NC 28327-9126

CHAPEL HILL

Freedom House Recovery Center
1477 Airport Road
Chapel Hill, NC 27514

CHARLESTON AFB

Charleston Air Force Base Substance Abuse Program
437 MDOS/SGOMH
204 West Hill Boulevard
Charleston AFB, NC 29404-4704

CHARLOTTE

Amethyst Charlotte, Inc.
1715 Sharon Road West
Charlotte, NC 28210

Assessment Dynamics
3127 Eastway Drive, Suite 212
Charlotte, NC 28205-5643

Behavioral Health Center Mercy
2001 Vail Avenue
Charlotte, NC 28207

Charlotte Rescue Mission
907 West First Street
Charlotte, NC 28202

Charter Pines Hospital
3621 Randolph Road
Charlotte, NC 28211

Chemical Dependency Center
100 Billingsley Road
Charlotte, NC 28211

Dillworth Center for Chemical Dependency
429 East Boulevard
Charlotte, NC 28203

McLeod Addictive Disease Center
145 Remount Road
Charlotte, NC 28203

Mecklenburg County Area Mental
Health Authority Substance Abuse Services
429 Billingsley Road
2nd Floor
Charlotte, NC 28211

New Beginnings of Southern Piedmont LLC
1508 Cleveland Avenue
Charlotte, NC 28203

Serenity Counseling Services
1409 East Boulevard
Charlotte, NC 28203

CHEROKEE

Cherokee Health Systems A Ye Ka Chemical Dependency Unit
Hospital Road
Cherokee, NC 28719

Unity Regional Youth Treatment Center
Sequoyah Trail Drive
Cherokee, NC 28719

CHERRY POINT

Substance Abuse Counseling Center MCAS Cherry Point
C Street, Building 294, Wing 7
Cherry Point, NC 28533

CONCORD

Cabarrus Family Recovery Center Substance Abuse Services
845 Church Street Commons, Suite 308
Concord, NC 28025

Serenity House, Inc.
172 Spring Street SW
Concord, NC 28025

Thrailkill Counseling
231 Branchview Drive NE, Suite C
Concord, NC 28025-3416

DOBSON

Hope Valley, Inc.
105 Country Home Road
Dobson, NC 27017

DURHAM

Duke Alcoholism and Addiction Program
2213 Elba Street
Durham, NC 27710

Durham Community Guidance Clinic
Turner Building
Trent and Elba Streets
Durham, NC 27705

Durham Regional Hospital Oakleigh
309 Crutchfield Street
Durham, NC 27704

Men and Women in Crisis Counseling Service
1413 Broad Street
Durham, NC 27705

Substance Abuse Services
304 West Main Street
Durham, NC 27701

Veterans' Affairs Medical Center Substance Abuse Program
508 Fulton Street
Durham, NC 27705

ELIZABETH CITY

Albemarle Mental Health Center
305 East Main Street
Elizabeth City, NC 27909

ELIZABETHTOWN

Bladen County Mental Health Center Alcoholism Program
East McKay Street
Elizabethtown, NC 28337

ELKIN

Crossroads Behavioral Health
130-A Hawthorne Lane
Elkin, NC 28621

FAYETTEVILLE

Behavioral Health Care
1830 Owen Drive, Suite 103
Fayetteville, NC 28304

Cardinal Clinic
351 Wagoner Drive, Suite 400
Fayetteville, NC 28303-4608

Cumberland County Mental
Health Center Family Recovery Services
109 Bradford Avenue
4th Floor
Fayetteville, NC 28301

Raintree Clinic
804 Stamper Road, Suite 201
Fayetteville, NC 28303

Roxie Avenue Center Substance Abuse Services
1724 Roxie Avenue
Fayetteville, NC 28304

FRANKLIN

Smoky Mountain Counseling Center
100 Thomas Heights Road
Franklin, NC 28734

GASTONIA

Family Service, Inc.
214 East Franklin Boulevard
Gastonia, NC 28052

Flynn Fellowship Home of Gastonia, Inc.
311 South Marietta Street
Gastonia, NC 28052

McLeod Addictive Disease Center
418 West Main Avenue
Gastonia, NC 28053-0596

New Beginnings of Gaston County
430 West Franklin Boulevard
Gastonia, NC 28052

GOLDSBORO

Carolina Care Center
206 North Spence Avenue
Goldsboro, NC 27534

Department of Corrections DART Cherry Facility
West Ash Street
Goldsboro, NC 27533

Wayne County Mental Health Center
301 North Herman Street
County Office Building
Goldsboro, NC 27530

GRAHAM

Family Consultants, Inc.
219 East Elm Street
Graham, NC 27253

GREENSBORO

Alcohol and Drug Services of Guilford
312 North Eugene Street
Greensboro, NC 27401

301 East Washington Street
Suite 101
Greensboro, NC 27403

Alternative Counseling Center
5415 West Friendly Street
Greensboro, NC 27410

Assessment Counseling and Testing Services
320 South Eugene Street
Greensboro, NC 27401

Employee Counseling Associates, Inc.
612 Pasteur Drive Suite 207
Greensboro, NC 27403

Fellowship Hall
5140 Dunstan Road
Greensboro, NC 27405

Guilford County Mental Health Center Substance Abuse Program
201 North Eugene Street
Greensboro, NC 27401

Jacqueline W Trotter Associates, Inc.
612 Pasteur Drive, Suite 104
Greensboro, NC 27403-1120

Ringer Center
213 East Bessemer Avenue
Greensboro, NC 27401-1415

Southeastern Counseling Center
1207 West Bessemer Avenue
Suite 227
Greensboro, NC 27408

TRC Counseling
1401 Sunset Drive, Suite 203
Greensboro, NC 27408-7230

GREENVILLE

Hatteras House
215 South Meade Street
Greenville, NC 27858

Pitt County Mental Health Center
203 Government Circle
Greenville, NC 27834

Side By Side Recovery Program
315 South Evans Street, Suite B
Greenville, NC 27858-1832

W. B. Jones Alcohol and Drug Abuse Treatment Center
2577 West Fifth Street
Greenville, NC 27834

HENDERSON

Franklin/Granville/Vance/
Warren Area Mental Health Program
125 Emergency Road
Henderson, NC 27536

HENDERSONVILLE

Horizon Recovery
132-B 3rd Avenue East
Hendersonville, NC 28792-4302

Trend Community Mental Health Services
800 Fleming Street
Hendersonville, NC 28739

HICKORY

Alcohol and Drug Abuse Services of Catawba County
120 Fairgrove Church Road SE
Suite 23
Hickory, NC 28602

Phoenix Lawhon
910 Tate Boulevard SE
Suite 102
Hickory, NC 28602

HIGH POINT

Alcohol and Drug Services of Guilford
119 Chestnut Drive
High Point, NC 27262

Adult Residential
5209 West Wendover
High Point, NC 27260

High Point Behavioral Health
601 North Elm Street
High Point, NC 27261-1899

Incentives, Inc.
212 East Green Drive
High Point, NC 27260-6654

JACKSONVILLE

Bryann Marr Behavioral Healthcare Systems
192 Village Drive
Jacksonville, NC 28546

Chemical Dependency Training Evaluation and Guidance, Inc.
230 New Bridge Street
Jacksonville, NC 28540

Onslow County Behavioral Health Services
215 Memorial Drive
Jacksonville, NC 28546

U.S. Marine Corp Substance Abuse Counseling Center
Marine Corps Air Station New River
Jacksonville, NC 28540-5000

JAMESTOWN

Alcoholics Home, Inc.
5884 Riverdale Road
Jamestown, NC 27282

KENANSVILLE

Chemical Dependency Training Evaluation and Guidance
106 South Street, Suite E
Kenansville, NC 28349

Duplin/Sampson Area MH/DD/SAS Kenansville and Clinton Division Outpatient
117 Beasley Street
Kenansville, NC 28349

KERNERSVILLE

Twin City Counseling Center Inc
119 South Main Street
Kernersville, NC 27284

KINSTON

Lenoir Area MH/MR Substance Abuse Program
2901 North Heritage Street
Kinston, NC 28501

LAURINGURG

Scotland County Mental Health Center Substance Abuse Services
1224 Biggs Street
Laurinburg, NC 28352

LENOIR

Foothills Mental Health Center
901 Ashe Avenue
Lenoir, NC 28645

LEXINGTON

Davidson Alcoholic Care, Inc.
1675 East Center Street
Lexington, NC 27292

Davidson Assessment and Counseling
110-C Cotton Grove Road
Lexington, NC 27292

LINCOLNTON

Acts in Recovery, Inc.
326 East Main Street
Lincolnton, NC 28092

LOUISBURG

Franklin County Mental Health Clinic
107 Industrial Drive, Suite B
Louisburg, NC 27549

Genesis Substance Abuse Services
167 Highway 56 East
Louisburg, NC 27549-9449

LUMBERTON

Carolina Manor Treatment Center
1100 Pine Run Drive
Lumberton, NC 28358

Robeson County Mental Health Clinic
Non-Hospital Detoxification Center
450 Country Club Road
Lumberton, NC 28359

207 West 29th Street
Lumberton, NC 28358

MARBLE

Smoky Mountain Counseling Center
Highway 19
Marble, NC 28905

MARION

Foothills Mental Health Program
122 South Main Street
Marion, NC 28752

McDowell Council on Alcohol and Drug Abuse
17 North Garden Street
Marion, NC 28752

MONROE

Friendship Home, Inc.
2111 Stafford Street Extension
Monroe, NC 28110

New Beginnings of Southern Piedmont LLC
5719 Highway 74 West
Monroe, NC 28110

Piedmont Behavioral Health Union Center
1190 West Roosevelt Boulevard
Monroe, NC 28110

Union Regional Medical Center Behavorial Health Center/ First Step
600 Hospital Drive
Monroe, NC 28110

MOREHEAD CITY

Carteret Counseling Services, Inc.
105 North 10th Street
Morehead City, NC 28557

MORGANTON

Broughton Hospital
1000 South Sterling Street
Morganton, NC 28655

Foothills Area Mental Health Center
1001 B East Union Street
Morganton, NC 28655

Foothills Detox/Crisis Program
2130 NC 18/U.S. 64
Morganton, NC 28655

TLC Human Resources, Inc.
132 South Sterling Street
Evion Building
Morganton, NC 28680-1447

MOUNT AIRY

Crossroads Behavioral Health Center
351 Riverside Drive
Mount Airy, NC 27030

Delphi Counseling Services
201 North Main Street, Suite 307
Mount Airy, NC 27030

NEW BERN

Assessment and Counseling Services,Inc.
249 Craven Street
New Bern, NC 28560

Child Family Psychological
1425 South Glenburnie Road, Suite 1
New Bern, NC 28562-2610

New Bern Family Services Substance Abuse Services
403 George Street
New Bern, NC 28563

NEWLAND

New River Mental Health Center Avery Cares Center
636 Cranberry Street
Newland, NC 28657

NEWTON

Doris Lasley and Associates Abuse Services
116 North College Avenue
Newton, NC 28658-3237

NORTH WILKESBORO

New River Mental Health Wilkesboro Detox Unit
118 Peace Street
North Wilkesboro, NC 28659

PEMBROKE

Robeson Health Care Corp Our House
302 East 3rd Street
Pembroke, NC 28372

PILOT MOUNTAIN

Hope Valley/Women's Division
136 Hope Valley Road
Pilot Mountain, NC 27041

PINEHURST

Moore Regional Hospital Pinehurst Treatment Center
Page Road
Pinehurst, NC 28374

PITTSBORO

Chatham Counseling Center
40 Camp Drive
Pittsboro, NC 27312

POPE AFB

Pope Air Force Base Substance Abuse Program
23 MDOS/SGOMH
383 Maynard Street
Pope AFB, NC 28308-2383

RALEIGH

Charter Behavioral Health System Holly Hill/Charter Behavioral Health
3019 Falstaff Road
Raleigh, NC 27610

Jamie Norton and Associates Keys to Recovery
1110 Navaho Drive
Tower One Building, Suite 103
Raleigh, NC 27609

PSI Solution Center
801 Jones Franklin Road
Suite 210
Raleigh, NC 27606-3381

Pathways Counseling Center
2809 Highwoods Boulevard
Suite 103
Raleigh, NC 27604

Recovery Partnership, Inc.
3900 Barrett Drive
Suite 301
Raleigh, NC 27609

Southlight, Inc. Community Treatment Project Lifeplus
2101 Old Garner Road, Suite 111
Raleigh, NC 27610

Wake County Alcoholism Treatment Center
3000 Falstaff Road
Raleigh, NC 27610

REIDSVILLE

Rockingham County Mental
Health Center Substance Abuse Services
405 NC 65
Reidsville, NC 27320

ROANOKE RAPIDS

Riverstone Counseling and Personal Development
210 Smith Church Road
Roanoke Rapids, NC 27870

ROBBINSVILLE

Smoky Mountain Counseling Center
217 South Main Street
Robbinsville, NC 28771

ROCKINGHAM

Recovery Associates
208 East Franklin Street, Suite C
Rockingham, NC 28379-3640

Samaritan Colony
136 Samaritan Drive
Rockingham, NC 28379

ROCKY MOUNT

Edgecombe/Nash Mental Health Center Substance Abuse Program
500 Nash Medical Arts Mall
Rocky Mount, NC 27804

Urton Associates
3300 Sunset Avenue
Rocky Mount, NC 27804-3571

ROXBORO

Person County Mental Health Center
204 West Barden Street
Roxboro, NC 27573

SALISBURY

Rowan Regional Medical Lifeworks Center
612 Mocksville Avenue
Salisbury, NC 28144

Veterans' Affairs Medical Center Substance Abuse Treatment Program
1601 Brenner Avenue
Unit 4-2B (116A3)
Salisbury, NC 28144

SANFORD

Harbor Clinic
138 South Steele Street
Sanford, NC 27330-4201

SELMA

Day by Day Treatment Center
1110 River Road
Selma, NC 27576

SHALLOTTE

Coast Behavioral Health Services
624 Village Road, Suite 1
Shallotte, NC 28470

SHELBY

Cleveland Center
917 First Street
Shelby, NC 28150

New Beginnings of Southern Piedmont
115 North Lafayette Street
Shelby, NC 28150-4445

Wellness Training Association, Inc.
217 North Lafayette Street
Shelby, NC 28150

SMITHFIELD

Johnston Substance Abuse
Program Treatment and Rehabilitation
521 Bright Leaf Boulevard
Smithfield, NC 27577

SOUTHERN PINES

Recovery Associates
770 NW Broad Street
Southern Pines, NC 28387

SPARTA

New River Mental Health Center
West Doughton Street
Sparta, NC 28675

SPINDALE

Rutherford Substance Abuse Services
271 Callahan Koon Road
Spindale, NC 28160-2207

STATESVILLE

Carolina Psychiatric Group
515 Brookdale Drive
Statesville, NC 28677

Counseling Center of Iredell
125 West Bell Street
Statesville, NC 28677

Steps to Success
211 South Center Street
City Center Building, 4th Floor
Statesville, NC 28677-5258

TARBORO

Urton Associates
102 East Granville Street
Tarboro, NC 27886-5002

TAYLORSVILLE

Foothills Mental Health Center Alcohol and Drug Abuse Program
326 First Avenue SW
Taylorsville, NC 28681

THOMASVILLE

Davidson County MH/DD and Substance Abuse Services
205 Old Lexington Road
Thomasville, NC 27360

Green Center of Growth Development
25 West Guilford Street
Thomasville, NC 27360-3945

WARRENTON

John A. Hyman Substance Abuse Services
Rural Route 3
Warrenton, NC 27589-9803

WASHINGTON

Tideland Mental Health Center Substance Abuse Division
1308 Highland Drive
Washington, NC 27889

WAYNESVILLE

Gateway
1406 Dellwood Road
Waynesville, NC 28786

Smokey Mountain Counseling Center
131 Walnut Street
Waynesville, NC 28786

Smoky Mountain Area Mental Health Haywood County Center
1207 East Street
Waynesville, NC 28786

WEST END

Sandhills Mental Health Center Substance Abuse Services
7 Lakes Drive
West End, NC 27376

WEST JEFFERSON

Yadkin Valley Extended Services
106 Jefferson Drive
West Jefferson, NC 28694-7245

WHITEVILLE

Columbus County Mental Health Center
306 Jefferson Street
Whiteville, NC 28472

WHITTIER

Smoky Mountain Center for MH/MR/SA Services Substance Abuse Services Program
1450 Smoky Cove Road
Whittier, NC 28789

WILKESBORO

New River Substance Abuse Services Wilkes County
1226 School Street
Wilkesboro, NC 28697

Yadkin Valley Extended Services
Wilkesboro, NC 28697

WILMINGTON

Coastal Horizons Center Outpatient Treatment Services
721 Market Street, 3rd Floor
Wilmington, NC 28401

Harvest of Wilmington, Inc.
3805 Wrightsville Avenue, Suite 17
Wilmington, NC 28403-8464

Kelly House East Coast Solutions
1507 Martin Street
Wilmington, NC 28401-6483

Recovery Center of Richmond
2520 Troy Drive
Wilmington, NC 28401

Southeastern Center for Mental Health/DD Substance Abuse Services
2023 South 17 Street
Wilmington, NC 28401

Stepping Stone Manor
416 Walnut Street
Wilmington, NC 28401

WILSON

Alternatives
2122 West Nash Road
Wilson, NC 27893-1728

Wilson/Greene Substance Abuse Center
208 North Goldsboro Street
Wilson, NC 27895

WINSTON SALEM

Addiction Recovery Care Association
1931 Union Cross Road
Winston Salem, NC 27107

Behavioral Health Resources Novant Health Resources Network
3333 Silas Creek Parkway
Winston Salem, NC 27103

Centerpoint Substance Abuse Services
725 North Highland Avenue
Winston Salem, NC 27101

First, Inc.
316 North Spring Street
Winston Salem, NC 27101

Friendship House
533 Summit Street
Winston Salem, NC 27101

Lifeskills
1001 South Marshall Street
Winston-Salem, NC 27101

Step One, Inc.
665 West 4th Street
Winston Salem, NC 27101

WINTERVILLE

Community Wellness Center Recovery Place
108 West Firetower Road, Suite H
Winterville, NC 28590

YADKINVILLE

Surry Yadkin Area MH/DD/SA Authority
320 East Lee Avenue
Yadkinville, NC 27055

Yadkin Valley Counseling Services
202 East Main Street
Yadkinville, NC 27055

NORTH DAKOTA

BELCOURT

Turtle Mountain Counseling Center
Highway 5
Belcourt, ND 58316

BISMARCK

Basaraba, Rose, LAC Counseling Services
433 East Bismarck Expressway, Suite 3
Bismarck, ND 58504-6511

Burleigh County Detoxification Center
514 East Thayer Avenue
Burleigh County Sheriff's Department
Bismarck, ND 58501

De Counseling Service
418 East Rosser Avenue, Suite E
Bismarck, ND 58501

Heartview Foundation
105 East Broadway
Bismarck, ND 58501

New Freedom Center
2101 East Broadway
Bismarck, ND 58501

North Dakota State Penitentiary Addiction Treatment Program
Bismarck, ND 58502

West Central Human Service Center Chemical Dependency Program
600 South 2 Street
Bismarck, ND 58504

Whole Person Recovery Center
1138 Summit Boulevard
Bismarck, ND 58504

DEVILS LAKE

Alternatives to High Risk Substance Use
UND/Lake Region
1801 College Drive North
Devils Lake, ND 58703-1111

Lake Region Human Service Center Chemical Dependency Program
Highway 2 West
Devils Lake, ND 58301

DICKINSON

Badlands Human Service Center Chemical Dependency Program
Dickinson State University Campus
Pulver Hall
Dickinson, ND 58601

Heart River Alcohol/Drug Abuse Services
7 1st Avenue West, Suite 101
Dickinson, ND 58601

Prairie Echoes Counseling Services
135 West Villard Street
Dickinson, ND 58601-5121

FARGO

Centre, Inc.
123 North 15th Street
Fargo, ND 58107

Drake and Burau Counseling Services
1202 23rd Street South, Suite 6
Fargo, ND 58103

Human Services Associate
806 6th Avenue North
Fargo, ND 58102

Meritcare Hospital Psychiatric Services/Partial Hospitalization
720 4th Street North
Fargo, ND 58102

Meritcare Neuroscience Clinic
700 South First Avenue
Fargo, ND 58103

Prairie Psychiatric Center
510 4th Street South
Fargo, ND 58107-0827

Share House
4227 9th Avenue SW
Fargo, ND 58103

Southeast Human Service Center Alcohol and Drug Abuse Unit
2624 9 Avenue South
Fargo, ND 58103

Veterans' Affairs Medical Center Substance Abuse Treatment Program
2101 Elm Street North
Fargo, ND 58102

FORT TOTTEN

Spirit Lake Nation Recovery and Wellness Program
Fort Totton, ND 58335

FORT YATES

Standing Rock Nation Comprehensive Chemical Prevention Program
Main Street
Fort Yates, ND 58538

GARRISON

Ron Stanley Counseling Service
36 3 Avenue NW
Garrison, ND 58540

GRAFTON

MAB Counseling Services
625 Hill Avenue
Grafton, ND 58237

GRAND FORKS

Alcohol and Drug Services, Inc.
311 South 4th Street, Suite 1
Grand Forks, ND 58201-4726

Altru Hospital
1200 South Columbia Road
Grand Forks, ND 58201-6007

Wright, Katy Substance Abuse Counseling
1407 South 24 Avenue
Suite 214
Grand Forks, ND 58201

Northeast Human Service Center Chemical Dependency Program
1407 24 Avenue South
Grand Forks, ND 58201

Northridge Counseling Center, Inc.
215 North 3 Street, Suite 100
Grand Forks, ND 58203

JAMESTOWN

Alcohol/Families and Children
Jamestown Mall, Suite 221
Jamestown, ND 58401

DUI Seminar Program
624 9th Avenue SE
Jamestown, ND 58401

North Dakota State Hospital Chemical Dependency Unit
Jamestown, ND 58402

Northern Prairie Consultants
115 2nd Street SW
Jamestown, ND 58401

South Central Human Service Center Chemical Dependency Program
520 3rd Street NW
Jamestown, ND 58402

MANDAN

North Dakota Youth Counseling Program
701 16th Avenue SW
Mandan, ND 58554

MINOT

Bachmeier Counseling
1809 South Broadway Street
Minot, ND 58701

Dakota Boys' Ranch
6301 19th Avenue NW
Minot, ND 58702

Gateway Counseling Center
315 South Main Street
Suite 307-A
Minot, ND 58701

Mental Health Addiction Services Trinity Hospital
Burdick Expressway Main Street
Minot, ND 58702-5020

North Central Human Service
Center Chemical Dependency Program
400 22 Avenue NW
Minot, ND 58701

Unimed Medical Center Chemical Dependency Services
600 17th Avenue SE
Minot, ND 58701

MINOT AFB

Minot Air Force Base Substance Abuse Program
5 MDOS/SGOMH
10 Missile Avenue
Minot AFB, ND 58705-5024

NEW TOWN

Three Affiliated Tribes Circle of Life Alcohol Program
302 North Breslin Addition Street
New Town, ND 58763

TRENTON

Native American Resource Center
Trenton, ND 58853

WILLISTON

Mercy Recovery Center
1213 15 Avenue West
Williston, ND 58801

Northwest Human Service Center Chemical Dependency Program
316 2 Avenue West
Williston, ND 58801

OHIO

AKRON

Adolescent Counseling and Treatment, Inc.
Akron, OH 44319

Akron Health Department Alcoholism Division
177 South Broadway
Akron, OH 44308

Akron Urban Minority Alcohol and Drug Abuse Outreach Program, Inc. Addiction Treatment Services
665 West Market Street, Suite F
Akron, OH 44303

Community Drug Board
Women's Recovery Center
725 East Market Street
Akron, OH 44305

Genesis Program
386 South Portage Path
Akron, OH 44320

Ramar Center
380 South Portage Path
Akron, OH 44320

Family Services of Summit County
212 East Exchange Street
Akron, OH 44304

Interval Brotherhood Homes, Inc. Alcohol Rehabilitation Center
3445 South Main Street
Akron, OH 44319

Oriana House
ADM Crisis Center
15 Frederick Street
Akron, OH 44304

Adolescent Residential Center
885 East Buchtel Avenue
Akron, OH 44305

Community Based Correctional Facility
264 Crosier Street
Akron, OH 44311

Glenwood Site
40 East Glenwood Avenue
Akron, OH 44304

Residential Correction Center
222 Power Street
Akron, OH 44304

Senior Workers' Action Program Chemical Dependency Services
415 Portage Path
Akron, OH 44320

Tri-County Employee Assistance Program
450 Grant Street
Suite 2411
Akron, OH 44311

Urban Ounce of Prevention Services, Inc.
1501 Smith Hawkins Avenue
Akron, OH 44320

ALLIANCE

Quest Recovery Services, Inc. Alliance Division
724 South Union Street
Alliance, OH 44601

ALVORDTON

Fresh Start Home I
109 West Main Street
Alvordton, OH 43501

Fresh Start Home II
405 East Main Street
Alvordton, OH 43501

ASHLAND

Appleseed CMHC
1126 Cottage Street
Ashland, OH 44805

Ashland County Council on Alcoholism and Drug Abuse, Inc.
310 College Avenue
Ashland, OH 44805

ASHTABULA

Lake Area Recovery Center
Outpatient Drug Free Program
2801 C Court
Ashtabula, OH 44004

Turning Point
2711 Donohoe Drive
Ashtabula, OH 44004

ATHENS

Health Recovery Services, Inc.
Athens County Outpatient Clinic
100 Hospital Drive
Athens, OH 45701

Bassett House
10050 Bassett Road
Athens, OH 45701

Rural Women's Recovery Program
9908 Bassett Road
Athens, OH 45701

BAINBRIDGE

Lighthouse Youth Center Paint Creek Alcohol and Drug Outpatient Treatment
1071 Tong Hollow Road
Bainbridge, OH 45612

BATAVIA

Clermont Recovery Center, Inc.
Outpatient Services
2379 Clermont Center Drive
Batavia, OH 45103

Jail Program
4700 Filager Road
Batavia, OH 45103

Family Service of the Cincinnati Area
Clermont Center
2085-A Front Wheel Drive
Batavia, OH 45103

BEACHWOOD

Glenbeigh Center of Beachwood Alcohol/Drug Outpatient Treatment
3789-B South Green Road
Beachwood, OH 44122

Jewish Family Services Assoc. of
Cleveland Alcoholism/Chemical Dependency
24075 Commerce Park Road
Beachwood, OH 44122

Laurelwood Counseling Center of Beachwood Outpatient Program
25200 Chagrin Road
Water Tower Plaza
Beachwood, OH 44122

North East Ohio Health Services Alcohol and Drug Outpatient Treatment
23210 Chagrin Boulevard
Building One, Suite 400
Beachwood, OH 44122

BELLAIRE

Crossroads Counseling Service
First National Bank Building
Suite 210–211
Bellaire, OH 43906

BELLEFONTAINE

Logan/Champaign Consolidated Care
1513 Township Road, Suite 235
Bellefontaine, OH 43311

BELMONT

Awakenings
116 Main Street
Belmont, OH 43718

BIDWELL

Family Addiction Community Treatment Services
1770 Jackson Pike
Bidwell, OH 45614

BOWLING GREEN

Behavioral Connections of Wood County
320 West Gypsy Lane Road
Bowling Green, OH 43402

Women's Residence Program
1033 Devlac Grove
Bowling Green, OH 43402

BRECKSVILLE

Veterans Addiction Recovery Center Alcohol/Drug Dependence Treatment Program
10000 Brecksville Road
Suite 116-B
Brecksville, OH 44141

BROADVIEW HEIGHTS

New Directions Alcohol and Drug Outpatient Treatment
6640 Harris Road
Broadview Heights, OH 44147

BROOK PARK

Freedom House I Counter Attack DIP
Budget Inns of America
14043 Brook Park Road
Brook Park, OH 44142

BRUNSWICK

Alcohol and Drug Dependency
Services of Medina County/ Brunswick Office
4274 Manhattan Circle Drive
Brunswick, OH 44212

BRYAN

Five County Alcohol/Drug Program
125 East South Street
Bryan, OH 43506

BUCYRUS

Community Counseling Services, Inc. Bucyrus Office
820 Plymouth Street
Bucyrus, OH 44820

CADIZ

Crossroads Counseling Service
239 West Warren Street
Cadiz, OH 43907

CALDWELL

Noble Drug Abuse and Alcoholism Council, Inc.
48 Olive Street
Caldwell, OH 43724

CAMBRIDGE

Guernsey Health Choices, Inc. Drug Addiction Treatment Center Outpatient
111 North 7th Street
Cambridge, OH 43725

CANTON

Community Treatment and
Correction Center Inc./ Substance Abuse Program
1200 Market Avenue South
Canton, OH 44707

Crisis Intervention Center of Stark County, Inc.
2421 13 Street NW
Canton, OH 44708

Quest Recovery Services, Inc.
1341 Market Avenue North
Canton, OH 44714

Quest Deliverance House/Women's Residential Treatment
626 Walnut Avenue NE
Canton, OH 44702

Quest Recovery House
215 Newton Avenue NW
Canton, OH 44703

Stark County TASC
1375 Raff Road SW
Canton, OH 44710

Veterans Addiction Recovery Center Alcohol Dependency Treatment Unit
221 3rd Street SE
Canton, OH 44702

CELINA

Gateway Outreach Center
Nonresidential Alcohol Safety Program
Outpatient Services
800 Pro Drive
Celina, OH 45822

CHAGRIN FALLS

BHC Windsor Hospital Alcohol and Drug Treatment Program
115 East Summit Street
Chagrin Falls, OH 44022

CHARDON

Lake Geauga Center on Alcoholism and Drug Abuse
200 Center Street
Chardon, OH 44024

Ravenwood Center Drug and Alcohol Treatment Services
12557 Ravenwood Drive
Chardon, OH 44024

Stillwater Adolescent Intensive Outpatient Treatment Program
695 South Street, Suite 6
Chardon, OH 44024

CHILLICOTHE

Great Seal Family Care Center
425 Chestnut Street
Suite 6
Chillicothe, OH 45601

Ross Correctional Institute Substance Abuse Program
16149 State Route 104
Chillicothe, OH 45601

Scioto Paint Valley Mental Health Center
Martha Cottrill Clinic
4449 State Route 159
Chillicothe, OH 45601

Veterans' Affairs Medical Center Substance Abuse Treatment Program
17273 State Route 104
116-A3
Chillicothe, OH 45601

CINCINNATI

Alcoholism Council of Cincinnati Area
Alice Paul House
118 East William Howard Taft Road
Cincinnati, OH 45219

Mount Airy Shelter Program
2660 Diehl Road
Cincinnati, OH 45223

Beekman Work Release Center
2438 Beekman Street
Cincinnati, OH 45214

Bethesda Alcohol and Drug Treatment
619 Oak Street
Cincinnati, OH 45206

Center for Comprehensive Alcoholism Treatment
830 Ezzard Charles Drive
Cincinnati, OH 45214

Central Community Health Board Drug Services
5240 North Bend Road
Cincinnati, OH 45239

532 Maxwell Avenue
Cincinnati, OH 45219

Crossroads Center Outpatient Treatment Services
311 Martin Luther King Drive
C Building
Cincinnati, OH 45219

Family Services of the Cincinnati Area
205 West 4 Street
Cincinnati, OH 45202

Hyde Park Counseling Center
2727 Madison Road Suite 303
Cincinnati, OH 45209

Sharonville Counseling Center
4050 Executive Park Drive
Suite 404
Cincinnati, OH 45241

First Step Home, Inc.
2118 Saint Michael Street
Cincinnati, OH 45204

Fransiscan Behavioral Health Services Chemical Dependency Program
2446 Kipling Avenue
Cincinnati, OH 45239

Ikron Corporation Alcohol and Drug Treatment Program
2347 Vine Street
Cincinnati, OH 45219

Jewish Hospital of Cincinnati, Inc. Adolescent Chemical Dependency Unit
3200 Burnet Avenue
Cincinnati, OH 45229

Norcen Behavioral Health Systems Adolescent Recovery Program
7710 Reading Road, Suite 300
Cincinnati, OH 45237

Ohio River Valley, Inc.
115 West McMicken Street
Cincinnati, OH 45210

Prospect House
682 Hawthorne Avenue
Cincinnati, OH 45205

Shaffer House
583 Grand Avenue
Cincinnati, OH 45205

Shelterhouse Volunteer Group, Inc. Drop-In Center
217 West 12 Street
Cincinnati, OH 45210

Talbert House
3123 Woodburn Avenue
Cincinnati, OH 45207

Adapt
3009 Burnet Avenue
Cincinnati, OH 45219

Adapt for Women
3595 Washington Avenue
Cincinnati, OH 45229

Adolescent Services/Alternatives
3009 Burnet Avenue
Cincinnati, OH 45219

Cornerstone
2216 Vine Street
Cincinnati, OH 45219

Extended Treatment Program
1617 Reading Road
Cincinnati, OH 45202

McMillan House for Young Men
3123 Woodburn Avenue
Cincinnati, OH 45207

Outpatient Adult Services
308 Reading Road
Cincinnati, OH 45202

SA/MI Day Treatment
2433 Iowa Avenue
Cincinnati, OH 45206

Spring Grove Center
3129 Springrove Avenue
Cincinnati, OH 45225

Talbert House for Women
1617 Reading Road
Cincinnati, OH 45207

Talbert House Turning Point
2605 Woodburn Avenue
Cincinnati, OH 45206

Veterans Affairs Medical Center Chemical Dependence Treatment Program
3200 Vine Street
Building 1 8S 151
Cincinnati, OH 45220

CIRCLEVILLE

Haven House of Pickaway County, Inc.
1180 North Court Street, Suite G
Circleville, OH 43113

Pickaway Area Recovery Services
210 Sharon Road
Circleville, OH 43113

Scioto Paint Valley Mental Health Center Pickaway County Office
145 Morris Road
Circleville, OH 43113

CLEVELAND

Alcoholism Services of Cleveland
East Unit
2490 Lee Boulevard
Suite 320
Cleveland, OH 44118

Homeless Project
2219 Payne Avenue
Cleveland, OH 44114

Probation Recovery Project
1200 Ontario Avenue Court Tower
7th Floor
Cleveland, OH 44118

Bellefaire/Jewish Children's Bureau Pact Program
22001 Fairmount Boulevard
Cleveland, OH 44118

Berea Children's Home Wrap Around Family Service Center
3235 Prospect Avenue
Cleveland, OH 44115

Buckeye Health Center Ann Nelson Perinatal Substance Abuse Program
11819 Buckeye Road
Cleveland, OH 44120

Catholic Charities Services
Hispanic Program
2012 West 25th Street, Suite 516
Cleveland, OH 44113

DePaul Family Center
2320 East 24th Street
Cleveland, OH 44115

Matt Talbot Inn
2270 Professor Avenue
Cleveland, OH 44113

Catholic Social Services Counseling of Cuyahoga County
3135 Euclid Avenue, Suite 202
Cleveland, OH 44115

Center for Families and Children
1468 West Ninth Street
Cleveland, OH 44113

AIDS Initiative Project
2728 Euclid Avenue
Cleveland, OH 44115

Cares Plus Alcohol and Drug Counseling
3955 Euclid Avenue
Cleveland, OH 44115

Hispanic Counseling
4115 Bridge Avenue Suite 309
Cleveland, OH 44113

Safe Harbor Alcohol/Drug Treatment
1145 Galewood Drive
Cleveland, OH 44110

Cleveland Clinic Alcohol and Drug Recovery Center
9500 Euclid Avenue, Desk P-48
Cleveland, OH 44106

Cleveland Health Department Center Point I
3030 Euclid Avenue
Cleveland, OH 44114

Cleveland Treatment Center, Inc.
1127 Carnegie Avenue
Cleveland, OH 44115

Community Action Against Addiction, Inc.
5209 Euclid Avenue
Cleveland, OH 44103

Community Assessment Program
5163 Broadway Avenue
Cleveland, OH 44127

Southeast Women's Center
7835 Harvard Avenue
Cleveland, OH 44127

Covenant Adolescent CD Treatment and Prevention Center
1688 Fulton Road
Cleveland, OH 44113

Cuyahoga Dept Justice Affairs Division of Youth Services/ Aftercare
1276 West 3rd Street, Suite 319
Cleveland, OH 44113

East Cleveland Straight Talk Alcohol and Drug Outpatient Treatment
12921 Euclid Avenue
Cleveland, OH 44112

Freedom House, Inc.
Alcohol and Drug Treatment Programs
12160 Triskett Road
Cleveland, OH 44111

Halfway House Treatment Program
2121 West 117th Street
Cleveland, OH 44111

East Side Catholic Shelter
11811 Shaker Boulevard
Cleveland, OH 44120

Fresh Start Alcohol and Drug Outpatient Treatment
4807 Cedar Avenue
Cleveland, OH 44103

Fresh Start II
16801 Euclid Avenue
Cleveland, OH 44112

Fresh Start III
1809 East 89th Street
Cleveland, OH 44120

Fresh Start IV
11811 Shaker Boulevard, Suite 411
Cleveland, OH 44120

Harbor Light Substance Abuse
Division Outpatient/Detox Unit 1
1710 Prospect Avenue
Cleveland, OH 44115

Hispanic Urban Minority
Alcoholism and Drug Abuse Outreach Program
3305 West 25 Street
Suite 517
Cleveland, OH 44113

Hitchcock Center for Women
1227 Ansel Road
Cleveland, OH 44108

HUMADAOP/Casa Alma/Casa Maria
3387 Fulton Road
Cleveland, OH 44109

Laurelwood Counseling Center of University Circle
1909 East 101st Street, Suite 203
Cleveland, OH 44106

McIntyre Foundation Driver Intervention Program
4805 Pearl Road
Cleveland, OH 44109

Meridia Euclid Hospital Recovery Center
18901 Lakeshore Boulevard
Cleveland, OH 44119

Meridia Health System
6700 Beta Drive Suite 200
Cleveland, OH 44143

Meridia Huron Hospital Recovery Center
13951 Terrace Road
Cleveland, OH 44112

Metrohealth Medical Center Alcohol CD Services
2500 Metrohealth Drive
Hamann Building 842
Cleveland, OH 44104

Miracle Village Chemical Dependency Treatment Program
2500 East 79th Street
Metrohealth Clement Center
Cleveland, OH 44104

Murtis H Taylor Multi-Service Center STAAR Program
13411 Union Avenue
Cleveland, OH 44120

Neighborhood Counseling Service SAMI Program
1702 West 28th Street
Cleveland, OH 44113

New Directions
30800 Chagrin Boulevard
Cleveland, OH 44124

Northeast Ohio Health Services
1909 East 101st Street Suite 201
Cleveland, OH 44106

Northeast Pre-Release Center Substance Abuse Services
2675 East 30 Street
Cleveland, OH 44101

Project East
22001 Fairmount Boulevard
Cleveland, OH 44118

Recovery Resources
3950 Chester Avenue
Cleveland, OH 44114-4625

Prep Program
3950 Chester Avenue
Cleveland, OH 44114-4625

Women/Children's Center
Metzenbaum Children's Center
3343 Community College Avenue
Cleveland, OH 44115

Saint John West Shore Hospital Area Healthcare System
2351 East 22nd Street
Cleveland, OH 44115

Southwest General Health Center Oakview Program
18697 Bagley Road
Cleveland, OH 44130

Stella Maris Washington Avenue Unit
1320 Washington Avenue
Cleveland, OH 44113

University MacDonald Women's Hospital Ann Nelson Perinatal Substance Abuse
11100 Euclid Avenue
Cleveland, OH 44106

Veterans' Addiction Recovery
Center Alcohol/Drug Dependence Treatment Unit
10701 East Boulevard
Cleveland, OH 44106

Women's Center of Greater Cleveland
6209 Storer Avenue
Cleveland, OH 44102

Y Haven/Its A New Day
3210 Franklin Boulevard
Cleveland, OH 44113

Y Haven II
6001 Woodland Avenue
Cleveland, OH 44104

CLEVELAND HEIGHTS

Center for Families and Children Rap Art Center
1941 South Taylor Road
Cleveland Heights, OH 44118

CLINTON

Barberton Rescue Mission
6694 Taylor Road
Clinton, OH 44216

COLUMBUS

Africentric Personal Development Shop ATOP/ Alcohol/Drug Outpatient Treatment
1409 Livingston Avenue, Suite 104
Columbus, OH 43205

Columbus Area Community Mental Health Center Alcohol and Drug Abuse Treatment
3035 West Broad Street
Columbus, OH 43204

Columbus Health Department
Alcoholism and Drug Abuse Programs
181 Washington Boulevard
Columbus, OH 43215

Community Counseling Centers
3025 West Broad Street
Columbus, OH 43204

COMPDRUG Corporation
Alvis House
Outpatient Services
700 Bryden Road
Columbus, OH 43215

Vita Treatment Center/Methadone Services
156 Parsons Avenue
3rd Floor
Columbus, OH 43215

Comprehensive Offender Program Effort DBA Ralph W Alvis House
1991 Bryden Road
Columbus, OH 43205

Crittenton Family Services Cedars Branch
1414 East Broad Street
Columbus, OH 43205

Department Youth Services Freedom Center
1414 East Broad Street
Columbus, OH 43205

Directions for Youth Alcohol and Drug Treatment Program
1515 Indianola Avenue
Columbus, OH 43201

Diversified Community Services Community Based Therapeutic Services
1651 East Main Street
Columbus, OH 43205

Franklin Pre-Release Center Residential Substance Abuse Program
1800 Harmon Avenue
Columbus, OH 43223

House of Hope for Alcoholics
177 West Hubbard Avenue
Columbus, OH 43215

Stevens House
1320 Parsons Avenue
Columbus, OH 43206

Maryhaven, Inc.
217 South Hamilton Road
Columbus, OH 43213

Mount Carmel Behavioral Healthcare
2238 South Hamilton Road
Columbus, OH 43232

NCC Associates North Central Mental Health
338 Granville Street
Columbus, OH 43230

Neighborhood House, Inc.
Alcohol/Drug Counseling Program
1000 Atcheson Street
Columbus, OH 43203

North Central Mental Health Services
Drug and Alcohol Treatment Program
1301 North High Street
Columbus, OH 43201

3035 West Broad Street
Columbus, OH 43204

Family Focus
40 Spruce Street
Columbus, OH 43215

Fowler House
422 East Lane Avenue
Columbus, OH 43201

Soaring Sober Day Options
595 East Rich Street
Columbus, OH 43215

North Community Counseling Centers, Inc.
The Bridge
4897 Karl Road
Columbus, OH 43229

1495 Morse Road, Suite B-3
Columbus, OH 43229

Northwest Counseling Services
1560 Fishinger Road
Columbus, OH 43221

Parenthesis Behavioral Healthcare Alcohol and Drug Outpatient Treatment
2242 South Hamilton Road
Suite 200
Columbus, OH 43232

Parkside Recovery Services
349 Olde Ridenour Road
Columbus, OH 43230

Project Linden
1500 East 17 Avenue
Columbus, OH 43219

Rosemont Center Marian Hall Dual Diagnosis Program
2440 Dawnlight Avenue
Columbus, OH 43211

Southeast, Inc.
217 South Hamilton Road
Columbus, OH 43213
Alcohol/Drug Outpatient
16 West Long Street
Columbus, OH 43215

1455 South 4th Street
Columbus, OH 43207

Substance Abuse Services, Inc.
3556 Sullivan Avenue, Room 106
Columbus, OH 43205

Syntaxis Youth Homes Joyce Group Home
2824 Joyce Avenue
Columbus, OH 43211

Talbot Hall at Park Medical Center
1492 East Broad Street
Columbus, OH 43205

Traumatic Brain Injury Network Alcohol/Drug Outpatient Treatment
1581 Dodd Drive
106 McCampbell Hall
Columbus, OH 43210-9110

United Behavioral Health Alcohol and Drug Outpatient Treatment
6096 East Main Street, Suite 110
Columbus, OH 43213

Wellness Group, Inc.
1660 NW Professional Plaza
Suite E
Columbus, OH 43220

COSHOCTON

Coshocton County
Drug and Alcohol Council, Inc.
140 1/2 South 6th Street
Coshocton, OH 43812

Coshocton Counseling Center
710 Main Street
Coshocton, OH 43812

CRESTLINE

Community Counseling Services Inc
224 North Seltzer Street
Crestline, OH 44827

MedCentral Crestline Hospital Freedom Hall
291 Heiser Court
Crestline, OH 44827

CUYAHOGA FALLS

Family solutions
Alcohol and Drug Outpatient
2100 Front Street
Cuyahoga Falls, OH 44221

DAYTON

Alvis House Alcohol and Drug Outpatient Treatment
42 Arnold Place
Dayton, OH 45407

Born Free
Miami Valley Hospital Turning Point
Dayton, OH 45409

Combined Health District Center for Alcohol and Drug Addiction Services
4100 West 3rd Street
VA Medical Center Building 410
3rd Floor
Dayton, OH 45428

600 Wayne Avenue Oregon Plaza
Dayton, OH 45410-1122

Day Mont Behavioral Health Care Substance Abuse Services
1520 Germantown Street
Dayton, OH 45408

Dayton Correctional Institute Project Rebound
4104 Germantown Street
Dayton, OH 45417

Diversion Alternatives for Youth
330 South Ludlow Street
Dayton, OH 45402

Eastway Behavioral Healthcare
600 Wayne Avenue
Dayton, OH 45410

Eastway Corporation Pathways Residential Program
4950 Northcutt Place
Dayton, OH 45414

Franciscan Stress Care Center Alcohol and Drug Treatment
One Franciscan Way
Dayton, OH 45408

Grandview Hospital Careview Chemical Dependency Program
405 Grand Avenue
Dayton, OH 45405-4796

Monday Community Correctional Institution Alcohol/Drug Outpatient Treatment
1951 South Gettysburg Avenue
Dayton, OH 45418-2313

Nova House Association Treatment Program
732 Beckman Street
Dayton, OH 45410

Project Cure, Inc.
1800 North James H. McGee Boulevard
Dayton, OH 45427

South Community, Inc. Alcohol/Drug Treatment Program
238 Yuma Court
Dayton, OH 45458

Wright State University School of Medicine RRTC on Drugs and Disability
Dayton, OH 45438

DEFIANCE

Community Counseling of Northwest Ohio
1103 Holgate Avenue
Defiance, OH 43512

Five County Alcohol/Drug Program
418 Auglaize Street
Defiance, OH 43512

DELAWARE

Delaware Area Recovery Resources, Inc.
540 U.S. Route 36 East
Delaware, OH 43015

Ohio Department of Youth Services Scioto Juvenile Correctional Center
5993 Home Road
Delaware, OH 43015

DUBLIN

Dublin Counseling Center
6077 Frantz Road
Suite 103
Dublin, OH 43017

EASTLAKE

North Coast Student Assistance Corp. Alcohol and Drug Outpatient Treatment
34050 Glen Drive
Eastlake, OH 44095

EATON

Preble County Recovery Center, Inc.
100 East Somers Street
Eaton, OH 45320

ELYRIA

Lorain County Alcohol and Drug Abuse Services
215 Court Street
Elyria, OH 44035

230 4 Street
Elyria, OH 44035

FAIRBORN

Community Network, Inc.
919 South Central Street
Fairborn, OH 45324

FOSTORIA

Firelands Community Hospital Counseling and Recovery
301 South Main Street
Fostoria, OH 44830

Fostoria Alcohol/Drug Center
114 West North Street
Fostoria, OH 44830

FREMONT

Firelands Community Hospital Counseling and Recovery
675 Bartson Road
Fremont, OH 43420

GALION

Community Counseling Services, Inc. Galion Office
269 Portland Way South
Galion, OH 44833

GEORGETOWN

Brown County Counseling
75 Banting Drive
Georgetown, OH 45121

GRAFTON

Lorain Correctional Institution Substance Abuse Services
2075 South Avon/Beldon Road
Grafton, OH 44044

GREENVILLE

Darke County Recovery Services
134 West 4 Street
Greenville, OH 45331

GROVE CITY

Wellness Group, Inc. Learn Driver Intervention Program
Ramada Inn South
1879 Stringtown Road
Grove City, OH 43123

HAMILTON

Alcohol and Chemical Abuse Council of Butler County Ohio, Inc.
111 Buckeye Street
Hamilton, OH 45011

Butler County Mental Health Center Harbor House
140 Buckeye Street
Hamilton, OH 45011

Fort Hamilton Hughes Memorial Hospital Center Horizon Services
630 Eaton Avenue
Hamilton, OH 45013

Sojourner Home
449 North 3 Street
Hamilton, OH 45011

Herland Family Center
520 High Street
Hamilton, OH 45011

Intensive Outpatient Program
625 High Street
Hamilton, OH 45011

Southwestern Ohio Serenity Hall, Inc.
439 South 2 Street
Hamilton, OH 45011

24 North 7th Street
Hamilton, OH 45011

Transitional Living Drug/ Alcohol Addiction Disorder Program
117 Park Avenue
Hamilton, OH 45013

HILLSBORO

Family Recovery Services for Alcohol and Drug Abuse, Inc.
Driver Intervention Program
972 West Main Street
Hillsboro, OH 45133

Scioto Paint Valley Mental Health Center Highland County Office
108 Erin Court
Hillsboro, OH 45133

HOLLAND

Comprehensive Addiction Service System (COMPASS)
1150 South McCord Street, Suite 101
Holland, OH 43528

HUDSON

Youth Development Center Genesis Program
996 Hines Hill Road
Hudson, OH 44236

INDEPENDENCE

Marycrest
7800 Brookside Road
Independence, OH 44131

Saint Vincent Charity Hospital
Rosary Hall
6701 Rockside Road
Independence, OH 44131

IRONTON

River Valley Health System
2228 South 9th Street
Ironton, OH 45638

KENTON

Tri-Star Community Counseling
718 East Franklin Street
Kenton, OH 43326

LAKEWOOD

River Valley Health System
2228 South 9th Street
Ironton, OH 45638

Alcoholism Services of Cleveland
14805 Detroit Avenue, Suite 320
Lakewood, OH 44107

LANCASTER

Center for Families and Children Mental Health Counseling
14701 Detroit Avenue, Suite 620
Lakewood, OH 44107

LEBANON

Center of Warren/Clinton Counties
107 Oregonia Road
Lebanon, OH 45036

Warren Detention Center
550 Justice Drive
Lebanon, OH 45036

Talbert House Community Correctional Center
5234 State Route 63
Lebanon, OH 45036

Warren Correctional Institution Recovery Services Department
State Route 63
Lebanon, OH 45036

LIBERTY CENTER

Maumee Youth Center
RFD 2
Liberty Center, OH 43532

LIMA

Northwest Family Services
DBA Family Resource Centers Project Inroads
799 South Main Street
Lima, OH 45804

Saint Rita's Medical Center Addiction Services
730 West Market Street
Lima, OH 45801

Tri-Star Community Counseling, Inc. Recovery Services
530 South Main Street
Lima, OH 45804

LISBON

Columbiana County Mental Health Center Substance Abuse Program
40722 State Route 154
Lisbon, OH 44432

Family Recovery Center
Outpatient Program
964 North Market Street
Lisbon, OH 44432

Vista Centre Outpatient Treatment
100 Vista Drive
Lisbon, OH 44432

LOGAN

Health Recovery Services, Inc. Hocking County Outpatient Clinic
4 East Hunter Street
Logan, OH 43138

LONDON

London Correctional Institution Recovery Services
State Route 56
London, OH 43140

Madison Correctional Institute
1851 State Route 56
London, OH 43140

Madison County Alcohol and Drug Services
210 North Main Street
London, OH 43140

LORAIN

Compass House
2130 East 36 Street
Lorain, OH 44055

1440 Lexington Avenue
Lorain, OH 44052

Lorain County Alcohol and Drug Abuse Services
225 West 6th Street
Lorain, OH 44052

625 Reid Avenue
Lorain, OH 44052

Recovery Resources Juvenile Offenders/Pairs Program
203 West 8th Street
Lorain, OH 44053

LOUDONVILLE

Mohican Youth Center Substance Abuse Program
741 West Main Street, Suite 1
Loudonville, OH 44842

LOUISVILLE

Stark Regional Community Correction Center Alcohol and Drug Outpatient Treatment
4433 Lesh Street NE
Louisville, OH 44641

LUCASVILLE

Southern Ohio Correctional Facility Substance Abuse Program
Lucasville-Minford Road
Lucasville, OH 45699

MANSFIELD

Center for Individual and Family Services Drug Abuse Program
741 Scholl Road
Mansfield, OH 44907

Mansfield Correctional Institution Innervisions
1150 North Main Street
Mansfield, OH 44901

Mansfield Urban Minority Alcoholism and Drug Abuse Outreach Program
400 Bowman Street
Mansfield, OH 44901

Richland Hospital
Serenity Hall
Substance Abuse Services
1451 Lucas Road
Mansfield, OH 44901

Volunteers of America Central Ohio, Inc.
290 North Main Street
Mansfield, OH 44901

MAPLE HEIGHTS

Center for Families and Children Cleveland CARES/ Southgate
5398 Northfield Road
Maple Heights, OH 44137

MARIETTA

Marietta College
210 Thomas Hall
Marietta, OH 45750

Marietta Memorial Hospital Substance Abuse Services
401 Matthew Street
Marietta, OH 45750

MARION

Marion Area Counseling Center
Alcohol and Drug Program
320 Executive Drive
Marion, OH 43302

Crossroads Recovery
286 Patterson Street
Marion, OH 43302

Professional Treatment Systems
310 Executive Drive
Marion, OH 43302

MARTINS FERRY

East Ohio Regional Hospital Touchstones Treatment Center
90 North 4th Street
Martins Ferry, OH 43935

MARYSVILLE

Charles B. Mills Center
715 South Plum Street
Marysville, OH 43040

COMPDRUG Corporation Tapestry/TC Program
1479 Collins Avenue
Ohio Reformatory for Women
Marysville, OH 43040

MASON

Center of Warren/Clinton Counties
201 Reading Road
Mason, OH 45040

MASSILLON

Longford Health Sources of Massillon Community Hospital
875 8th Street NE
Massillon, OH 44648

Massillon Division of Quest Recovery Services
325 3rd Street SE
Massillon, OH 44646

Nova Behavioral Health
39 Tremont Avenue SW
Massillon, OH 44646

MAUMEE

Professional Systems Addiction Treatment Service
1627 Hen Thorn Drive
Maumee, OH 43537

MCCONNELSVILLE

Morgan Behavioral Health Choices Morgan Drug and Alcohol Council
915 South Riverside Drive
Morgan County Prep Center
McConnelsville, OH 43756

MEDINA

Alcohol and Drug Dependency Services of Medina County, Inc.
246 Northland Drive
Suite 140
Medina, OH 44256

MENTOR

Crossroads Counseling Services, Inc. Adolescent Counseling Service
8445 Munson Road
Mentor, OH 44060

Lake Geauga Center on Alcohol and Drug Abuse, Inc.
8827 Mentor Avenue
Mentor, OH 44060

Laurelwood Counseling Center of Mentor
7060 Wayside Drive
Mentor, OH 44060

MIDDLEPORT

Health Recovery Services, Inc. Meigs County Clinic
138 North 2nd Avenue
Middleport, OH 45760

MIDDLETOWN

Comprehensive Counseling Service Intensive Outpatient Treatment
1659 South Breiel Boulevard
Middletown, OH 45044

Fort Hamilton Hospital Horizon Services
829 Elliott Drive
Middletown, OH 45042

MILFORD

Cincinnati Teen Challenge, Inc.
1466 Route 60
Milford, OH 45150

Kids Helping Kids
6070 Branch Hill Guinea Pike
Milford, OH 45150

MILLERSBURG

Human Resource Center
186 West Jackson Street
Millersburg, OH 44654

MINGO JUNCTION

Jefferson Behavioral Health System Care Network/ Residential Facility
202 Township Road
Route 164
Mingo Junction, OH 43938

MOUNT GILEAD

Morrow County Council on Alcohol and Drugs, Inc.
950 Meadow Drive
Mount Gilead, OH 43338

Drugs/Jail Outpatient Treatment Program
Morrow County Jail
State Route 42
Mount Gilead, OH 43338

MOUNT ORAB

Brown County Counseling Service Alcohol/Drug Program
13679 State Route 68
Mount Orab, OH 45154

MOUNT VERNON

Alcohol and Drug Freedom Center of Knox County
106 East Gambier Street
Mount Vernon, OH 43050

NAPOLEON

Five County Alcohol/Drug Program
444 Independence Drive
Suite 110
Napoleon, OH 43545

Henry County Hospital Help Center
11-600 State Road 424
Napoleon, OH 43545

NELSONVILLE

Hocking Correctional Facility Substance Abuse Department
16759 Snake Hollow Road
Nelsonville, OH 45764

Septa Correctional Facility Alcohol and Drug Outpatient Clinic
7 West 29 Drive
Nelsonville, OH 45764

NEWARK

Licking County Alcoholism Prevention Program
Outpatient Services
62 East Stevens Street
Newark, OH 43055

Shepherd Hill Hospital Substance Abuse Services
200 Messimer Drive
Newark, OH 43055

Spencer Halfway House, Inc.
69 Granville Street
Newark, OH 43055

NEW LEXINGTON

Perry County Alcohol and Drug Abuse Council, Inc.
227 North Main Street
New Lexington, OH 43764

NEW PHILADELPHIA

Harbor House, Inc.
Shelter House
New Philadelphia, OH 44663

Outpatient for Women
349 East High Street
New Philadelphia, OH 44663

NILES

Glenbeigh Center of Niles Alcohol/Drug Outpatient Treatment
29 North Road SE
Niles, OH 44446

NORTH CANTON

Walsh University Counselor in Residence
2020 Easton Street NW
North Canton, OH 44720

NORWALK

Firelands Community Hospital Counseling and Recovery Services
292 Benedict Avenue
Norwalk, OH 44857

OAK HARBOR

Giving Tree, Inc. Mental Health and Drug Addiction Services
11969 WSR 105
Oak Harbor, OH 43449

ORIENT

Correctional Reception Center
11271 State Route 762
Orient, OH 43146

Pickaway Correctional Institution
Oasis Therapeutic Community Prison Project
11781 State Route 762
Orient, OH 43146

ORRVILLE

Education and Counseling Services, Inc.
Wayne County Residential Program
1022 West High Street
Orrville, OH 44667

Wayne County Alcoholism Services
1710 West Paradise Road
Orrville, OH 44667

OTTAWA

Pathways Counseling Center
117 Court Street
Ottawa, OH 45875

PAINESVILLE

Catholic Services of Lake County
8 North State Street Room 455
Painesville, OH 44077

Lake Geauga Center on Alcohol/Drug Abuse, Inc.
Lake House
42 East Jackson Street
Painesville, OH 44077

Oak House
796 Oak Street
Painesville, OH 44077

PARMA

Center for Families and Children Southwest Alcohol/Drug Counseling
5955 Ridge Road
Parma, OH 44129

PAULDING

Paulding County Alcohol and Drug Services Council, Inc.
501 McDonald Pike
Paulding, OH 45879

PERRYSBURG

Behavioral Connections of Wood County, Inc.
27072 Carronade Street
Suite A and B
Perrysburg, OH 43551

PICKERINGTON

Fairfield County Drug/Alcohol Recovery Center Pickerington Office
437 Hill Road North
Pickerington Professional Park
Pickerington, OH 43147

PIQUA

Miami County Alcoholism Program
Outpatient Services
423 North Wayne Street
Piqua, OH 45356

PORT CLINTON

Bayshore Counseling Services, Inc. Ottawa County Outpatient Office
201 West Madison Street
Port Clinton, OH 43452

Giving Tree, Inc. Dual Diagnosis Alcohol/Drug Treatment
335 Buckeye Boulevard
Port Clinton, OH 43452

PORTSMOUTH

James T. Marsh Male Halfway House
1216 4th Street
Portsmouth, OH 45662

River Valley Health System Behavioral Health Services
2201 25th Street
Behavioral Health Services Campus
Portsmouth, OH 45662-3252

Stepping Stone House
1409 2nd Street
Portsmouth, OH 45662

PROCTORVILLE

Family Guidance Center
209 State Street
Proctorville, OH 45669

RAVENNA

Townhall II
Serenity Halfway House
151 East Spruce Avenue
Ravenna, OH 44266

Alcohol and Drug Outpatient Treatment
223 West Main Street
Ravenna, OH 44266

Horizon Halfway House
147 East Spruce Avenue
Ravenna, OH 44266

REYNOLDSBURG

NCC Reynoldsburg North Central Mental Health
6432 East Main Street
Reynoldsburg, OH 43068

RIPLEY

Brown County Counseling Services Alcohol/Drug Program
Early Childhood Resource Center
500 South Second Street
Ripley, OH 45167

RITTMAN

Your Human Resource Center
51 North Main Street
Rittman, OH 44270

ROCK CREEK

Glenbeigh Health Sources
2863 State Route 45
Rock Creek, OH 44084

ROCKY RIVER

Glenbeigh Center of Rocky River Alcohol Drug Outpatient Treatment
20800 Center Ridge Road
Suite 202
Rocky River, OH 44116

SAINT CLAIRSVILLE

Crossroads Counseling Service
255 West Main Street
Saint Clairsville, OH 43950

SANDUSKY

Bayshore Counseling Services, Inc. Erie County Outpatient Office
1218 Cleveland Road, Suite B
Sandusky, OH 44870-4787

Firelands Community Hospital Firelands Center
2020 Hayes Avenue
Sandusky, OH 44870

Providence Hospital Alcohol and Drug Detox and Outpatient Treatment
1912 Hayes Avenue
Sandusky, OH 44870

SHELBY

Cornell Abraxas Group
2775 State Route 39
Shelby, OH 44875

SIDNEY

Shelby County Counseling Center Alcohol and Drug Outpatient Treatment
500 East Court Street
Sidney, OH 45365

SMITHVILLE

Boys Village, Inc. Alcohol/Drug Treatment Program
2803 State Route 585
Smithville, OH 44677

SOLON

Center for Families and Children Reach Out Program
33995 Bainbridge Road
Solon, OH 44139

SOUTH POINT

Family Guidance Center
305 North 4th Street
South Point, OH 45680

Ironton/Lawrence County CAO River Valley Driver Intervention Program
103 East 4th Street
South Point, OH 45680

SPRINGBORO

Center of Warren/Clinton Counties Greenwood Center
50 Greenwood Lane
Springboro, OH 45066

SPRINGFIELD

Alcohol/Drug Abuse Programs For Treatment (ADAPT)
825 East High Street
Springfield, OH 45501

Matt Talbot House
809 South Limestone Street
Springfield, OH 45505

McKinley Hall, Inc.
Inpatient Program
225 East Street
Springfield, OH 44505

Outpatient Program
1101 East High Street
Springfield, OH 45505

Mental Health Services for Clark County
Outpatient Adolescent Recovery
1835 Miracle Mile
Springfield, OH 45504

Outpatient Adult Recovery/Jail
120 North Fountain Boulevard
Springfield, OH 45501

Mercy Memorial Hospital Mercy Reach Substance Abuse Program
1343 North Fountain Boulevard
Springfield, OH 45501-1380

Youth Challenges
CAF Street
Springfield, OH 45504

STEUBENVILLE

Jefferson Behavioral Health System Drug and Alcohol Outpatient Treatment
200 North 4th Street
Steubenville, OH 43952

Trinity Medical Center West Addiction Recovery Program
4000 Johnson Road
Steubenville, OH 43952

TIFFIN

Firelands Community Hospital Firelands Counseling and Recovery
181 East Perry Street
Tiffin, OH 44883

TOLEDO

Adelante
Los Ninos Substance Abuse Prevention
520 Broadway
Toledo, OH 43602

Boysville of Michigan, Inc. Saint Anthony Villa
2740 West Central Avenue
Andre Hall
Toledo, OH 43606

Comprehensive Addiction Services Systems (COMPASS)
3001 Hill Avenue
Toledo, OH 43607

Fresh Attitude, Inc. Alcohol and Drug Halfway House
3211 Mayo Street
Toledo, OH 43620

3212 Chase Street
Toledo, OH 43611

Outpatient Treatment
2700 Monroe Street, Suite K
Toledo, OH 43606

Rescue Mental Health Services
3350 Collingwood Boulevard
Toledo, OH 43610

Saint Charles Hospital
Westgate Outpatient Behavioral Services
3140 West Central Avenue
Toledo, OH 43606

Saint Paul's Community Center Intervention Program
230 13 Street
Toledo, OH 43624

Substance Abuse Services, Inc.
701 Adams Street
Toledo, OH 43624

Outpatient Services
1832 Adams Street
Toledo, OH 43624

Talbot Outpatient Center
732 South Main Street
Toledo, OH 43605

Toledo Hospital Alcohol and Drug Treatment Center
2142 North Cove Boulevard
Toledo, OH 43606

Unison Behavioral Health Group Dual Recovery Program
1425 Starr Avenue
Toledo, OH 43605

TROY

Dettmer Recovery Services
3130 North Dixie Drive
Troy, OH 45373

Miami County Mental Health Center Choices/Troy Satellite
1059 North Market Street
Troy, OH 45373

UPPER SANDUSKY

Firelands Community Hospital Firelands Counseling and Recovery
132 East Wyandot Avenue
Upper Sandusky, OH 43351

URBANA

Logan/Champaign Alcohol and Drug Addiction Services
40 Monument Square
Suite 301
Urbana, OH 43078

Mercy Memorial Hospital Mercy Substance Abuse Program
904 Scioto Street
Urbana, OH 43078

VAN WERT

Fountainview Center
120 West Main Street
2nd Floor
Van Wert, OH 45891

WADSWORTH

Alcohol and Drug Dependency Services of Medina County/ Wadsworth Office
180 High Street
Wadsworth, OH 44281

WAPAKONETA

Tri-Star Community Counseling
15 Willipie Street
Wapakoneta, OH 45895

WARREN

Saint Joseph Health Center
667 Eastland Avenue
Warren, OH 44484

Saint Joseph Riverside Hospital New Start Treatment Center
1370 Tod Avenue NW
Warren, OH 44485

Two North Park, Inc.
720 Pine Avenue SE
Warren, OH 44481

York Avenue Church of God Treatment Center Alcohol/ Drug Outpatient Treatment
872 York Avenue
Warren, OH 44485

WASHINGTON COURT HOUSE

Scioto Paint Valley Mental Health Center Fayette County Office
1300 East Paint Street
Washington Court House, OH 43160

WAUSEON

Five County Alcohol/Drug Program
125 North Fulton Street
Wauseon, OH 43567

Fulton County Health Center
725 South Shoop Avenue
Wauseon, OH 43567

Fulton Stress Unit Fulcare Daytox Alcohol and Drug Treatment
725 South Shoop Avenue
Wauseon, OH 43567

WAVERLY

Pike County Recovery Outpatient Services
111 North High Street
Waverly, OH 45690

Scioto Paint Valley Mental Health Center Pike County Office
102 Dawn Lane
Waverly, OH 45690

WESTERVILLE

Concord Counseling Services, Inc.
924 Eastwind Drive
Westerville, OH 43081

WESTLAKE

Saint John West Shore Hospital Serenity Center
29000 Center Ridge Road
Westlake, OH 44145

WEST LIBERTY

L/C Consolidated Care, Inc.
1521 North Detroit Street
West Liberty, OH 43357

WILLARD

Firelands Community Hospital Firelands Counseling and Recovery
302 Woodland Avenue
Willard, OH 44890

WILLOUGHBY

Laurelwood Hospital Addictive Disease Unit
35900 Euclid Avenue
Willoughby, OH 44094

WILMINGTON

Center of Warren/Clinton Counties
Alcohol and Drug Treatment Programs/Hopewell BHCS
610 West Main Street
Floor 2 East
Wilmington, OH 45177

Wilmington Center
1216 West Locust Street
Wilmington, OH 45177

WOODSFIELD

Crossroads Counseling Services
37984 Airport Road
Woodsfield, OH 43793

WOOSTER

College of Wooster Alcohol/ Drug Prevention Project
Wooster, OH 44691

Human Resource Center
2692 Akron Road
Wooster, OH 44691

Wayne County Alcoholism Services
Beacon House
732 Spink Street
Wooster, OH 44691

Pathway House
550 North Grant Street
Wooster, OH 44691

WORTHINGTON

Focus Health Care
5701 North High Street Suite 8
Worthington, OH 43085

Harding Hospital Adult and Adolescent Services
445 East Dublin Granville Road
Worthington, OH 43085

XENIA

Stepping In Recovery (SIR) Alcohol and Drug Outpatient Treatment
39 Greene Street
Xenia, OH 45385

TCN Behavioral Health
476 West Market Street
Xenia, OH 45385

Greene County Jail Outpatient Treatment
77 East Market Street
Xenia, OH 45385

Women's Recovery Center
515 Martin Drive
Xenia, OH 45385

YOUNGSTOWN

Addiction Programs of Mahoning County, Inc.
Donofrio Alcoholism Rehabilitation Center
1161 McGuffey Road
Youngstown, OH 44505

Donofrio Womens Center
64 Ridge Street
Youngstown, OH 44507

Alcoholism Programs of Mahoning County
Alma L Field 3/4 House
145 Illinois Avenue
Youngstown, OH 44505

Bodnar 3/4 Way Home
2516 Market Street
Youngstown, OH 44507

Community Corrections Association, Inc.
Community Corrections Facility
1740 Market Street
Youngstown, OH 44507

Residential Treatment Center I
1764 Market Street
Youngstown, OH 44507

Residential Treatment Center II
1620 Market Street
Youngstown, OH 44507

Neil Kennedy Recovery Clinic
2151 Rush Boulevard
Youngstown, OH 44507-1598

Northside Medical Center Adolescent Recovery Services
500 Gypsy Lane
Youngstown, OH 44501

Parkside Behavioral Healthcare Parkside Counseling Services
7536 Market Street
Youngstown, OH 44501-0240

Chemical Abuse Center, Inc.
5211 Mahoning Avenue, Suite 110
Youngstown, OH 44515

ZANESVILLE

Genesis Recovery Program
716 Adair Avenue
Zanesville, OH 43701

Good Samaritan Medical Center Alcoholism and Drug Recovery Treatment Program
716 Adair Avenue
Zanesville, OH 43701

Muskingum Behavioral Health
575 Harding Road
Zanesville, OH 43701

OKLAHOMA

ADA

Ada Area Chemical Dependency Center
727 Arlington Street
Ada, OK 74820

Rolling Hills Hospital Substance Abuse Services
1000 Rolling Hills Lane
Ada, OK 74820

ALTUS

New Hope Halfway House of Division of New Hope of Mangum
710 East Southerland Street
Altus, OK 73521

ALTUS AIR FORCE BASE

Altus Air Force Base Substance Abuse Program
97 MDOS/SGOMH
301 North First Street
Altus AFB, OK 73523-5005

U.S. Air Force Hospital Altus
Altus Air Force Base
Altus AFB, OK 73523

ALVA

Freedom Ranch, Inc. CBTI
Route 1, Box 48
Alva, OK 73717

ANADARKO

Consortium Against Substance Abuse
115 East Broadway
Anadarko, OK 73005

ANTLERS

Oaks Behavioral Center
414 West Main Street
Antlers, OK 74523-2661

ARCADIA

Drug Recovery Adolescent Program
505 North Broadway
Arcadia, OK 73007

ARDMORE

Arbuckle Drug and Alcohol Information Center, Inc.
1219 K Street NW
Ardmore, OK 73401

Broadway House, Inc.
214 North Washington Street
Ardmore, OK 73403

Mental Health Services of Southern Oklahoma Vantage Pointe
2530 South Commerce Street
Building C
Ardmore, OK 73401

ATOKA

Oaks Behavioral Center
211 East Court Street
Atoka, OK 74525-2000

BARTLESVILLE

Alcohol and Drug Center, Inc.
615 SE Frank Phillips Boulevard
Bartlesville, OK 74003

BROKEN ARROW

Healthcare Management Alliance, Inc. DBA Recovery Plus
817 South Elm Place, Suite 105
Broken Arrow, OK 74012

CHANDLER

Gateway to Prevention Recovery
102 East 7th Street
Chandler, OK 74834-2820

CHICKASHA

Southwest Youth and Family Services, Inc.
198 East Almar Drive
Chickasha, OK 73023

CHOCTAW

Tri-City Youth and Family Center, Inc.
14625 NE 23 Street
Choctaw, OK 73020

CLAREMORE

Rogers County Drug Abuse Program, Inc.
118 West North Seminole Street
Claremore, OK 74018

CLINTON

Opportunities, Inc. Rehabilitation Center Behavioral Care Services
720 South 8th Street
Clinton, OK 73601

COALGATE

Oaks Behavioral Health Center
2 South Main Street
Coalgate, OK 74538-2829

CONCHO

Cheyenne/Arapaho Substance Abuse
700 North Black Kettle Drive
Concho, OK 73022

CUSHING

Valley Hope Alcoholism Treatment Center
100 South Jones Avenue
Cushing, OK 74023

DURANT

Kiamichi Council Alcoholism
307 West Elm Street, Suite 2
Durant, OK 74701-4109

EDMOND

Edmond Family Services, Inc. Outpatient Drug/Alcohol Services
7 North Broadway, Suite E
Edmond, OK 73034-1085

EL RENO

Chisholm Trail Counseling Services Substance Abuse Services
200 North Choctaw Street
Suite 110
El Reno, OK 73036

ENID

Wheatland Mental Health Center, Inc.
702 North Grand Street
Enid, OK 73701

EUFAULA

Oaks Behavioral Health Center
119 McKinley Street
Eufaula, OK 74432-2853

FORT SILL

Alcohol and Drug Abuse Prevention and Control Program (ADAPCP)
2870-B Craig Road
Fort Sill, OK 73503-5100

FORT SUPPLY

Western State Psychiatric Center
Highway 270 East
Fort Supply, OK 73841

GROVE

House of Hope, Inc.
East 32 South 625 Road
Grove, OK 74344

GUTHRIE

Eagle Ridge Family Treatment Center
1916 East Perkins Street
Guthrie, OK 73044

Logan County Youth and Family Service
4710 South Division Street
Guthrie, OK 73044

Wheatland Mental Health Center
1923 South Division Street
Guthrie, OK 73044

GUYMON

Next Step Network
1004 Highway 54 NE
Guymon, OK 73942

HOMINY

Hominy Health Services, Inc.
211 East 5th Street
Hominy, OK 74035

HUGO

Kiamichi Council on Alcoholism and Other Drug Abuse, Inc.
308 East Jefferson Street
Hugo, OK 74743

IDABEL

Kiamichi Council on Alcoholism and Other Drug Abuse, Inc.
104 North East Avenue A
Idabel, OK 74745

People Plus, Inc.
103 NE Avenue A
Idabel, OK 74745

KINGFISHER

Wheatland Mental Health Center
124 East Sheridan Street Suite 200
Kingfisher, OK 73750

LAWTON

Comanche County Memorial Hospital Memorial Pavilion
3401 West Gore Boulevard
Lawton, OK 73505

1602 SW 82nd Street
Lawton, OK 73505

Jim Taliaferro CMHC
602 SW 38 Street
Lawton, OK 73505

New Pathways Halfway House
1401 NE Laurie Tatum Road
Lawton, OK 73502

Roadback, Inc.
1502 D Street SW
Suite 4
Lawton, OK 73501

LONE WOLF

Southwestern Oklahoma Adolescent Addiction Rehab Ranch, Inc. (SOAARR)
Route 1, Box 69
Lone Wolf, OK 73655

MANGUM

New Hope of Mangum Chemical Dependency Unit
2 Wickersham Drive
Mangum, OK 73554

MARIETTA

Morning Star Adolescent Treatment Unit
Route 1, Box 14
Marietta, OK 73448

MCALESTER

Brown Schools of Oklahoma
1401 East Cherokee Avenue
McAlester, OK 74501-5635

Carl Albert Community Mental Health Center
1101 East Monroe Street
McAlester, OK 74502

The Oaks Rehabilitative Services Center
628 East Creek Street
McAlester, OK 74501

MCLOUD

Kickapoo Alcohol and Substance Abuse Program
State Highway 102
McLoud, OK 74851

MIAMI

Inter Tribal Substance Abuse/ Prevention and Treatment Center
101 South Main Street
Miami, OK 74354

Northeastern Oklahoma Council on Alcoholism
316 Eastgate Boulevard
Miami, OK 74355

MUSKOGEE

Green Country Behavioral Health Services, Inc. Alcohol and Drug Abuse Services
619 North Main Street
Muskogee, OK 74401

Monarch Incorporated
501 Fredonia Street
Muskogee, OK 74403

Muskogee County Council of Youth Services
4409 Eufaula Avenue
Muskogee, OK 74401

Recovery Plus
1805 North York Street, Suite G
Muskogee, OK 74403-1442

NORMAN

Central Oklahoma CMHC
909 East Alameda Street
Norman, OK 73071

NAIC/Center for Oklahoma Alcohol and Drug Services, Inc.
215 West Linn Street
Norman, OK 73069

Norman Alcohol and Drug Treatment Center
East Main Street and State Drive
Norman, OK 73071

Norman Regional Hospital Behavioral Medicine Services
708 24th Avenue NW
Norman, OK 73069

NOWATA

Grand Lake Mental Health Center, Inc. Alcohol and Drug Abuse Services
114 West Delaware Street
Nowata, OK 74048

Recovery Way, Inc. Inpatient Program
237 South Locust Street
Nowata, OK 74048

OKEMAH

Gateway to Prevention Recovery
119 South 1st Street
Okemah, OK 74859

OKLAHOMA CITY

A Chance to Change Foundation
5228 Classen Boulevard
Oklahoma City, OK 73118

Alcohol Training and Education, Inc.
2800 NW 36 Street
Suite 101
Oklahoma City, OK 73112

Carver Correction Center
2801 SW 3 Street
Oklahoma City, OK 73108

Community Counseling Center
1140 North Hudson Street
Oklahoma City, OK 73103

Community House
1501 NE 11th Street
Oklahoma City, OK 73117

Cope, Inc.
3033 North Walnut Street
Suite 200-W
Oklahoma City, OK 73105

Deaconess Hospital
5501 North Portland Avenue
Oklahoma City, OK 73112-2099

Drug Recovery, Inc.
415 NW 7 Street
Oklahoma City, OK 73102

Ivanhoe Facility
415 NW 8th Street
Oklahoma City, OK 73102-2603

Outpatient
425 NW 7th Street
Oklahoma City, OK 73101-1256

Integris Mental Health Integris Recovery Network
3300 NW Expressway
Oklahoma City, OK 73112

Mercy Health Center Outpatient Alcohol Treatment Program
4300 West Memorial Road
Oklahoma City, OK 73120

Moore Alcohol/Drug Center, Inc.
624 NW 5th Street
Oklahoma City, OK 73160

New Direction Centers of America
3115 North Lincoln Boulevard
Oklahoma City, OK 73105

North Care Center Substance Abuse Services
6300 North Classen Boulevard
Building A
Oklahoma City, OK 73118

Oklahoma County Crisis Intervention Center
1200 NE 13th Street
Oklahoma City, OK 73117

Oklahoma Halfway House, Inc.
517 SW 2 Street
Oklahoma City, OK 73109

Orange Quarters, Inc. DBA The Life Improvement Center
1017 10th Street NW
Oklahoma City, OK 73107

Phoenix House
824 East Drive
Oklahoma City, OK 73105

Red Rock Behavioral Health Services Substance Abuse Services
4400 North Lincoln Boulevard
Oklahoma City, OK 73105

Referral Center for Alcohol and Drug Service of Central Oklahoma
1215 NW 25th Street
Oklahoma City, OK 73106

Saint Anthony Hospital Recovery and Treatment (START)
1000 North Lee Street
Oklahoma City, OK 73101

Veterans' Affairs Medical Center Substance Abuse Treatment
921 NE 13 Street
116C
Oklahoma City, OK 73104

Total Life Counseling TLC Foundation
5900 Mostellar Drive, Suite 333
Oklahoma City, OK 73112

Turning Point South
1607 SW 15th Street
Oklahoma City, OK 73108

Valley Hope Alcoholism and Drug Center of Oklahoma City
5010 North Drexel Boulevard
Oklahoma City, OK 73112

OKMULGEE

Behavioral Health Services of the Creek Nation
410 West 6th Street
Okmulgee, OK 74447

PAWHUSKA

Osage Nation Counseling Center Substance Abuse Program
518 Leahy Street
Pawhuska, OK 74056

PAWNEE

Community Alcoholism Services
600 Denver Street
Pawnee, OK 74058

PONCA CITY

Bridgeway, Inc.
620 West Grand Street
Ponca City, OK 74602

Edwin Fair Mental Health Center Alcohol and Drug Abuse Unit
1500 North 6 Street
Ponca City, OK 74601

Native American Women's Alcohol Rehabilitation Center
5856 South Highway 177
Ponca City, OK 74601

Social Development Center
Route 1, Box 1595
Ponca City, OK 74601

PRYOR

The Brown Schools at Shadow Mountain
5 South Vann
Pryor, OK 74361

RED ROCK

Otoe/Missouria Tribe Substance Abuse Program
Route 1
Red Rock, OK 74651

SAPULA

Freedom Ranch CBTI
14 South Water Street
Sapulpa, OK 74066

SEMINOLE

Tri-City Substance Abuse Center
214 East Oak Street
Seminole, OK 74868

SHAWNEE

Absentee Shawnee Tribe Substance Abuse Program
2025 South Gordon Cooper Drive
Shawnee, OK 74801

Gateway to Prevention and Recovery
1010 East 45 Street
Shawnee, OK 74802

Native American Center of Recovery, Inc.
420 North Kickapoo Street
Shawnee, OK 74802

STILLWATER

CBTI Drug Court Program
217 West 5th Avenue, Suite 7
Stillwater, OK 74074-4005

Payne County Counseling Services
801 South Main Street
Suite 5
Stillwater, OK 74074

Payne County Youth Services, Inc.
2224 West 12 Street
Stillwater, OK 74074

Recovery Plus
2324 North Perkins Road
Stillwater, OK 74075

Starting Point II, Inc.
608 Highpoint Drive
Stillwater, OK 74075

TAHLEQUAH

Jack Brown Regional Treatment Center
Tahlequah, OK 74465

Jim Taliaferro Community Mental Health and Substance Abuse Centers
1200 West 4th Street
Tahlequah, OK 74465

TALIHINA

Chi Hullo Li Choctaw Nation of Oklahoma
Route 2 Box 1774
Talihina, OK 74571

Choctaw Nation Recovery Center
Route 2
Talihina, OK 74571

TINKER AFB

Tinker Air Force Base Substance Abuse Program
72 MDOS/SGOMH
5700 Arnold Street
Tinker AFB, OK 73145-8102

TISHOMINGO

Oaks Behavioral Health Center
117 West Main Street
Tishomingo, OK 73460

TONKAWA

Alpha II, Inc.
1608 North Main Street
Tonkawa, OK 74653

Tonkawa Tribe Substance Abuse Program
Tonkawa, OK 74653

TULSA

Browns School of Oklahoma
6262 South Sheridan Street
Tulsa, OK 74133

CBTI Tulsa Freedom Ranch
6126 East 32nd Place
Tulsa, OK 74135

Children's Medical Center
5300 East Skelly Drive
Tulsa, OK 74135-6599

Davis Counseling Program
1419 East 15th Street
Tulsa, OK 74120-5840

First Wings of Freedom
12 East 12th Street
Tulsa, OK 74119

Hillcrest Behavioral Services
1120 South Utica Street
Tulsa, OK 74104

Hillcrest Health Care System l Behavioral Health Services of Tulsa
1418 East 71st Street, Suite E
Tulsa, OK 74136-5060

How Foundation Rehabilitation Center of Oklahoma, Inc.
5649 South Garnett Road
Tulsa, OK 74146

Indian Health Care Resource Center of Tulsa
915 South Cincinnati Street
Tulsa, OK 74119

Life Improvement Center
5550 South Garnet Street
Tulsa, OK 74147-1903

Metro Tulsa Counseling Services
1602 North Cincinnati Avenue
Tulsa, OK 74106

New Choice and Associates
4833 South Sheridan Road
Suite 408
Tulsa, OK 74135

Parkside, Inc.
1620 East 12 Street
Tulsa, OK 74120

Street School, Inc.
1135 South Yale Avenue
Tulsa, OK 74112

Tulsa Regional Medical Center Chemical Dependency Unit
744 West 9 Street
Tulsa, OK 74127

Twelve and Twelve, Inc.
6333 East Skelley Drive
Tulsa, OK 74135

1214 South Baltimore Avenue
Tulsa, OK 74119-2820

Veterans' Affairs Medical Center Outpatient Clinic
635 West 11th Street
Tulsa, OK 74127

VANCE AFB

Vance Air Force Base Substance Abuse Program
527 Gott Road
Building 606
Vance AFB, OK 73705-5105

VINITA

Vinita Alcohol and Drug Treatment Center
Vinita, OK 74301

Vinita Alcohol and Drug Treatment Center
Vinita, OK 74301

WALTERS

Jim Taliaferro Community Mental Health and Substance Abuse Centers
319 South 3rd Street
Walters, OK 73572

WATONGA

Opportunities, Inc. Chemical Dependency Treatment Center
117 East First Street
Watonga, OK 73772

WAURIKA

Jim Taliaferro Community Mental Health and Substance Abuse Centers
431 East C Avenue
Waurika, OK 73573-2435

WETUMKA

Wetumka General Hospital Second Chance Substance Abuse Services
325 South Washita Street
Wetumka, OK 74883

WEWOKA

Mental Health Services of Southern Oklahoma
110 North Wewoka Street
Wewoka, OK 74884

Seminole Nation of Oklahoma Alcohol/Substance Abuse Program
400 South Brown Street
Wewoka, OK 74884

WILBURTON

Oaks Behavioral Health Center
113 West Ada Avenue
Wilburton, OK 74578-4008

WOODWARD

Western States Psychiatric Center
1222 10 Street
Suite 211
Woodward, OK 73801

OREGON

ALBANY

Addiction Counseling and Education Services, Inc. (ACES)
1856 Grand Prairie Road SE
Albany, OR 97321

Catherine Freer Wilderness Therapy Expeditions
420 SW 3rd Street
Albany, OR 97321

Linn County Alcohol and Drug Treatment Program
104 SW 4th Street
Albany, OR 97321

Serenity Lane
1209 Shortridge SE
Albany, OR 97321

ALOHA

BI, Inc.
18475 SW Alton Street
Aloha, OR 97006

ASHLAND

Community Works Lithia Springs Programs
695 Mistletoe Road, Suite H
Ashland, OR 97520

ASTORIA

Alcohol/Drug Programs
10 6th Street, Suite 103
Astoria, OR 97103

Clatsop Behavioral Health Center
10 6th Street
Astoria, OR 97103

Heron Outpatient Counseling Services
53 Portway Street
Astoria, OR 97103

BAKER CITY

Elkhorn Adolescent Treatment Center
3700 Midway Street
Baker City, OR 97814

New Directions Baker House
2330 5TH Street
Baker City, OR 97814

Powder River Alcohol and Drug Treatment Program
3600 13 Street
Baker City, OR 97814

BEAVERTON

Evans and Sullivan
97660 SW Beaverton-Hillsdale Highway
Beaverton, OR 97005

BEND

Central Oregon Extended Unit for Recovery
644 NE Greenwood Avenue
Bend, OR 97701

Deschutes County Human Services Substance Abuse Services
409 NE Greenwood Avenue
Suite 2
Bend, OR 97701

Serenity Lane
601 NW Harmon Street
Bend, OR 97701

BROOKINGS

Southcoast Addictions Program
505 Hemlock Street
Brookings, OR 97415

BURNS

Harney Counseling and Guidance Services
415 North Fairview Street
Burns, OR 97720

Wada Tika Health Center
HC-71 100 Pasigo Street
Burns, OR 97720

CANBY

Oregon Chicano Concilio
139 SW 2nd Avenue
Canby, OR 97013

CENTRAL POINT

Genesis Recovery Center
600 South 2nd Street
Central Point, OR 97502

CONDON

Mid-Columbia Center For Living Gilliam County Office
422 North Main Street
Condon, OR 97823-0705

COOS BAY

Ambit Southwestern Oregon Community Action Committee
2110 Newmark Street
Coos Bay, OR 97420

Better Options to Corrections
320 Central Street
Suite 408
Coos Bay, OR 97420

Coos Lowen Umpqua and Siuslaw Alcohol and Drug Program
338 Wallace Street
Coos Bay, OR 97420

Coquille Indian Tribe
Coos Bay, OR 97420

CORVALLIS

Addiction Counseling and Education Services, Inc.
885 NW Grant Street
Corvallis, OR 97330

Benton County Alcohol Treatment Program
530 NW 27 Street
Public Service Building
Corvallis, OR 97330

Discovery Counseling
260 SW Madison Street, Suite 101
Corvallis, OR 97339

Milestones Family Recovery Program
306 SW 8 Street
Corvallis, OR 97333

Outpatient Services
5185 SW 3rd Street
Corvallis, OR 97333

DALLAS

Polk County Mental Health Alcohol and Drug Treatment Program
182 SW Academy Street
Suite 304
Dallas, OR 97338

Valley Community Hospital Addiction Health Services
550 SE Clay Street
Dallas, OR 97338

ENTERPRISE

Wallowa County Mental Health Clinic Alcohol and Drug Program
207 SW 1st Street
Enterprise, OR 97828

EUGENE

Addiction Counseling and Education Services, Inc. (ACES)
84 Centennial Loop
Eugene, OR 97401

Bridge Program
1040 Oak Street
Eugene, OR 97401

Buckley Detoxification Services
605 West 4th Street
Eugene, OR 97402

Building Recovery
1210 Pearl Street
Eugene, OR 97401

Centro Latino Americana
944 West 5th Street
Eugene, OR 97402

Eugene Center for Family Development
1258 High Street
Eugene, OR 97401

Lane County Alcohol/Drug/ Offender Program
135 East 6 Avenue
Eugene, OR 97401

Looking Glass Adolescent Recovery Program
1675 West 11th Street
Eugene, OR 97401

Passages
1079 Alder Street
Eugene, OR 97401

Pathways
2391 Centennial Boulevard
Eugene, OR 97401

Prevention and Recovery Northwest
1188 Olive Street
Eugene, OR 97401

Serenity Lane, Inc. New Hope
2133 Centennial Plaza
Eugene, OR 97401

White Bird Clinic Chrysalis Program
332 East 12 Street
Eugene, OR 97401

Willamette Family Treatment Services
1420 Green Acres Road
Eugene, OR 97408

Women's Outpatient Program
687 Cheshire Street
Eugene, OR 97402

FOREST GROVE

Pacific Alcohol and Drug Counseling, Inc.
2021 Hawthorne Street
Forest Grove, OR 97116

GOLD BEACH

Curry County Substance Abuse Treatment Program
29821 Colvin Street
Gold Beach, OR 97444

GRAND RONDE

Confederated Tribes of Grand Ronde Human Services Division Alcohol and Drug Program
9615 Grand Ronde Road
Grand Ronde, OR 97347

GRANTS PASS

Adapt
424 NW 6th Street, Suite 102
Grants Pass, OR 97526

Choices Counseling Center
310 6th Street NW
Grants Pass, OR 97526

Genesis Recovery Center
124 NW Midland Avenue
Suite 104
Grants Pass, OR 97526-1269

Josephine County Community Corrections
304-306 D Street
Grants Pass, OR 97526

GRESHAM

Change Point
1217 NE Burnside Street
Gresham, OR 97030-5771

Network Project Stop
515 North East Roberts Street
Gresham, OR 97030

HEPPNER

Morrow/Wheeler Behavioral Health Alcoholism Services
120 South Main Street
Heppner, OR 97836

HERMISTON

Umatilla County Mental Health Services
405 North 1st Street, Suite 111
Hermiston, OR 97838-1843

HILLSBORO

Oregon Human Development Corporation Ayuda Community Services
441 South 1st Avenue
Hillsboro, OR 97123

Tuality Counseling and Addiction Services Alcohol and Drug Outpatient Program
848 SE Baseline Street
Hillsboro, OR 97123

Youth Contact
447 SE Baseline Street
Hillsboro, OR 97123

HOOD RIVER

Gorge Counseling/Treatment Services of Hood River Memorial Hospital
216 Columbia Avenue
Hood River, OR 97031

Mid-Columbia Center for Living Hood River Alcohol and Drug Program
1235 State Street
Hood River, OR 97031

JEFFERSON

Pacific Ridge
1587 Pacific Ridge Lane SE
Jefferson, OR 97352-9654

JOHN DAY

Grant County Center for Human Development
166 SW Brent Street
John Day, OR 97845

KLAMATH FALLS

Consortium Jail Treatment Program
3300 Vandenberg Road
Klamath Falls, OR 97603-3730

Corrections Annex Treatment
220 Main Street
Klamath Falls, OR 97601

Klamath Alcohol and Drug Abuse, Inc. (KADA)
310 South 5 Street
Klamath Falls, OR 97601

Klamath Consortium
296 Main Street
Klamath Falls, OR 97601

Lutheran Family Services
2545 North Eldorado Avenue
Klamath Falls, OR 97601

LINCOLN CITY

Discovery Counseling
1424 SE 51st Street
Room 202-A
Lincoln City, OR 97367

MADRAS

Chenan, Inc. Counseling and Intervention
27 D Street SE
Madras, OR 97741

MARYLHURST

Clackamas County Mental Health Center Alcohol and Drug Program
Marylhurst Campus Education Hall
Marylhurst, OR 97036

MCMINNVILLE

Yamhill County Mental Health Chemical Dependency Program
627 North Ford Street
McMinnville, OR 97128

MEDFORD

Jackson County Substance Abuse Program
338 North Front Street
Medford, OR 97501

KILPIA Counseling Services
111 Genessee Street
Medford, OR 97504

Ontrack, Inc.
221 West Main Street
Medford, OR 97501

3397 Delta Waters Road
Medford, OR 97501

Rogue Valley Addictions Recovery Center
1003 West Main Street
Medford, OR 97501

MILTON FREEWATER

Umatilla County Mental Health Milton Freewater Clinic
810 South Main Street
Milton Freewater, OR 97862

NEWBERG

Springbrook Northwest, Inc.
2001 Crestview Drive
Newberg, OR 97132

NEWPORT

Discovery Counseling
1628 North Coast Highway
Seatowne Shopping Center
Newport, OR 97365

Lincoln County Alcohol and Drug Program
255 SW Coast Highway
Newport, OR 97365

Lincoln County Council on Alcohol and Drug Abuse
155 SW High Street
Newport, OR 97365

Lincoln County Human Services Alcohol/Tobacco and Other Drugs Program
36 SW Nye Street
Newport, OR 97365-3823

Reconnections
1164 SW Coast Highway
Suites I and J
Newport, OR 97365

NORTH BEND

Center for Holistic Therapy
625 Oconnell Street
North Bend, OR 97459

Coos County Correctional Treatment Program
1975 McPherson Street
North Bend, OR 97459

ONTARIO

Lifeways Behavioral Health Counseling Center
1108 SW 4th Street
Ontario, OR 97914

Malheur County Alcohol and Drug Authority Alcohol Recovery Center
686 NW 9 Street
Ontario, OR 97914

OREGON CITY

Clackamas County Mental Health Center Alcohol and Drug Program
821 Main Street
Oregon City, OR 97045

Network Addiction Treatment Project Stop
1001 Molalla Avenue
Oregon City, OR 97045

Northwest Treatment Services
702 Main Street
Oregon City, OR 97045

PENDLETON

Brady and Associates
4705 NW Pioneer Place
Pendleton, OR 97801

Eastern Oregon Alcoholism Foundation
216 SW Hailey Avenue
Pendleton, OR 97801

Umatilla County Mental Health Program Substance Abuse Treatment Unit
721 SE 3 Street, Suite B
Pendleton, OR 97801

Yellow Hawk Tribal Health Center Chemical Dependency Program
Pendleton, OR 97801

PHOENIX

Phoenix Counseling Service
153 South Main Street
Phoenix, OR 97535

PORTLAND

Addictions Recovery Association
Letty Owings Center
2545 NE Flanders Street
Portland, OR 97232

Alpha Family Treatment Center
1427 SE 182nd Street
Portland, OR 97233

Annand Counseling Center
7320 SW Hunziker Road
Suite 200
Portland, OR 97223-2301

ASAP Treatment Services, Inc.
2130 SW 5th Avenue
Portland, OR 97201

BHC Pacific Gateway Hospital
1345 SE Harney Street
Portland, OR 97202

Caremark Chemical Dependency
3001 North Gantenbein Avenue
Portland, OR 97227

Cedar Hills Plaza Chemical Dependency Services
10300 SW Eastridge Road
Providence Cedar Hills Plaza
Portland, OR 97225

Center for Community Mental Health
3716 NE Martin Luther King Boulevard
Portland, OR 97211

Changepoint Diversion Association
1949 SE 122nd Avenue
Portland, OR 97233

Comprehensive Options for Drug Abusers CODA)
1027 East Burnside Street
Portland, OR 97214

Columbia River Correctional Institution Turning Point
9111 NE Sunderland Avenue
Portland, OR 97211

De Paul Adult Treatment Center
1320 SW Washington Street
Portland, OR 97205

De Paul Youth Treatment Center
4411 NE Emerson Street
Portland, OR 97218

General Health, Inc.
2600 SE Belmont Street
Portland, OR 97212

Hooper Detox
20 NE Martin Luther King Boulevard
Portland, OR 97232

Legacy Emanuel Hospital Project Network
2631 North Mississippi Avenue
Portland, OR 97227

Native American Rehabilitation
Association of The Northwest,Inc.
17645 NW Saint Helens Highway
Portland, OR 97231

Network Behavioral Healthcare
Addiction Treatment Services
2415 SE 43rd Avenue,Suite 200
Portland, OR 97206

Harmony House
2270 SE 39th Avenue
Portland, OR 97214

Northwest Treatment Services
9370 SW Greenburg Road
Suite 601
Portland, OR 97223

948 NE 102 Street
Suite 101
Portland, OR 97220

OHSU Behavioral Health Services
621 SW Alder Street, Suite 520
Portland, OR 97205-3620

Oregon Chicano Concilio
1732 NE 43rd Street
Portland, OR 97213

Pacific Alcohol and Drug Counseling Inc
11515 SW Durham Road
Suite E-8
Portland, OR 97224

Portland Addictions/ Acupuncture Center
120 SW Morrison Street
Portland, OR 97205

Project for Community Recovery
3525 NE Martin Luther King Jr. Blvd.
Portland, OR 97212

Providence Medical Center Addictions Treatment Services
5211 NE Glisan Street
Portland, OR 97213

Providence Milwaukie Hospital Chemical Dependency Services
10150 SE 32nd Avenue
Portland, OR 97222

Ram Clinic
3610 NE 82nd Avenue, Suite 100
Portland, OR 97220

Serenity Lane
9414 SW Barbur Boulevard
Suite B
Portland, OR 97219

Stay Clean, Inc.
1223 Alberta Street NE
Portland, OR 97211

Tualatin Valley Centers
14600 NW Cornell Road
Portland, OR 97229

9111 Sunderland Road NE
Portland, OR 97211

2130 SW 5th Avenue, Suite 210
Portland, OR 97201-4934

Volunteers of America
Mens Residential Center
2318 NE Martin Luther King Boulevard
Portland, OR 97212

Women's Residential Center
200 SE 7th Street
Portland, OR 97214

Woodland Park Behavioral Health Service
10300 NE Hancock Street
Portland, OR 97220

PRINEVILLE

Lutheran Family Services Crook County Mental Health Program
203 North Court Street
Prineville, OR 97754

Rimrock Trails
1333 NW 9th Street
Prineville, OR 97754

REDMOND

Visions of Hope Recovery Center
676 Negus Way
Redmond, OR 97756

REEDSPORT

Adapt
2785 Frontage Road
Reedsport, OR 97467-1814

ROSEBURG

Adapt
548 SE Jackson Street, Suite 1
Roseburg, OR 97470

Deer Creek Adolescent Treatment Center
2064 Douglas Street SE
Roseburg, OR 97470

Roseburg Recovery Services
727-B Southeast Main Street
Roseburg, OR 97470

Crossroads
3099 NE Diamond Lake Boulevard
Roseburg, OR 97470

SAINT HELENS

Columbia Community Mental Health
105 South 3rd Street
Saint Helens, OR 97051

SALEM

Bridgeway
3325 Harold Street NE
Salem, OR 97305

Chemawa Alcoholism Education Center
3760 Chemawa Road NE
Salem, OR 97305

Hillcrest Youth Correctional Facility
2450 Strong Road SE
Salem, OR 97310

Inside Out Care, Inc.
780 Commercial Street SE
Suite 105
Salem, OR 97302

Marion County Health Department
3180 Center Street NE
Room 2274
Salem, OR 97301

Multicultural Consultants, Ltd.
3760 Market Street NE 316
Salem, OR 97301

Nanitch Sahallie Treatment Center
5119 River Road NE
Salem, OR 97303

Network, Inc. Harmony House of Marion County
3040 Center Street NE
Salem, OR 97301

New Step Behavioral Health
1655 Capitol Street NE, Suite 1
Salem, OR 97303

Pacific Alcohol and Drug, Inc. Step Program
4005 Aumsville Highway SE
Salem, OR 97301

Pacific Recovery
1235 Woodrow Street NE
Salem, OR 97303

Seasons
1582 Lancaster Drive NE
Salem, OR 97301

Serenity Lane
910 Capitol Street NE
Salem, OR 97301

Tahana Whitecrow Foundation
2350 Wallace Road NW
Salem, OR 97304-2127

SANDY

Sandy Family Services, Inc.
39365 Proctor Boulevard
Sandy, OR 97055

SCAPPOOSE

Heart to Heart Counseling Center
52700 North East 1st Street
Scappoose, OR 97056

SILETZ

Siletz Tribal Council Alcohol and Drug Program
201 SW Swan Street
Siletz, OR 97380

SILVERTON

Seasons
209 C Street
Silverton, OR 97381

STAYTON

Stayton Counseling
223 Locust Street
Stayton, OR 97383

THE DALLES

Mid-Columbia Center for Living
400 East 5 Street
Room 207
The Dalles, OR 97058

TIGARD

Tigard Recovery Center
10362 SW McDonald Road
Tigard, OR 97224

WARM SPRINGS

Confederated Tribes of Warm Springs Alcohol and Drug Abuse Program
Warm Springs, OR 97761

WOODBURN

Bridgeway, Inc.
399 Young Street
Woodburn, OR 97071

PENNSYLVANIA

AKRON

Recovery Unlimited, Inc.
115 North 9th Street
Akron, PA 17501-1341

ALIQUIPPA

Drug and Alcohol Services of Beaver Valley
524 Franklin Avenue
Aliquippa, PA 15001

Gateway Rehabilitation Center
Economy Village
Road 2
Aliquippa, PA 15001

Linmar Terrace
1200 Tyler Street Rental Office
Aliquippa, PA 15001

Mount Washington Homes
Pleasantview Homes
Moffett Run Road
Aliquippa, PA 15001

Tom Rutter House
100 Moffet Run Road
Aliquippa, PA 15001

ALLENTOWN

Family House
112 North 9th Street
Allentown, PA 18102

Florence Child Guidance Center
1812 Allen Street
Allentown, PA 18104

Livengrin Counseling Center
961 Marcon Boulevard Suite 304
Allentown, PA 18103

Saint Luke's Addictions Service Halfway Home of Lehigh Valley
121 North 8th Street
Allentown, PA 18101

Saint Luke's Hospital Allentown Campus
1736 Hamilton Street
Allentown, PA 18104

32 North 18th Street
Allentown, PA 18104

Recovery Center
33 North Saint George Street
Allentown, PA 18104

Treatment Trends, Inc.
Confront Program
1130 Walnut Street
Allentown, PA 18102

Keenan House
18-22 South 6th Street
Allentown, PA 18105

White Deer Run of Allentown
1132 Hamilton Street, Suite 300
Allentown, PA 18101

ALLENWOOD

White Deer Run
Devitt Camp Road
Allenwood, PA 17810

ALTOONA

Altoona Hospital Mental Health Alcohol and Drug Services
620 Howard Avenue
Altoona, PA 16601

AMP/CEP Group Homes, Inc.
T/A Right Turn
901 6 Avenue
Altoona, PA 16602

830 6 Avenue
Altoona, PA 16602

825 1/2 7th Avenue
Altoona, PA 16603

Blair County Community Action Program Substance Abuse Services
2100 6th Avenue
Altoona, PA 16601

Home Nursing Agency Community Support Alternatives
500 East Chestnut Avenue
Altoona, PA 16601

AMBLER

Northwestern Human Services of Montgomery County
600 North Bethlehem Pike
Ambler, PA 19002

ARDMORE

Jewish Family and Children's Service of Philadelphia
133 Coulter Avenue
Ardmore, PA 19003

Lower Merion Counseling Services
7 East Lancaster Avenue
Ardmore, PA 19003

Womanspace
120 Ardmore Avenue
Ardmore, PA 19003

ASHLAND

Gaudenzia at Fountain Springs Women and Children Program
95 Broad Street
Ashland, PA 17921

AUDUBON

Saint Gabriel's Hall
1300 Pawlings Road
Audubon, PA 19407

BANGOR

Community Psychological Center Inc
715 Pennsylvania Avenue
Bangor, PA 18013

BEAVER

Drug and Alcohol Services of Beaver Valley, Inc.
697 State Street
Beaver, PA 15009

BEAVER FALLS

Gateway Rehabilitation Center
Harmony Dwellings
Rent Office 9th Street
Beaver Falls, PA 15010

Morada Dwellings
Apartment 136, Morada Dwellings
Beaver Falls, PA 15010

BELLEFONTE

Comprehensive Recovery Care, Inc.
323 West High Street
Bellefonte, PA 16823-1303

Counseling Services, Inc.
Drug and Alcohol Program
441 North Spring Street
Bellefonte, PA 16823

BENSALEM

De Lasalle Vocational
Street Road and Bristol Street
Bensalem, PA 19020

Libertae, Inc.
5245 Bensalem Boulevard
Bensalem, PA 19020

Livengrin Foundation Inc
4833 Hulmeville Road
Bensalem, PA 19020-3099

BERLIN

Twin Lakes Center
426 Main Street
Berlin, PA 15530

BERWICK

Berwick's Recovery System
701 East 16th Street
Berwick, PA 18603

BETHLEHEM

Hogar Crea of Bethlehem
1409 Pembroke Road
Bethlehem, PA 18017-7198

Saint Luke's Addictions Treatment Services Incorporated
50 East Broad Street
Bethlehem, PA 18018

1107 Eaton Avenue
Bethlehem, PA 18018

Step By Step, Inc.
623 West Union Boulevard
Bethlehem, PA 18018

BIRDSBORO

Center for Mental Health
201 East Main Street
Birdsboro, PA 19508

BLOOMSBURG

Behavioral Health Resource Group of Bloomsburg
603 West Main Street
Bloomsburg, PA 17815

Bloomsburg Hospital New Hope Drug And Alcohol Services
480 Central Road
Bloomsburg, PA 17815

BOYERTOWN

Inner Direction Counseling Center
400 Sweinhart Road
Boyertown, PA 19512

BRADDOCK

UPMC Braddock
400 Holland Avenue
Braddock, PA 15104

BRADFORD

Alcohol and Drug Abuse Services Bradford Unit
2 Main Street
Seneca Building, Suite 600
Bradford, PA 16701

Bradford Regional Medical Center Mentally/Chem Add/ Dual Diag/Psych Unit
116-156 Interstate Parkway
Bradford, PA 16701-1097

BRISTOL

Livengrin
1270 New Rogers Road
Bristol, PA 19007

Lower Bucks Hospital Mental Health Services
501 Bath Road
Bristol, PA 19007

BUTLER

Butler Memorial Hospital
Regional Recovery Program/ Outpatient
911 East Brady Street
Butler, PA 16001

Regional Recovery Program
911 East Brady Street
Butler, PA 16001

Charter Outpatient Recovery Center
118 South Church Street
Butler, PA 16001

Irene Stacy Community Mental Health Center
112 Hillvue Drive
Butler, PA 16001

Veterans' Affairs Medical Center Substance Abuse Treatment Unit (SATU)
325 New Castle Road
Butler, PA 16001

CAMP HILL

Guidance Associates
412 Erford Road
Camp Hill, PA 17011

Holy Spirit Hospital Drug and Alcohol Medical Service Unit
503 North 21 Street
Camp Hill, PA 17011

Roxbury in Camp Hill Intensive Outpatient/Outpatient
3300 Trindle Road
Camp Hill, PA 17011

Russell, Russell and Associates, Inc.
1940 Market Street
Camp Hill, PA 17011

CANONSBURG

Gateway Greentree
6000 Waterdam Plaza Drive
Suite 260
Canonsburg, PA 15317

CARBONDALE

Drug and Alcohol Treatment Service
9 North Main Street
Carbondale, PA 18407-2316

CARLISLE

Carlisle Area Counseling Services
700 Clay Street
Carlisle, PA 17013

Carlisle Hospital
246 Parker Street
Carlisle, PA 17013

Stevens Center
401 East Louther Street
Carlisle, PA 17013

CHAMBERSBURG

Manito, Inc.
7564 Browns Mill Road
Chambersburg, PA 17201

Twin Lakes Center Drug and Alcohol Rehabilitation
166 South Main Street
Kerrstown Square Suite 202
Chambersburg, PA 17201

CHESTER

Ches Penn Health Services, Inc.
619 Welsh Street
Chester, PA 19013

1300 West 9th Street
Chester, PA 19015

Crozer Chester Medical Center
CHS Methadone Program
CHS Outpatient Service
2600 West 9th Street
Chester, PA 19013

UHS Keystone Center
2001 Providence Road
Chester, PA 19013

CLARION

Clarion County Counseling Center
Drug/Alcohol Administration
214 South 7 Avenue
Clarion, PA 16214

CLARKS SUMMIT

Lourdesmont Good Shepherd Youth and Family Services
537 Venard Road
Clarks Summit, PA 18411

CLIFTON HEIGHTS

Family and Community Service of Delaware County
37 North Glenwood Avenue
Clifton Heights, PA 19018

COATESVILLE

Continuum, Inc.
131 Harmony Street
Coatesville, PA 19320

Samara House YWCA
423 East Lincoln Highway
Coatesville, PA 19320

Veterans Affairs Medical Center Substance Abuse Treatment Program
1400 Black Horse Hill Road
Coatesville, PA 19320-2097

COLUMBIA

Lancaster General Hospital Susquehanna Division Addictions Center
306 North Seventh Street
Columbia, PA 17512-0926

CONNELLSVILLE

Fayette County Drug and Alcohol Commission, Inc.
1032 Morrell Avenue
Connellsville, PA 15425

COUDERSPORT

Charles Cole Memorial Hospital Alcohol and Drug Abuse Services
107 East Second Street
Coudersport, PA 16915

CRANBERRY TOWNSHIP

Butler Regional Recovery Evening Program
20421 Route 19 Suite 100
Butler Centre
Cranberry Township, PA 16066-7514

Discovery House
326 Thomson Park Drive
Building 300
Cranberry Twp, PA 16066

Irene Stacy CMHC Drug and Alcohol Unit
Butler Center
20421 Route 19, Suite 310
Cranberry Township, PA 16066

Saint Francis Medical Center North Center for Addiction Services
1 Saint Francis Way
Cranberry Township, PA 16066-5119

CRUM LYNNE

Teencare
1124 Chester Pike First Floor
Crum Lynne, PA 19022

DANVILLE

Penn State Geisinger Health System Alcohol/Chemical Dependency Outpatient Services
12 Poplar Street
Danville, PA 17822

Psychological Services Clinic
405 Bloom Street
Danville, PA 17821

DELTA

Adams Hanover Counseling Services Delta
5 Pendyrus Street
Delta, PA 17314

DOYLESTOWN

Aldie Counseling Center
228 North Main Street
Doylestown, PA 18901

Bucks County Correctional Facility Drug and Alcohol Unit
1730 South Easton Road
Doylestown, PA 18901

Bucks County Council on Alcoholism and Drug Dependence
Routes 313 and 611
252 West Swamp Road/Unit 33
Doylestown, PA 18901

Livengrin Counseling Center
275 South Main Street, Suite 11
Terrace Office Center
Doylestown, PA 18901

DREXEL HILL

Delaware County Memorial Hospital Alcoholism and Addiction Treatment Center
501 North Lansdowne Avenue
Drexel Hill, PA 19026

DU BOIS

Concerns Counseling and Consultation Firm
90 Beaver Drive
Du Bois, PA 15801

EAGLEVILLE

Eagleville Hospital Inpatient Program
100 Eagleville Road
Eagleville, PA 19408

EASTON

Saint Luke's Addiction Treatment Services, Inc.
158-160 South 3rd Street
Easton, PA 18042

Twin Rivers Medical Inc
158 South 3rd Street
Easton, PA 18042-4518

EAST PETERSBURG

Lancaster Area Psychological Services
6079 Main Street
East Petersburg, PA 17520-1267

EBENSBURG

Home Nursing Agency Community Services
594 Manor Drive
Ebensburg, PA 15931

ELIZABETHTOWN

HSA Counseling Inc
11 Center Square
Elizabethtown, PA 17022

Naaman Center
4600 East Harrisburg Pike
Elizabethtown, PA 17022

ELKINS PARK

Jewish Family and Children's Service of Philadelphia
7607 Old York Road Lower Level
Elkins Park, PA 19027

ELKLAND

Laurel Health Center
103 Forestview Drive
Elkland, PA 16920

ELLWOOD CITY

Drug and Alcohol Community Treatment Services, Inc.
720 Lawrence Avenue
Ellwood City, PA 16117

ELWYN

ChesPenn Health Services
176 South Middletown Road
Elwyn, PA 19063

EMPORIUM

Alcohol and Drug Abuse Center
107 South Cherry Street
Emporium, PA 15834

EPHRATA

Terraces
1170 South State Street
Ephrata, PA 17522

ERIE

Charter Behavior Health System at Cove Forge/Frontier Place
1371 West 6th Street
Erie, PA 16505

Community House, Inc.
521 West 7 Street
Erie, PA 16502

Cornell Abraxas II
502 West 6th Street
Erie, PA 16502

Crossroads/Serenity Hall
414 West 5 Street
Erie, PA 16507

Dr. Daniel S. Snow Recovery House
361 West 5th Street
Erie, PA 16507

Family Services of Northwestern Pennsylvania
121 West 10th Street
Erie, PA 16501

Perseus House, Inc.
132 West 26 Street
Erie, PA 16508

516 West 7 Street
Erie, PA 16502

Saint Vincent Health Center Serenity Recovery Center for Substance Abuse Outpatient Program
2409 State Street
Erie, PA 16544

Stairways Mental Health Drug and Alcohol Unit
531 West 10th Street
Erie, PA 16504

Veterans Affairs Medical Center Substance Abuse Treatment Program
135 East 38th Street
Psychology Service 116-B
Erie, PA 16504

EXTON

Alcoholism and Addictions Council Holcomb Behavioral Health
930 East Lancaster Avenue
Suite 220
Exton, PA 19341

UHS Recovery Foundation, Inc. Key Recovery Center
319 North Pottstown Pike
Suite 102
Exton, PA 19341

FARRELL

Insights Chemical Dependency Program Outpatient/Shenango
1980 Green Street
Farrell, PA 16121

FORD CITY

Ministries of Eden Inc
837 5th Avenue
Ford City, PA 16226

FORT WASHINGTON

Livengrin Counseling Center
520 Pennsylvania Avenue
Fort Washington, PA 19034

FRANKLIN

Family Services and Children's Aid Society Society Drug Alcohol Program
1243 Liberty Street
Franklin, PA 16323

GETTYSBURG

Adams Hanover Counseling Services, Inc.
44 South Franklin Street
Gettysburg, PA 17325

Cornerstone Counseling and Education Services
108 North Stratton Street
Gettysburg, PA 17325

The Recovery Place
69 West Middle Street
Gettysburg, PA 17325

GLENSIDE

Milestones
614 North Easton Road
Glenside, PA 19038

GREENSBURG

CSAS, Inc. Myriad Program
211 Huff Avenue, Suite D
Greensburg, PA 15601

GREENVILLE

Insights Chemical Dependency Program Outpatient/ Greenville
60 South Race Street
Greenville, PA 16125

GROVE CITY

George Junior Republic
200 George Junior Road
Grove City, PA 16127-5058

Horizon Hospital Insights Chemical Dependency Program
430 Hillcrest Avenue
Grove City, PA 16127

HANOVER

Adams Hanover Counseling Services, Inc.
625 West Elm Avenue
Hanover, PA 17331

Cornerstone Counseling and Education Services
11 York Street Suite 101
Hanover, PA 17331

HARRISBURG

Another Chance Counseling
200 Shell Street
Harrisburg, PA 17109

Conewago Place Outpatient
2901 North 6th Street
Harrisburg, PA 17110

Discovery House
99 South Cameron Street
Harrisburg, PA 17101

Gaudenzia
Chambers Hill Adolescent Program
3740 Chambers Hill Road
Harrisburg, PA 17111

Common Ground
2835 North Front Street
Harrisburg, PA 17110

90 Concept
Spruce Road
Harrisburg State Hospital Building
21 Harrisburg, PA 17105

Outpatient Services
2039 North 2nd Street
Harrisburg, PA 17102

Harrisburg Area Counseling Services
3907 Derry Street
Harrisburg, PA 17111

Hoffman Psychological Associates
3029 North Front Street
Suite 102
Harrisburg, PA 17110-1220

Pinnacle Health Psychological Associates
205 South Front Street
Harrisburg, PA 17105

Riegler Shienvold and Associates
2151 Linglestown Road, Suite 200
Harrisburg, PA 17110-9455

Teen Challenge
1421 North Front Street
Harrisburg, PA 17102

Tressler Greater Harrisburg Alcohol and Drug Counseling
3309 Spring Street, Suite 204
Harrisburg, PA 17109

Weaver Counseling
4607 Locust Lane
Harrisburg, PA 17109

HAVERTOWN

Mercy Haverford Hospital Substance Abuse Services
2000 Old West Chester Pike
Havertown, PA 19083

HAZLETON

A Better Today, Inc.
21 North Church Street
Hazleton, PA 18201

Northeast Counseling Services
750 East Broad Street
Hazleton, PA 18201

Serento Gardens Alcohol and Drug Services
145 West Broad Street, 2nd Floor
Hazleton, PA 18201

HENRYVILLE

Greenway Center
State Route 715-314
Henryville, PA 18332

HERMITAGE

Sharon Regional Health System Behavioral Health Services
2375 Garden Way
Hermitage, PA 16148

HERSHEY

Bennett, Timothy
825 Fishburn Road
Hershey, PA 17033

Guidance Associates of Pennsylvania
475 West Governor Road
Hershey, PA 17033

University Recovery Center Department of Psychiatry
500 University Drive
Hershey, PA 17033

HILLER

Fayette County Drug and Alcohol Commission Inc
903 First Street
Hiller, PA 15444

HOMESTEAD

Caty Services Family Recovery Center
120 East 9th Street
Homestead, PA 15120

HUMMELSTOWN

Conewago Place
424 Nye Road
Hummelstown, PA 17036

HUNTINGDON

Mainstream Counseling
1001 Washington Street
Huntingdon, PA 16652

HYNDMAN

Twin Lakes Center for Drug and Alcohol Rehabilitation
Hyndman Area Health Center
Hyndman, PA 15545

INDIANA

The Open Door, Inc.
20 South 6 Street
Indiana, PA 15701

Twin Lakes
840 Philadelphia Street
Indiana, PA 15701

JEANNETTE

Adelphoi Village McKee Home
109 North 2nd Street
Jeannette, PA 15644

Monsour Medical Center
70 Lincoln Way East
Jeannette, PA 15644-3167

JOHNSTOWN

Croyle Psychological Associates
1450 Scalp Avenue, Suite 209
Johnstown, PA 15904

New Visions/Mercy Hall Drug and Alcohol Program
1020 Franklin Street
Johnstown, PA 15905

Peniel Drug and Alcohol Treatment Facility
760 Copper Avenue
Johnstown, PA 15906

Twin Lakes Center for Drug Alcohol Rehabilitation
406 Main Street, Suite 408
Johnstown, PA 15901

KANE

Alcohol and Drug Abuse Services Kane Unit
16 Greeves Street
Kane, PA 16735

Kane Community Hospital Detox Program
North Fraley Street
Kane, PA 16735

Nelson Behavioral Center
Presbyterian Church Greene Street
Kane, PA 16735

KEMPTON

Blue Mountain House of Hope
8284 Leaser Road
Kempton, PA 19529-0067

KENNETT SQUARE

Bowling Green Inn Brandywine
1375 Newark Road
Kennett Square, PA 19348

NHS Help Counseling Division
500 North Walnut Street
Kennett Square, PA 19348

KING OF PRUSSIA

Rehabilitation After Work
700 South Henderson Road
Suite 10 Merion Building
King of Prussia, PA 19406

KINGSTON

Choices A Divsion of Community Counseling Services
518 Wyoming Avenue
Kingston, PA 18704

Clem/Mar House, Inc.
540-542 Main Street
Kingston, PA 18704

KITTANNING

Armstrong County Memorial Hospital Alcohol and Drug Services
1 Nolte Drive
Kittanning, PA 16201

Armstrong County Council On Alcohol and Other Drugs, Inc./ARC Manor
200 Oak Avenue
Kittanning, PA 16201

KUTZTOWN

Center for Mental Health Care
Trexler and Noble Street
Kutztown, PA 19530

LANCASTER

Drug and Alcohol Rehab Service, Inc. Manos Residential Therapeutic Community
121 South Prince Street
Lancaster, PA 17603

Family Service of Lancaster County
630 Janet Avenue
Lancaster, PA 17601

HSA Counseling, Inc.
48 North Queen Street, 3rd Floor
Lancaster, PA 17603

Lancaster Clinical Counseling Assoc.
131 East Orange Street
2nd Floor/Rear
Lancaster, PA 17602

Lancaster Freedom Center
436 North Lime Street
Lancaster, PA 17602

Nuestra Clinica of Saca Da Program
545 Pershing Avenue
Lancaster, PA 17602

T.W. Ponessa Associates Counseling
448 Murry Hill Circle
Lancaster, PA 17601-4141

White Deer Run of Lancaster
53-55 North West End Avenue
Lancaster, PA 17604

LANGHORNE

Jewish Family and Children's Service of Philadelphia
340 East Maple Avenue
Suite 107
Langhorne, PA 19047

LANSDALE

Help Line Center, Inc.
306 A Madison Avenue
Lansdale, PA 19446

LEBANON

Another Chance Counseling
607 South 14th Avenue
Lebanon, PA 17042-8805

Renaissance Counseling
701 Chestnut Street
Lebanon, PA 17042

Veterans' Affairs Medical Center Substance Abuse Treatment Unit (SATU)
1700 South Lincoln Avenue
Lebanon, PA 17042

LEHIGHTON

Carbon/Monroe/Pike Drug/ Alcohol Commission, Inc.
128 South First Street
Lehighton, PA 18235

LEWISTOWN

Clear Concepts
218 Electric Avenue
Lewistown, PA 17044

N/P Health Services
400 Highland Avenue
Lewistown, PA 17044-1198

LITITZ

Hear, Inc. Gate House for Men
649 East Main Street
Lititz, PA 17543

LOCK HAVEN

Green Ridge Counseling Center
Unit IV
25 West Main Street
Lock Haven, PA 17745

MALVERN

Malvern Institute
940 King Road
Malvern, PA 19355

MANSFIELD

Laurel Health Center
40 West Wellsboro Street
Mansfield, PA 16933

MARIENVILLE

Abraxas Foundation, Inc. Abraxas I
Blue Jay Village
Marienville, PA 16239

MARS

Gateway North Hills
1559 Route 228
Mars, PA 16046

MCKEESPORT

Center for Substance Abuse
120 5 Avenue
McKeesport, PA 15132

Mon Yough Women and Family Center
515 Sinclair Street
McKeesport, PA 15132

Whales Tale Substance Abuse Treatment Services
416 Olive Street
McKeesport, PA 15132

MCKEES ROCKS

Northern Southwest Community MH/MR/DA Services
McKees Rocks Center
710 Thompson Avenue
McKees Rocks, PA 15136

MEADVILLE

Crawford County Drug and Alcohol Executive Commission
898 Park Avenue
Suite 12
Meadville, PA 16335

Meadville Medical Center Stepping Stones
1034 Grove Street
Meadville, PA 16335

MECHANICSBURG

Gaudenzia Foundation Inc. West Shore Outpatient Program
6 State Road
Suite 115
Mechanicsburg, PA 17055

MEDIA

Family and Community Service of Delaware County
100 West Front Street
Media, PA 19063

Focus Counseling Center, Inc.
700 North Jackson Street
Media, PA 19063-2527

Mirmont Treatment Center
100 Yearsley Mill Road
Media, PA 19063

MIFFLINTOWN

Clear Concepts
Rural Route 4
Mifflintown, PA 17059

MILFORD

Carbon/Monroe/Pike Drug/ Alcohol Commission Pike County Clinic
10 Buist Road
Milford, PA 18337

MILTON

Bethesda Day Treatment Center
Milton Center 49 Lower Market Street
Milton, PA 17847

Green Ridge Counseling Center Unit I
28 North Front Street
Milton, PA 17847

MOHNTON

Rose Kearney Halfway House
225 East Wyomissing Avenue
Mohnton, PA 19540

MONESSEN

CSAS, Inc. Mon Valley Drug and Alcohol Program
8 Eastgate Street
Monessen, PA 15062-1385

MONONGAHELA

Whale's Tale Freedom
1290 Chess Street
Monongahela, PA 15063

Monongahela Valley Hospital
Country Club Road
Monongahela, PA 15063

MONROEVILLE

Gateway/Monroeville
4327 Northern Pike
Monroeville, PA 15146

Saint Francis Medical Center Center for Chemical Dependency
2550 Mosside Boulevard
Medical Arts Building, Suite 212
Monroeville, PA 15146

MONTROSE

Trehab Center, Inc.
10 Public Avenue
Montrose, PA 18801

MORRISVILLE

Good Friends, Inc.
868 West Bridge Road
Morrisville, PA 19067

MOUNTAINHOME

Performance Strategies, Inc.
Route 390
Mountainhome, PA 18342

MOUNTVILLE

Gatehouse for Women
465 West Main Street
Mountville, PA 17554-0403

NANTICOKE

Northeast Counseling Services
130 West Washington Street
Nanticoke, PA 18634

NATRONA HEIGHTS

Butler Regional Recovery Program Outpatient
1301 Carlisle Street
Natrona Heights, PA 15065

NEW BLOOMFIELD

Perry Human Services
New Bloomfield, PA 17068

NEW CASTLE

Drug and Alcohol Community Treatment Services, Inc.
332 Highland Avenue
New Castle, PA 16101

Essawi Counseling Services
343 East Washington Street
New Castle, PA 16101

Highland House
312 Highland Avenue
New Castle, PA 16101

Saint Francis Hospital Detox Unit
1000 South Mercer Street
New Castle, PA 16101

NEW CUMBERLAND

New Insights, Inc.
R 320 Bridge Street
Suite 96
New Cumberland, PA 17070

NEW KENSINGTON

Alle Kiski Pavilion
4th and 17th Avenue
New Kensington, PA 15068

Csas Alle Kiski Drug and Alcohol Program
2120 Freeport Road
New Kensington, PA 15068

Greenbriar Treatment Center
251 7th Street, Suite F
New Kensington, PA 15068

NEWTOWN

Today, Inc.
1990 North Woodbourne Road
Newtown, PA 18940-0841

NORRISTOWN

Family House/Norristown
901 Dekalb Street
Norristown, PA 19401

Montgomery County Methadone Center
316 Dekalb Street
Norristown, PA 19401

Montgomery County MH/MR Emergency Service
50 Beech Drive
Norristown, PA 19401

Programs in Counseling
20 West Main Street
Norristown, PA 19401

Valley Forge Medical Center and Hospital
1033 West Germantown Pike
Norristown, PA 19403

OIL CITY

Northwest Medical Center Drug and Alcohol Program
174 East Bissell Avenue
Oil City, PA 16301

Northwest Medical Center
136 East Bissell Avenue
Oil City, PA 16301

PAOLI

Center for Addictive Diseases
21 Industrial Boulevard, Suite 200
Paoli, PA 19301

Constructive Living
63 Chestnut Road, Suite 3
Paoli, PA 19301

Rehab After Work
1440 Russell Road
Paoli, PA 19301

PHILADELPHIA

Abbottsford Community Health Center
3205 Defense Terrace
Philadelphia, PA 19129

Achievement Through Counseling and Treatment
1745 North 4th Street
Philadelphia, PA 19122

Alcohol and Mental Health Associates
1200 Walnut Street
2nd Floor
Philadelphia, PA 19107

Alleghany University
Hanneman Division
Institute for Addictive Disorders Youth Opportunity Program
511 North Broad Street
Philadelphia, PA 19123

Asociacion de Puertorriquenos En Marcha Inc.
2147 North 6th Street
Philadelphia, PA 19122

Proyecto Borinquen
520 West Venango Street
Philadelphia, PA 19140

Proyecto Nueva Vida
2143 North 6th Street
Philadelphia, PA 19122

Beacon House at Episcopal Hospital
100 East Lehigh Avenue
Philadelphia, PA 19125

Bowling Green/Center City
1420 Walnut Street Suite 1212
Philadelphia, PA 19102-4017

Caring Together Perinatal Addictions Program
3300 Henry Avenue
Philadelphia, PA 19129

Community Council for Mental Health and Mental Retardation
4900 Wyalusing Avenue
Philadelphia, PA 19131

Consortium, Inc. University City Counseling Center
451 University Avenue
Philadelphia, PA 19104

Cora Services
Community Services Division
733 Susquehanna Road
Philadelphia, PA 19111-1399

Neumann Program
Adams Avenue and Orthodox Road
Philadelphia, PA 19124

De Lasalle in Towne
25 South Van Pelt Street
Philadelphia, PA 19103

Diagnostic and Rehabilitation Center
Main Clinic
229 Arch Street
Philadelphia, PA 19106

Hutchinson Place
3439 West Hutchinson Street
Philadelphia, PA 19140

Dr. Warren E. Smith Community Substance Abuse Centers, Inc.
1315 Windrim Avenue
Philadelphia, PA 19141

Family Center
1201 Chestnut Street 11th Floor
Philadelphia, PA 19107

Family Preservation Program
4219 Chester Avenue
Philadelphia, PA 19104

Frankford Hospital
First Days Program
Frankford Avenue and Wakeling Street
Philadelphia, PA 19124

Friends Hospital
4641 Roosevelt Boulevard
Philadelphia, PA 19124

Gaudenzia
5401 Wayne Avenue
Philadelphia, PA 19144

1415 North Broad Street
Room 116
Philadelphia, PA 19122

1300 East Tulpehocken Street
Philadelphia, PA 19138

3025 North Broad Street
Philadelphia, PA 19132

1834 West Tioga Street
Philadelphia, PA 19140

Genesis II, Inc.
1214 North Broad Street
Philadelphia, PA 19121

Caton House
1239 Spring Garden Street
Philadelphia, PA 19123

Girard Medical Center Return Program/Forensic Intensive Rehab
8th and Girard Avenue
Philadelphia, PA 19122

Horizon House
Outpatient Substance Abuse Program
120 South 30th Street
5th Floor
Philadelphia, PA 19104

Susquehanna Park Residential Community
2137 North 33rd Street
Philadelphia, PA 19121

Hospitality House Outpatient Services
2134 North Hancock Street
Philadelphia, PA 19122

Hutchinson Place
3439 North Hutchinson Street
Philadelphia, PA 19140

Intercommunity Action, Inc. (Interac) Alcohol/Education/ and Family Counseling Program
6122 Ridge Avenue
Philadelphia, PA 19128

Interim House West
4150-52 Parkside Avenue
Philadelphia, PA 19104

Interim House West II
4234 Parkside Avenue
Philadelphia, PA 19104

Interphase Recovery System
814 East Allegheny Avenue
Philadelphia, PA 19134-2402

Jefferson Intensive Outpatient Program
Jefferson Methadone Clinic
21 Street and Washington Avenue
Philadelphia, PA 19146

Jefferson Outreach Drug and Alcohol Program
Central District
1201 Chestnut Street
14th Floor
Philadelphia, PA 19107

JEVS/ACT Achievement Through Counseling and Treatment
5820 Old York Road
Philadelphia, PA 19141-2598

Jewish Family and Children's Service Project Pride
10125 Verree Road
Suite 200
Philadelphia, PA 19116

John F. Kennedy Comm. MH/ MR Center Walk-In Clinic
112 Broad Street
Philadelphia, PA 19102

John F Kennedy Memorial Hospital Substance Abuse Services
Cheltenham Avenue and Langdon Street
Philadelphia, PA 19124

Kensington Hospital Alcohol and Drug Services
136 West Diamond Street
Philadelphia, PA 19122

Kensington Project
2907 Kensington Avenue
Philadelphia, PA 19134

Mercy Hospital of Philadelphia
5301 Cedar Avenue
Philadelphia, PA 19143

Methadone Maintenance
16th and Girard Avenue
Philadelphia, PA 19122

My Sisters Place
5601 Kingsessing Avenue
Philadelphia, PA 19143

Net
497 North 5th Street
Philadelphia, PA 19123

New Journeys in Recovery
2927 North 5th Street
Philadelphia, PA 19133

North Philadelphia Health System Girard Medical Center
Comprehensive Addictions Program
Dual Diagnosis Residential
Forensic Intensive Recovery Residence
Girard Avenue and 8 Street
Philadelphia, PA 19122

Northeast Treatment Centers Wharton Center
2205 Bridge Street
Philadelphia, PA 19125

NU Stop
2221-25 North Broad Street
2nd Floor
Philadelphia, PA 19132

Parkside Recovery
5000 Parkside Avenue
Philadelphia, PA 19131

Pennsylvania Hospital Hall Mercer Center
800 Spruce Street
Philadelphia, PA 19107-6192

Phase III Outpatient Counseling Services
555 East Indiana Avenue
Philadelphia, PA 19134

Philadelphia Consultation Center
313 South 16th Street
Philadelphia, PA 19102

Philadelphia Teen Challenge Women's Home
329 East Wister Street
Philadelphia, PA 19144

Presbyterian Medical Center
39th and Market Street
Philadelphia, PA 19104

R. W. Brown Community Center New Life Program
1701 North 8th Street
Philadelphia, PA 19122

Re-Enter, Inc.
3331 Powelton Avenue
Philadelphia, PA 19104

Rehab After Work
2821 Island Avenue, Suite 111
Philadelphia, PA 19153

15th Locust Street
Lewistower Building, Suite 201
Philadelphia, PA 19102

River's Bend Drug and Alcohol Unit
2401 Penrose Avenue
Philadelphia, PA 19145

Riverside House Inc
9549 Milnor Street
Philadelphia, PA 19114

Saint Gabriel's Hall Delasalle Aftercare
3509 Spring Garden Street
Philadelphia, PA 19104

Saint Gabriel System
117 South 17th Street
Suite 1701
Philadelphia, PA 19103

Saint Joseph Hospital
16th Street and Girard Avenue
Philadelphia, PA 19130

Self Help Movement, Inc.
2600 South Hampton Road
Philadelphia, PA 19116

Self, Inc.
121 North Broad Street
11th Floor
Philadelphia, PA 19107

Shalom, Inc.
311 South Juniper Street
Philadelphia, PA 19107

Sobriety Through Outpatient Inc
2221-25 North Broad Street
3rd Floor
Philadelphia, PA 19132

Teen Challenge Philadelphia Mens Home
156 West Schoolhouse Lane
Philadelphia, PA 19144

Therapeutic Center at Fox Chase
8400 Pine Road
Philadelphia, PA 19111

Veterans' Affairs Medical Center Alcohol/Drug Dependence Treatment Program
University and Woodland Avenues
Philadelphia, PA 19104

Wedge Medical Center
6701 North Broad Street
Philadelphia, PA 19126

1710 North 22nd Street
Philadelphia, PA 19122

2009 South Broad Street
Philadelphia, PA 19148

PHILIPSBURG

Quest Services
15th and Pine Streets
Philipsburg, PA 16866-9560

PHOENIXVILLE

Help Counseling Center
21 Gay Street
Phoenixville, PA 19460

PITTSBURGH

Abraxas Foundation, Inc.
Abraxas Center for Adolescent Females
437 Turrett Street
Pittsburgh, PA 15206

Abraxas III
936 West North Avenue
Pittsburgh, PA 15233

Addison Terrace Learning Center, Inc.
5937 Broad Street Mall
Suite 226-227
Pittsburgh, PA 15206

Alpha House
435 Shady Avenue
Pittsburgh, PA 15206

Alternative Program Associates, Inc.
6117 Broad Street
Pittsburgh, PA 15206

Center for Addiction Services Saint Francis Outreach
712 South Avenue
Pittsburgh, PA 15221

Charter Behavioral Health Systems Outpatient Recovery Center
2100 Wharton Street Birmingham Towers Suite 120
Pittsburgh, PA 15203

Circle C Specialized Group Home for Chemically Dependent Adolescents
227 Seabright Street
Pittsburgh, PA 15214

Discovery House
1391 Washington Boulevard
Pittsburgh, PA 15206

Gateway Allegheny Valley
1385 Old Freeport Road
Pittsburgh, PA 15238

Gateway Greentree
2121 Noblestown Road Rear
Pittsburgh, PA 15205

Greenbriar Robinson Township
4955 Steubenville Street
Twin Towers, Suite 303
Pittsburgh, PA 15205

Homewood/Brushton YMCA Counseling Services
7140 Bennett Street
Pittsburgh, PA 15208

House of the Crossroads
2012 Centre Avenue
Pittsburgh, PA 15219

Ielase Institute Mon Yough Corrections Program
232 First Avenue, 3rd Floor
Pittsburgh, PA 15222

Mercy Behavioral Health
2100 Wharton Street Suite 200
Pittsburgh, PA 15203-1942

OUR House ARTP
735 North Highland Avenue
Pittsburgh, PA 15206

PBA, Inc. Second Step Program
1425 Beaver Avenue
Pittsburgh, PA 15233

Pennsylvania Organization for Women in Early Recover
7445 Church Street
Pittsburgh, PA 15218

Program for Female Offenders, Inc. Allegheny County Treatment Alternative Program
2410 5th Avenue
Pittsburgh, PA 15213

Progressive Medical Specialists Inc
2900 Smallman Street
Pittsburgh, PA 15201

Saint Francis Medical Center
Center for Chemical Dependency Treatment
6714 Kelly Street
Pittsburgh, PA 15208

Outpatient/Inpatient Detox
400 45th Street
Pittsburgh, PA 15201-1198

Uptown Center
1945 5th Avenue
Pittsburgh, PA 15219

Salvation Army
Harbor Light Center
865 West North Avenue
Pittsburgh, PA 15233

Public Inebriate Program
54 South 9th Street
Pittsburgh, PA 15203

Sojourner House
5460 Penn Avenue
Pittsburgh, PA 15206

South Hills Health System Counseling Center
4129 Brownsville Road
Pittsburgh, PA 15227

Whale's Tale
Family Treatment Center
844 Proctor Way
Pittsburgh, PA 15210

Shadyside Office
250 Shady Avenue
Pittsburgh, PA 15206

Substance Abuse Treatment Services
413 Evergreen Avenue
Pittsburgh, PA 15209

801 Wallace Avenue
Pittsburgh, PA 15221

Veterans' Affairs Medical Center Substance Abuse Treatment Unit
7180 Highland Drive
Unit 116A3/5
Pittsburgh, PA 15206

PITTSTON

Wyoming Valley Alcohol and Drug Services, Inc.
49 South Main Street
Pittston, PA 18640

PORT ALLEGANY

Alcohol and Drug Abuse Services, Inc.
118 Chestnut Street
Port Allegany, PA 16743

120 Chestnut Street
Port Allegany, PA 16743-1251

POTTSTOWN

Addiction Counseling Services, Inc.
78 Savage Road
Pottstown, PA 19465

Alternative Counseling Associates
438-440 High Street
Pottstown, PA 19464

Creative Health Services, Inc.
Drug and Alcohol Outpatient
365 High Street
Pottstown, PA 19464

Creative Health Systems, Inc.
101 King Street
Pottstown, PA 19464

Programs in Counseling
262 King Street Suite 320
Pottstown, PA 19464-5571

QUAKERTOWN

Renewal Centers, Inc. Quakertown Office
2705 Old Bethlehem Pike
Quakertown, PA 18951

Saint Luke's Renewal Centers
2705 Old Bethlehem Pike
Quakertown, PA 18951

READING

Berks Counseling Center
700 Lancaster Street
Reading, PA 19602

Callowhill Family Therapy
244 North 5th Street
Reading, PA 19601

Center for Mental Health Drug and Alcohol Center
6 and Spruce Streets
Building J
Reading, PA 19611

Chor Youth and Family Services, Inc. Drug and Alcohol Center
1010 Centre Avenue
Reading, PA 19601

Hogar Crea Reading
302 South Fifth Street
Reading, PA 19602

Livengrin Counseling Center
Crestwood Street East
Hearthstone Court, Building 5
Reading, PA 19606

Pennsylvania Counseling Services
501 Washington Street, Suite 301
Reading, PA 19601

RED LION

Human Services Associates
424 South Pine Street
Red Lion, PA 17356

REHRERSBURG

Teen Challenge Training Center, Inc.
Teen Challenge Road
Rehrersburg, PA 19550

RIDGWAY

Nelson Behavioral Center
102 Center Street
Ridgway, PA 15853-1716

RURAL RIDGE

Teen Challenge of Western Pennsylvania
Lefever Hill Road
Rural Ridge, PA 15075

SAINT MARYS

Alcohol and Drug Abuse Services Saint Marys Unit
625 Maurus Street
Saint Marys, PA 15857

SALTSBURG

Adelphoi Village Keystone House
114 Washington Street
Saltsburg, PA 15681-1130

SAXTON

Twin Lakes Center for Drug and Alcohol Rehabilitation
805 Lower Main Street
Saxton, PA 16678

SCIOTA

Bethesda Day Treatment Center, Inc.
Business Route 209
Sciota, PA 18354

SCRANTON

A Better Today, Inc.
1339 North Main Avenue
Scranton, PA 18508

Drug and Alcohol Treatment Service, Inc. Outpatient Services
116 North Washington Avenue
3rd Floor
Scranton, PA 18503

Northeastern Pennsylvania Counseling Services
116 North Washington Avenue
Scranton, PA 18503

SELLERSVILLE

Community Service Foundation, Inc.
253 North Main Street
Sellersville, PA 18960

Penn Foundation, Inc. Recovery Center
807 Lawn Avenue
Sellersville, PA 18960

SHAMOKIN

Green Ridge Counseling Center Unit II
117 East Independence Street
Shamokin, PA 17872

SHARON

New Choices Sharon Regional Behavioral Health Services
740 East State Street
Sharon, PA 16146

SHARON HILL

Northwestern Human Services Delaware County/Life Guidance Division
800 Chester Pike
Sharon Hill, PA 19079

SHICKSHINNY

Clear Brook Lodge
RD 2
Shickshinny, PA 18655

SHIPPENSBURG

UHS PA Roxbury
601 Roxbury Road
Shippensburg, PA 17257

SHREWSBURY

Adams Hanover Counseling Services, Inc. Crossroads Counseling and Education Services
73 East Forest Avenue
Shrewsbury, PA 17361

SOMERSET

Twin Lakes Center for Drug/ Alcohol Rehabilitation
7 Byers Road
Somerset, PA 15501

SPARTANSBURG

Perseus House, Inc. Andromeda House II
39132 Mount Pleasant Road
Spartansburg, PA 16434

SPRING CITY

Creative Health Services, Inc.
1 Mennonite Church Road
Spring City, PA 19475

STATE COLLEGE

Counseling Alternatives Group
444 East College Avenue
Suite 300
State College, PA 16801

Counseling Service Drug and Alcohol Program
233 Easterly Parkway
State College, PA 16801

Lawrence T. Clayton and Counseling Associates, Inc.
230 South Fraser Street
State College, PA 16801

STROUDSBURG

Carbon/Monroe/Pike Drug/ Alcohol Commission Monroe County Clinic
Penn Square
724 Phillips Street, Suite A
Stroudsburg, PA 18360

SUNBURY

Green Ridge Counseling Center Unit V
1070 Market Street
Sunbury, PA 17801

Psychological Services Clinic
352 Arch Street
Sunbury, PA 17801

Valley Counseling Services
21 North 4th Street
Sunbury, PA 17801

TAMAQUA

Family Service Agency
37 West Broad Street
Tamaqua, PA 18252

TARENTUM

Saint Francis Medical Center
Office 3
400 Lock Street
Tarentum, PA 15084

TIONESTA

Forest/Warren Dept. of Human Services Alcohol and Drug Unit
Highland Street
Tionesta, PA 16353

TITUSVILLE

Crawford County Drug and Alcohol Executive Commission Incorporated
127 West Spring Street
Titusville, PA 16354

Deerfield Behavioral Health Network, Inc. Deerfield Centers of Addictions Treatment
605 North 1st Street
Titusville, PA 16354

TOWANDA

Mental Health Associates of North Central Pennsylvania/ Towanda
5 Lombard Street
Towanda, PA 18848

TREVOSE

Community Service Foundation, Inc.
3949 Brownsville Road
Trevose, PA 18901

TUNKHANNOCK

Catholic Social Services
Route 92
Tunkhannock, PA 18657

UNIONTOWN

Fayette County Drug and Alcohol Commission, Inc.
100 New Salem Road
Suite 106
Uniontown, PA 15401

UPPER DARBY

ChesPenn Health Services
20 South 69th Street, 2nd Floor
Upper Darby, PA 19082

Harwood House
9200 West Chester Pike
Upper Darby, PA 19082

VERONA

Whales Tale Penn Hills Office
6149 Saltsburg Road
Verona, PA 15147

WARMINSTER

Allegheny University Hospitals Bucks County
225 Newtown Road
Warminster, PA 18974

WARREN

Deerfield Behavioral Health of Warren Deerfield Centers of Addictions Treatment
414 Market Street
Warren, PA 16365

Forest/Warren Dept of Human Services Drug and Alcohol Program
27 Hospital Drive
Warren, PA 16365

WARRINGTON

Project Transition
1700 Street Road
Warrington, PA 18976

WASHINGTON

Abstinent Living at The Turning Point
199 North Main Street
Washington, PA 15301

Catholic Charities Diocese PGH Outpatient
331 South Main Street
Washington, PA 15301

Greenbriar Treatment Center
800 Manor Drive
Washington, PA 15301

Try Again Homes, Inc.
365 Jefferson Avenue
Washington, PA 15301-4245

WAVERLY

Marworth
Lily Lake Road
Waverly, PA 18471

WAYNESBORO

Roxbury Treatment Center
40 West North Street
Waynesboro, PA 17268-1257

WELLSBORO

Harbor Counseling
25 Water Street
Wellsboro, PA 16901

Laurel Behavioral Health
32-36 Central Avenue
Wellsboro, PA 16901

Laurel Health Center
15 Meade Street Suite L4/6
Wellsboro, PA 16901

Mental Health Associates of North Central Pennsylvania
68 Main Street
Wellsboro, PA 16901

WERNERSVILLE

Caron Foundation Treatment Services
Galen Hall Road
Wernersville, PA 19565

WEST CHESTER

Gaudenzia House
1030 South Concord Road
West Chester, PA 19382

Kindred House for Women and Children
1030 South Concord Road
West Chester, PA 19380

NHS Help Counseling Division
790 East Market Street, Suite 300
West Chester, PA 19382

WEST READING

Center for Mental Health Care
6th and Spruce Streets
Building 1C
West Reading, PA 19611

Jeter Counseling Services
529 Reading Avenue
West Reading, PA 19611

New Directions Treatment Services
20-22 North 6th Avenue
West Reading, PA 19611

WEXFORD

Mercy Behavioral Health
9983 Perry Highway
Wexford, PA 15090

WILKES-BARRE

Catholic Social Services
33 East Northampton Street
Wilkes-Barre, PA 18701

Clear Brook Lodge
1100 East Northampton Street
Wilkes-Barre, PA 18702

Family Service Association of Wyoming Valley
31 West Market Street
Wilkes-Barre, PA 18701

Ferrell and Associates, Inc.
111 North Franklin Street
Wilkes-Barre, PA 18701

First Hospital Wyoming Valley Adult II Dual Diagnosis
149 Dana Street
Wilkes-Barre, PA 18702

Veterans' Affairs Medical Center Substance Abuse Treatment Unit
1111 East End Boulevard
Wilkes-Barre, PA 18711

Wyoming Valley Alcohol and Drug Services, Inc.
437 North Main Street
Wilkes-Barre, PA 18705

WILLIAMSPORT

Crossroads Counseling, Inc.
2128 west 4th Street
Williamsport, PA 17701

Genesis House Inc Professional Counseling Services
1247 West 4th Street
Williamsport, PA 17701

Green Ridge Counseling Center Unit III
520 West 4 Street
Williamsport, PA 17701

White Deer Run of Williamsport
915 Vine Avenue
Williamsport, PA 17701

WILLOW GROVE

Health Care Options, Inc.
500 North Easton Road, 2nd Floor
Willow Grove, PA 19090

WYOMISSING

Caron Counseling Services
845 Park Road
Wyomissing, PA 19610

WYOMISSING HILLS

Pennsylvania Counseling Services
1733 Penn Avenue
Wyomissing Hills, PA 19609

YORK

Atkins House
313 East King Street
York, PA 17403

Colonial House Inc
1300 Woodbury Road
York, PA 17405

Craig and Associates
3550 Concord Road
York, PA 17402-8626

Family and Community Health Associates
810 Bonneview Road
York, PA 17402

1689 Kenneth Road, Suite 202
York, PA 17403

25 Monument Road
York, PA 17403

1030 Plymouth Road
York, PA 17402

New Insights, Inc.
707 Loucks Road
York, PA 17404

Stepping Stone Counseling and Education Services, Inc.
1776 South Queen Street
York, PA 17043

211 South George Street
York, PA 17403

Susquehanna Counseling
2300 East Market Street, Suite 4
York, PA 17402-2858

York Area Counseling Services
26 Mount Zion Road
York, PA 1740

RHODE ISLAND

CHARLESTOWN

South Shore Mental Health Center Addiction Program
4705A Old Post Road
Charlestown, RI 02813

CRANSTON

Addiction Services Comprehensive Community Action Program
311 Doric Avenue
Cranston, RI 02910

Eastman House, Inc.
1545 Pontiac Avenue
Cranston, RI 02920

SSTAR of Rhode Island, Inc. Birth
80 East Street
Cranston, RI 02920

EAST PROVIDENCE

East Bay Mental Health Center Substance Abuse Services
610 Wampanoag Trail
East Providence, RI 02914

EXETER

Marathon House
Exeter, RI 02822

JOHNSTON

Center for Behavioral Health
985 Plainfield Street
Johnston, RI 02919

Tri-Town Community Action Agency
1126 Hartford Avenue
Johnston, RI 02919-7130

MIDDLETOWN

Child and Family Services of Newport
19 Valley Road
Middletown, RI 02840

Newport County Community Mental Health Center
127 Johnnycake Hill Road
Middletown, RI 02842

NARRAGANSETT

Galilee Mission to Fishermen, Inc.
268 Kingstown Road
Narragansett, RI 02882

NEWPORT

New Visions of Newport County SSTAR
19 Broadway
Newport, RI 02840

NORTH KINGSTOWN

Meadows Edge
580 Ten Rod Road
North Kingstown, RI 02852-4220

SSTAR of Rhode Island Residential Alcohol/Drug Detox
1950 Tower Hill Road
North Kingstown, RI 02852

PASCOAG

Marathon, Inc. The Lodge at Wallum Lake
2198 Wallum Lake Road
Pascoag, RI 02859

Long-Term Care
2198 Wallum Lake Road
Pascoag, RI 02859

PAWTUCKET

Community Counseling Center, Inc.
101 Bacon Street
Pawtucket, RI 02860

Friends of Caritas House, Inc.
166 Pawtucket Avenue
Pawtucket, RI 02860

Robert J. Wilson House, Inc.
Outpatient Counseling Center
Residential
80 Summit Street
Pawtucket, RI 02860

Tri Hab House, Inc. Pawtucket Addictions Counseling Services
104 Broad Street
Pawtucket, RI 02860

PROVIDENCE

Alcohol and Drug Rehab Services, Inc.
Minority Alcohol Prog Outpatient Counseling
66 Burnett Street
Providence, RI 02905

Butler Hospital Alcohol and Drug Treatment Service
345 Blackstone Boulevard
Providence, RI 02906

Discovery House South Providence Addiction Center
66 Pavillion Avenue
Providence, RI 02905

Discovery Program
520 Hope Street
Providence, RI 02906

Family Service Incorporated Substance Abuse Program
55 Hope Street
Providence, RI 02906

Marathon House, Inc.
Outpatient
131 Wayland Avenue
Providence, RI 02906

Multicultural Counseling Center
280 Broadway Street Suite 100
Providence, RI 02903

Providence Community Action
Division of Clinical Services
662 Hartford Avenue
Providence, RI 02909

Divison of Clinical Services
16 Borinquen Street
Providence, RI 02905

SSTAR of Rhode Island, Inc.
Residential Alcohol and Drug Detox
Short Term Residential Program for Pregnant Women
21 Peace Street
Providence, RI 02907

Talbot Residential Services
Talbot Outpatient
Womens Day Treatment
90 Plain Street
Providence, RI 02903

265 Oxford Street
Providence, RI 02905

WAKEFIELD

Marathon Sympatico
57 Columbia Street
Wakefield, RI 02879

WARWICK

Addiction Recovery Institute South
205 Helene Road Suite 102
Warwick, RI 02886

Kent County Mental Health Center Alcohol/Drug and Family Counseling
300 Centerville Road Suite 301-S
Warwick, RI 02886

Kent County Mental Health Center
50 Health Lane
Warwick, RI 02886

Kent House, Inc.
2020 Elmwood Avenue
Warwick, RI 02888

Mental Health Services Counseling and Intervention Services, Inc.
422-A Post Road
Warwick, RI 02886

WEST WARWICK

Directions
1071 Main Street
West Warwick, RI 02893

WOONSOCKET

Discovery House Woonsocket
1625 Diamond Hill Road
Woonsocket, RI 02895

Family Resources, Inc.
245 South Main Street
Woonsocket, RI 02895

Tri-Hab Counseling
58 Hamlet Avenue
Woonsocket, RI 02895

Tri-Hab House, Inc.
79 Asylum Street
Woonsocket, RI 02895

King House
80 Hamlet Avenue
Woonsocket, RI 02895

WYOMING

Friends of Caritas House, Inc. Corkery House
15 Baker Pines Road
Wyoming, RI 02898-1000

SOUTH CAROLINA

AIKEN

Aiken Center
1105 Gregg Highway
Aiken, SC 29801-0535

Aiken Regional Medical Center Aurora Pavilion
655 Medical Park Drive
Aiken, SC 29801

ANDERSON

Anderson/Oconee Counties Behavioral Health Services
226 McGee Road
Anderson, SC 29625

Patrick B. Harris Hospital Substance Abuse Services
130 Highway 252
Anderson, SC 29621

BARNWELL

Axis I Center of Barnwell
2606 Jackson Avenue
Barnwell, SC 29812

BEAUFORT

Beaufort County Alcohol and Drug Abuse Department
1905 Duke Street
Suite 270
Beaufort, SC 29902

Coastal Empire Community Mental Health Center Substance Abuse Services
1050 Ribaut Road
Beaufort, SC 29901

Joint Substance Abuse Counseling Center
Marine Corps Air Station MCAS
Beaufort, SC 29904

MCRD Parris Island South Carolina Substance Abuse Counseling Center
Building 911
Beaufort, SC 29905-5001

BISHOPVILLE

Lee County Commission on Alcohol and Drug Abuse
180 East Church Street
Bishopville, SC 29010

CAMDEN

Alpha Center
416 Rutledge Street
Camden, SC 29020

CHARLESTON

Columbia/Trident Behavioral Health Services
9225 University Boulevard
Suite 2-E
Charleston, SC 29406

Department of Alcohol and Other Drug Abuse Services of Charleston County
615 Wesley Drive
Charleston, SC 29417-1398

Medical University of South Carolina Drugs and Alcohol Program
171 Ashley Avenue
Charleston, SC 29425

Roper North Treatment Center
2750 Speissegger Drive
Charleston, SC 29405

Veterans' Affairs Medical Center Substance Abuse Treatment Center
100 Bee Street
Charleston, SC 29401

CHESTER

Hazel Pittman Center
130 Hudson Street
Chester, SC 29706

CHESTERFIELD

Alpha Center/Chesterfield/Kershaw/Lee Alcohol and Drug Abuse Commission
141 West Main Street
Chesterfield, SC 29709

COLUMBIA

Columbia Area Mental Health Center
1611 Devonshire Drive
Columbia, SC 29204

Earle E. Morris, Jr. Alcohol and Drug Addiction Treatment Center
610 Faison Drive
Columbia, SC 29203

Lexington/Richland Alcohol and Drug Abuse Council
1325 Harden Street
Columbia, SC 29204

Richland Springs Psychiatric Hospital
11 Medical Park
Columbia, SC 29203

CONWAY

Charter Sands Behavioral Health System
152 Waccamaw Medical Park Drive
Conway, SC 29526

Horry County Commission on Alcohol and Drug Abuse
1004 Bell Street
Conway, SC 29526

DILLON

Dillon County Commission on Alcohol and Drug Abuse
204 North Third Avenue
Dillon, SC 29536

FLORENCE

Bruce Hall
122 East Cedar Street
Florence, SC 29501

Palmetto Center
Florence, SC 29502

GAFFNEY

Cherokee County Commission on Alcohol and Drug Abuse
201 West Montgomery Street
Gaffney, SC 29341

GEORGETOWN

Georgetown County Alcohol and Drug Abuse Commission
1423 Winyah Street
Georgetown, SC 29440

GREENVILLE

Addcare Counseling, Inc.
11 Pointe Circle
Greenville, SC 29615

Addlife Addiction Services
701 Grove Road
Greenville, SC 29605

Don Foster and Associates, Inc.
104 Mills Avenue
Greenville, SC 29605

Greenville Metro Treatment Center
603 Arlington Avenue
Greenville, SC 29601

Greenville County Commission on Alcohol and Drug Abuse
3336 Buncombe Road
Greenville, SC 29609

Healthy Beginnings
730 South Pleasantburg Drive
Suite 109
Greenville, SC 29607

Holsmesview Center
Old Easley Bridge Road
Greenville, SC 29610

Rosewood House of Recovery, Inc.
9 Renrick Drive
Greenville, SC 29609

GREENWOOD

Faith Home Christian Alcohol and Drug Rehab
Buck Level Road
Greenwood, SC 29646

Greenwood/Edgefield/ McCormick Commission on Alcohol and Drug Abuse
1420 Spring Street
Greenwood, SC 29646

HAMPTON

New Life Center
First Street East
Second Floor, Annex Building
Hampton, SC 29924

LANCASTER

Springs Memorial Hospital Lancaster Recovery Center
800 West Meeting Street
Lancaster, SC 29720

LAURENS

Laurens Commission on Alcohol and Drug Abuse
Industrial Park Road
Laurens, SC 29360

MANNING

Clarendon County Commission on Alcohol and Drug Abuse
14 North Church Street
Manning, SC 29102

MARION

Marion County Alcohol and Drug Abuse Program
103 Court Street
Marion, SC 29571

MONCKS CORNER

Ernest E. Kennedy Center Alcohol and Drug Abuse Program
306 Airport Drive
Moncks Corner, SC 29461

NEWBERRY

ORANGEBURG

Westview Behavioral Health Services
800 Main Street
Newberry, SC 29108

Tri-County Commission on Alcohol and Drug Abuse
3190 Cook Road
Orangeburg, SC 29115

SALUDA

Saluda County Commission on Alcohol and Drug Abuse
204 Ramage Street
Saluda, SC 29138

Shaw Air Base 20th Medical Operations Squadron (SGOMH)
423 Lowry Avenue
Shaw AFB, SC 29152

SPARTANBURG

Spartanburg Area Mental Health Center Substance Abuse Services
149 East Wood Street
Spartanburg, SC 29303

Spartanburg County Commission on Alcohol and Drug Abuse
209 Catawba Street
Spartanburg, SC 29306

SUMMERVILLE

Dorchester County Commission on Alcohol and Drug Abuse
500 North Cedar Street
Summerville, SC 29483

UNION

Union County Commission on Alcohol and Drug Abuse
201 South Herndon Street
Union, SC 29379

WALTERBORO

Colleton County Commission on Alcohol and Drug Abuse
Walterboro, SC 29488

WEST COLUMBIA

Charter Rivers Hospital Alcohol and Drug Abuse Services
2900 Sunset Boulevard
West Columbia, SC 29169

Columbia Metro Treatment Center
421 Capital Square
West Columbia, SC 29169

WILLIAMSTON

Anmed Wellspring
313 William Street
Williamston, SC 29697

WINNSBORO

Fairfield County Substance Abuse Commission
200 Calhoun Street
Winnsboro, SC 29180

SOUTH DAKOTA

ABERDEEN

Northern Alcohol/Drug Referral and Information Center (NADRIC)
221 South First Street
Aberdeen, SD 57402

BELLE FOURCHE

Addiction Family Resources
608 5th Avenue
Belle Fourche, SD 57717

BERESFORD

Lutheran Social Services of South Dakota Woodfield Center
Beresford, SD 57004

BROOKINGS

East Central Mental Health Chemical Dependency Center
211 4 Street
Brookings, SD 57006

CANTON

Keystone Treatment Center
1010 East 2 Street
Canton, SD 57013

CHAMBERLAIN

Chamberlain Academy
211 West 16 Avenue
Chamberlain, SD 57325

HOT SPRINGS

Southern Hills Alcohol/Drug Referral Center
311 North River Street
Hot Springs, SD 57747

HURON

Community Counseling Services Alcohol and Drug Unit
1552 Dakota Street South
Huron, SD 57350

Our Home, Inc.
Rediscovery
360 Ohio Avenue NW
Huron, SD 57350

Inhalant Abuse Program
East Centennial Road
Huron, SD 57350

LEMMON

Three Rivers Chemical Dependency Center
11 East 4th Street
Lemmon, SD 57638

MADISON

Community Counseling Services
914 NE 3 Street
Madison, SD 57042

MITCHELL

Abbott House
909 Court Merrill Street
Mitchell, SD 57301-0700

Community Alcohol/Drug Center, Inc.
901 South Miller Street
Mitchell, SD 57301

PIERRE

Capital Area Counseling Service, Inc. Drug and Alcohol Unit
200 West Pleasant Street
Pierre, SD 57501

PLANKINTON

State Training School Alcohol and Drug Program
Plankinton, SD 57368

RAPID CITY

City/County Receiving and Referral Center
725 North Lacrosse Street
Rapid City, SD 57701

Focus, Inc.
114 Kinney Avenue
Rapid City, SD 57709

Youth and Family Counseling Services
924 North Maple Street
Rapid City, SD 57709

SIOUX FALLS

Carroll Institute
2nd Street Manor
826 West 2 Street
Sioux Falls, SD 57104

Outpatient Alcohol and Drug Center
310 South 1st Avenue
Sioux Falls, SD 57102

Arch Halfway House
Sioux Falls Detoxification Center
333 South Spring Avenue
Sioux Falls, SD 57104

Communication Services for The Deaf
3520 Gateway Lane
Sioux Falls, SD 57106-1558

Counseling Resources
707 East 41st Street
Suite 205
Sioux Falls, SD 57105-6405

First Step Counseling Services
4320 South Louise Street
Sioux Falls, SD 57106

Glory House of Sioux Falls
4000 South West Avenue
Sioux Falls, SD 57105

J. W. Doan and Associates Behavioral Health Services
625 South Minnesota Avenue
Suite 102
Sioux Falls, SD 57104-4872

Keystone Outreach Program
1908 West 42 Street
Sioux Falls, SD 57105

McKennan Behavioral Health Services Addiction Recovery Program
3926 South Western Avenue
Sioux Falls, SD 57101

Sioux Valley Hospital Behavioral Health Services
2812 South Louise Avenue
Sioux Falls, SD 57106

South Dakota State Penitentiary Alcohol and Drug Program
1600 North Drive
Sioux Falls, SD 57117

Turning Point
1401 West 51st Street
Sioux Falls, SD 57105

SISSETON

Tetakwitha Adolescent Treatment Center
Rural Route 2
Sisseton, SD 57262

SPRINGFIELD

Springfield State Prison Chemical Dependency Program
Springfield, SD 57062

YSI/Springfield Academy
709 6th Street
Springfield, SD 57062

STURGIS

Black Hills Special Services Cooperative Chemical Dependency Inpatient Program
1715 Lazelle Street
Sturgis, SD 57785

Northern Hills Alcohol and Drug Service
950 Main Street
Sturgis, SD 57785

VALE

New Dawn Center
Rural Route 1
Vale, SD 57788

VERMILLION

University of South Dakota Student Counseling Center
414 East Clark Street
Vermillion, SD 57069

WINNER

Southern Plains Mental Health Center Decision 1
500 East 9 Street
Winner, SD 57580

Winner Alcohol/Drug Counseling Service
223 South Main Street
Winner, SD 57580

YANKTON

Adolescent Chemical Dependency Program
North Highway 81
Yankton, SD 57078

South Dakota Human Services Center
Gateway Chemical Dependency Treatment Center
3515 Broadway
Yankton, SD 57078

TENNESSEE

ASHLAND CITY

Centerstone Mental Health Centers Harriett Cohn Center/ Ashland City
197 Court Street
Ashland City, TN 37015

ATHENS

Volunteer Counseling Center Hiwassee
1805 Ingleside Avenue
Athens, TN 37303

BEAN STATION

Cherokee Health System
Highway 11 West
Bean Station, TN 37708

BLAINE

Cherokee Health System
180 Emory Road
Blaine, TN 37709

BOLIVAR

Quinco Mental Health Center Alcohol and Drug Services
10710 Highway 64
Bolivar, TN 38008

BRISTOL

Bristol Residential Counseling Center
26 Midway Street
Bristol, TN 37620

CASTALIAN SPRINGS

Pathfinders Residential Treatment Center
875 Highway 231 South
Castalian Springs, TN 37031

CENTERVILLE

Centerstone Mental Health Centers Family Counseling and Mental Health Center
1680 Highway 100
Centerville, TN 37033

CHATTANOOGA

Council for Alcohol and Drug Abuse Services
207 Spears Avenue
Chattanooga, TN 37405

911 Pineville Road
Chattanooga, TN 37405

Fortwood Center, Inc.
1028 East 3 Street
Chattanooga, TN 37403

Parkridge Hospital, Inc. DBA Columbia Valley Hospital
2200 Morris Hill Road
Chattanooga, TN 37421

Volunteer Treatment Center, Inc.
2347 Rossville Boulevard
Chattanooga, TN 37408

CLARKSVILLE

Harriett Cohn Mental Health Center
511 8 Street
Clarksville, TN 37040

CLEVELAND

Greenleaf Outpatient Services
2650 Executive Park North
Suite 2
Cleveland, TN 37312

Volunteer Behavioral Healthcare System
Hiwassee
1855 Executive Park NW
Cleveland, TN 37312-2747

Reality House
360 Worth Street
Cleveland, TN 37311

COLUMBIA

Centerstone Mental Health Centers Maury County Mental Health Center
1222 Medical Center Drive
Columbia, TN 38401

COOKEVILLE

Volunteer Behavioral Healthcare System
1200 South Willow Avenue
Cookeville, TN 38501

COVINGTON

Professional Counseling Services, Inc. Alcohol and Drug Abuse Services
1997 Highway 51 South
Covington, TN 38019

CROSSVILLE

Volunteer Behavioral Healthcare System
Cumberland Mountain Unit
Route 13
Crossville, TN 38555

DANDRIDGE

Cherokee Health System
809 Peal Street
Dandridge, TN 37725

DAYTON

Volunteer Behavioral Healthcare System RHEA County Mental Health Center
7200 Rhea County Highway
Dayton, TN 37321

DECATURVILLE

Quinco Mental Health Center Alcohol and Drug Services
Highway 100
Decaturville, TN 38329

DICKSON

Centerstone Mental Health Centers Southridge Psychological Services
721 Highway 46
Dickson, TN 37055

FAYETTEVILLE

Centerstone Mental Health Centers Highland Rim Mental Health Center
2241 Thornton Taylor Parkway
Fayetteville, TN 37334

GALLATIN

Cumberland Mental Health Services, Inc. Alcohol and Drug Program
528 East Main Street
Gallatin, TN 37066

GREENEVILLE

Frontier Health, Inc. Church Street Pavilion
616 East Church Street, Suite A
Greeneville, TN 37743

Nolachuckey/Holston Mental Health Center
401 Holston Drive
Greeneville, TN 37744

HARRIMAN

Ridgeview Psychiatric Hospital and Center Alcohol/Drug Abuse Program
221 Devonia Street
Harriman, TN 37748

HENDERSON

Quinco Mental Health Center Alcohol and Drug Services
925 East Main Street
Henderson, TN 38340

HENDERSONVILLE

Cumberland Mental Health Services, Inc. Alcohol and Drug Program
133 Indian Lake Road
Hendersonville, TN 37075

HOHENWALD

Buffalo Valley, Inc.
221 South Maple Street
Hohenwald, TN 38462

501 Park Avenue South
Hohenwald, TN 38462

511 Park Avenue South
Hohenwald, TN 38462

Centerstone Mental Health Centers
912 Summertown Highway
Hohenwald, TN 38462-0513

JACKSON

Aspell Manor
331 North Highland Avenue
Jackson, TN 38301

Charter Lakeside/Jackson
106 Stonebridge Boulevard
Jackson, TN 38305

Jackson Area Council on Alcoholism and Drug Dependency
900 East Chester Street
Jackson, TN 38301

Pathways Substance Abuse Treatment Center
238 Summar Drive
Jackson, TN 38301

JASPER

Volunteer Behavioral Healthcare System Marion City Mental Health Center
443 Browder Switch Road
Jasper, TN 37347-0610

JOHNSON CITY

Comprehensive Community Services
323 West Walnut Street
Johnson City, TN 37604

Frontier Health, Inc.
Fairview Associates
607 Baxter Street
Johnson City, TN 37604

Watauga Mental Health Center
106 East Watauga Avenue
Johnson City, TN 37605

Woodridge Hospital
403 State of Franklin Road
Johnson City, TN 37604

Recovery North Side Hospital Chemical Dependency Unit
401 Princeton Road
Johnson City, TN 37601

KINGSPORT

Frontier Health, Inc. Holston Children and Youth Services
2001 Stonebrook Place
Kingsport, TN 37660

Indian Path Hospital
2000 Pavilion Drive
Kingsport, TN 37660

KNOXVILLE

Agape, Inc. Halfway House
205-211-215 East Scott Avenue
Knoxville, TN 37917

Baptist Hospital of East Tennessee
137 Blount Avenue
Knoxville, TN 37920

Center Point Adult Services
3510 Ball Camp Pike
Knoxville, TN 37921

Cherokee Health System
10263 Kingston Pike
Knoxville, TN 37922

Child and Family Services Great Starts
2601 Keith Avenue
Knoxville, TN 37921

DRD Knoxville Medical Clinic
1501 Cline Street
Knoxville, TN 37921

E. M. Jellinek Center
130 Hinton Street
Knoxville, TN 37917

Florence Crittenton Agency Outpatient/Pregnant Substance Abuse Program
1531 Dick Lonas Road
Knoxville, TN 37909

Helen Ross McNabb Center, Inc. Alcohol and Drug Program
1520 Cherokee Trail
Knoxville, TN 37920

1310 Oldham Avenue
Apartment 283
Knoxville, TN 37921

Outpatient
5310 Ball Camp Pike
Knoxville, TN 37921

Centerpointe
Adolescent Services
412 Citico Street
Knoxville, TN 37919

Knox County Detoxification
5908 Lyons View Drive
Knoxville, TN 37919

Knoxville Knox County Community Action Com. Counseling and Recovery Services
2247 Western Avenue
Knoxville, TN 37921

Midway Rehabilitation Center
1715 Magnolia Avenue
Knoxville, TN 37927

Overlook Center, Inc. Alcohol and Drug Abuse Services
3001 Lake Brook Boulevard
Knoxville, TN 37909

Peninsula Lighthouse
6800 Baum Drive NW
Knoxville, TN 37919

LAFAYETTE

Volunteer Behavioral Healthcare System Valley Ridge Unit
212 Public Square
Lafayette, TN 37083

LA FOLLETTE

Ridgeview Psychiatric Hospital and Center Alcohol/Drug Abuse Program
500 West Central Avenue
La Follette, TN 37766

LAWRENCEBURG

Centerstone Mental Health Centers Lawrence County Counseling
1090 Old Florence Road
Lawrenceburg, TN 38464

LEBANON

Cumberland Mental Health Services Drug and Alcohol Program
1404 Winter Drive
Lebanon, TN 37087

LEWISBURG

Buffalo Valley, Inc.
218 Martin Avenue South
Lewisburg, TN 37091

Centerstone Mental Health Centers Marshall County Mental Health Center
1221 Nashville Highway
Lewisburg, TN 37091

LEXINGTON

Turning Point
107 East Church Street
Lexington, TN 38351

LIVINGSTON

Dale Hollow Mental Health Center
501 Spruce Street
Livingston, TN 38570

LOUISVILLE

Peninsula Hospital Chemical Dependency Program
2347 Jones Bend Road
Louisville, TN 37777

MADISON

Dede Wallace Center Alcohol and Drug Program
620 Gallatin Road South
Madison, TN 37115

MADISONVILLE

Overlook Mental Health Center, Inc. Alcohol and Drug Abuse Services
100 Main Street
Madisonville, TN 37354

MANCHESTER

Bradford Health Services Manchester Outreach
1601 McArthur Street
Manchester, TN 37355

MARTIN

Baptist Memorial Hospital Behavioral Health Care
1201 Bishop Street
Martin, TN 38261

MARYVILLE

Blount Memorial Hospital Mountain View Recovery Center
907 East Lamar Alexander Parkway
Maryville, TN 37801

Overlook Mental Health Center, Inc. Alcohol and Drug Abuse Services
219 Court Street
Maryville, TN 37801

MAYNARDVILLE

Cherokee Health System
4330 Maynardville Highway
Maynardville, TN 37807

MCMINNVILLE

Cheer
120 Omni Drive
McMinnville, TN 37110

MEMPHIS

Baby Love
450 Pontotoc Street
Memphis, TN 38126

Charter Lakeside Behavioral Health System Dual Diagnosis Unit
2911 Brunsuick Road
Memphis, TN 38133-4199

Cocaine and Alcohol Awareness Program (CAAP)
1347 Ferguson Street
Memphis, TN 38106

Frayser/Millington Mental Health Center Alcohol and Drug Abuse Services
2150 Whitney Avenue
Memphis, TN 38127

Grace House, Inc.
329 North Bellevue Street
Memphis, TN 38105

Harbor House, Inc. Alcoholic Rehabilitation Center
1979 Alcy Road
Memphis, TN 38114

John A. Scott Sr. and Associates
5628 Murray Road, Suite 4
Memphis, TN 38119

Memphis Alcohol and Drug Council Prevention/Education
1430 Poplar Street
Memphis, TN 38104

Memphis City Schools Mental Health Center Substance Abuse Services
3782 Jackson Avenue
Room 102
Memphis, TN 38112

Memphis Mental Health Institute Substance Abuse Treatment Program
865 Poplar Avenue
Memphis, TN 38105

Memphis Recovery Centers, Inc.
219 North Montgomery Street
Memphis, TN 38104

1172 Vance Avenue
Memphis, TN 38104

Methodist Hospital
2009 Lamar Avenue
Memphis, TN 38114

Mid-Town Mental Health Center Alcohol and Drug Abuse Services
427 Linden Avenue
Memphis, TN 38126

New Directions, Inc.
642 Semmes Street
Memphis, TN 38111

Raleigh Professional Associates
2960-B Austin Peay Highway
Memphis, TN 38128

Saint Francis Hospital Addiction Treatment Program
5959 Park Avenue
Memphis, TN 38119

Serenity Houses Recovery Center
1094 Poplar Avenue
Memphis, TN 38105

Southeast Mental Health Center, Inc. Alcohol/Drug Abuse Program
3810 Winchester Road
Memphis, TN 38118

2579 Douglas Street
Memphis, TN 38114

3268 Summer Avenue
Memphis, TN 38122

Synergy Foundation, Inc.
2305 Airport Interchange
Memphis, TN 38132

Veterans' Affairs Medical Center Psychiatry Services/Alcohol/Drug Dependency Treatment Program
1030 Jefferson Avenue
Memphis, TN 38104

Whitehaven/Southwest Mental Health Center Alcohol and Drug Abuse Program
1087 Alice Avenue
Memphis, TN 38116

MORRISTOWN

Cherokee Mental Health Center Substance Abuse Treatment Program
815 West 5 North Street
Morristown, TN 37814

MOUNTAIN CITY

Frontier Johnson Community Counseling Center
318 Donnelly Street
Mountain City, TN 37683

MOUNTAIN HOME

Quillen, James H., VAMC
Mountain Home, TN 37684

MURFREESBORO

Alvin C. York VA Medical Center Substance Abuse Rehabilitation Program
3400 Lebanon Road
Murfreesboro, TN 37130

Pathfinders, Inc. Murfreesboro Outpatient Center
815 South Church Street, Suite 100
Murfreesboro, TN 37130

NASHVILLE

Bradford Health Services Nashville Outreach
2525 Perimeter Place Drive
Suite 110
Greenbriar Business Park
Nashville, TN 37214

Centerstone Mental Health Centers Luton Mental Health Services
1921 Ransom Place
Nashville, TN 37217

Cumberland Heights Alcohol and Drug Treatment
8283 River Road
Nashville, TN 37209

Davidson County Sheriff's Office New Avenues/Save
5115 Harding Place
Nashville, TN 37201

Life Challenge of Nashville Women's Residence
1017 Burchwood Avenue
Nashville, TN 37216

Lloyd C. Elam Mental Health Center Meharry Alcohol and Drug Abuse Program
1005 Dr. David B. Todd, Jr., Boulevard
Nashville, TN 37208

Metro Health Department Chemical Dependency Program
526 8th Avenue South
Nashville, TN 37203

Nashville Union Rescue Mission
129 7th Avenue South
Nashville, TN 37203

Parthenon Pavilion CMC Dual Treatment Program
2401 Murphy Avenue
Nashville, TN 37203

Psychiatric Hospital at Vanderbilt
1601 23rd Avenue South
Nashville, TN 37212

Samaritan Recovery Community, Inc.
319 South 4th Street
Nashville, TN 37206

Veterans Affairs Medical Center Substance Abuse Treatment Program
1310 24th Avenue South
Nashville, TN 37212-2637

OAK RIDGE

Hope of East Tennessee, Inc.
171 Waddell Circle
Oak Ridge, TN 37830

Methodist Medical Center Turning Point Recovery Center
990 Oak Ridge Turnpike
Oak Ridge, TN 37830

Ridgeview Psychiatric Hospital and Center Alcohol/Drug Abuse Program
240 West Tyrone Road
Oak Ridge, TN 37830

NEWPORT

Cherokee Health System
132 West Broadway Street
Newport, TN 37821

NEW TAZEWELL

Cherokee Health System
606 Broad Street
New Tazewell, TN 37825

OLD HICKORY

Torch Counseling Services
1053 Donelson Avenue
Old Hickory, TN 37138

ONEIDA

Scott County Hospital Recovery Center
Alberta Avenue
Oneida, TN 37841

PARIS

Carey Counseling Center Alcohol and Drug Abuse Program
408 Virginia Avenue
Paris, TN 38242

POWELL

Cherokee Health System
207 East Emory Road
Powell, TN 37849

RIPLEY

Baptist Memorial Hospital of Lauderdale County
326 Asbury Road
Ripley, TN 38063

ROGERSVILLE

Frontier Health, Inc. Hawkins County Mental Health Clinic
101 Lena Drive
Rogersville, TN 37857

SAVANNAH

Care of Savannah, Inc. Jack Gean Shelter for Women
Route 3
Savannah, TN 38372

Quinco Mental Health Center Alcohol and Drug Services
1105 Pickwick Road
Savannah, TN 38372

SELMER

Quinco Mental Health Center Alcohol and Drug Services
641 East Poplar Street
Selmer, TN 38375

SEVIERVILLE

Overlook Mental Health Center, Inc. Alcohol and Drug Abuse Services
124 North Henderson Avenue
Sevierville, TN 37862

SEYMOUR

Cherokee Health System
10341 Chapman Highway, Suite 3
Seymour, TN 37865

SHELBYVILLE

Centerstone Mental Health Centers Highland Rim Mental Health Center
712 North Main Street
Shelbyville, TN 37160

Tony Rice Center, Inc.
1300 Railroad Avenue
Shelbyville, TN 37160

SMITHVILLE

Wood, Deborah SM., and Carlton G. Wood, Ph.D.
Highway 70
Smithville, TN 37166

SNEEDVILLE

Frontier Health, Inc. Hancock County Mental Health Clinic
Buck Valley Road
Sneedville, TN 37869

SPRINGFIELD

Centerstone Mental Health Centers Harriett Cohn Center/ Springfield
713 Cheatharn Street
Springfield, TN 37172

TALBOTT

Cherokee Health Systems Substance Abuse Treatment Program
6350 West Andrew Johnson Highway
Talbott, TN 37877

TAZEWELL

Cherokee Health System
1409 Old Tazewell Road
Tazewell, TN 37879

TENNESSEE RIDGE

Centerstone Mental Health Centers Ridgeview Residential and Center Offices
Route 1 Box 107
Main Street-Highway 147
Tennessee Ridge, TN 37178

TRENTON

Carey Counseling Center
200 East Eaton Street
Trenton, TN 38382

TULLAHOMA

Highland Rim Mental Health Center
1803 North Jackson Street
Tullahoma, TN 37388

WAVERLY

Centerstone Mental Health Centers River Valley Psychological Services
811 East Railroad Street
Waverly, TN 37185

WAYNESBORO

Centerstone Mental Health Centers Wayne County Mental Health Center
Highway 135 South T
Waynesboro, TN 38485

WINCHESTER

Centerstone Mental Health Centers Highland Rim Mental Health Center
10 South Cedar Street
Winchester, TN 37398

TEXAS

ABILENE

Abilene Regional MH/MR Center Substance Abuse Services
2016 Clack Street, Suite 180
Abilene, TX 79603

Serenity Foundation of Texas
141 Mulberry Street
Abilene, TX 79601

Serenity Oak Tree Project
1533 North 3rd Street
Abilene, TX 79601

Serenity Women
1502 North 2nd Street
Abilene, TX 79601

ALICE

Alice Counseling Center Adolescent Supportive Outpatient
63 South Wright Street
Alice, TX 78333

Bay Area Health Care Group, Ltd. Columbia Counseling Center of Alice
1116 North Texas Boulevard
Alice, TX 78332

Recovery Campuses of Texas, Inc.
160 FM 2507
Alice, TX 78333

Treatment Associates
1315 East Main Street, Suite 104
Alice, TX 78332

ALPINE

Aliviane NO/AD, Inc. Project AHDRA
801 West Holland Street
Suite 102-A
Alpine, TX 79830

ALVIN

Alvin Counseling Services
304 Windsor Square
Alvin, TX 77511-4928

Bay Area Council
1111 West Adoue Street
Building C Suite 6
Alvin, TX 77511

Gulf Coast Recovery Center Alvin Recovery Program
2426 South Gordon Street
Alvin, TX 77511

AMARILLO

Amarillo Alcoholic Women's Recovery Center The Haven
1308 South Buchanan Street
Amarillo, TX 79102

Amarillo Council on Alcohol and Drug Abuse
616 North Polk Street
Amarillo, TX 79107

Outpatient Services
710 North Polk, Suite 707
Amarillo, TX 79107

One Day at A Time Ministries
3418 Olsen Boulevard, Suite B
Amarillo, TX 79109-3074

Veterans' Affairs Medical Center Substance Abuse Treatment Program
6010 Amarillo Boulevard West
Ward 2-A
Amarillo, TX 79106

West Texas Counseling and Rehabilitation Program
2300 Line Avenue
Amarillo, TX 79106

ANGLETON

Door to Recovery/Brazoria County
108 East Magnolia Street
Angleton, TX 77515

Gulf Coast Center Substance Abuse Recovery Program
101 Tigner Street
Angleton, TX 77515

ARANSAS PASS

Charter Counseling Center
423 West Cleveland Street Suite 1
Aransas Pass, TX 78336

ARGYLE

Sante Center for Healing
914 Country Club Road
Argyle, TX 76226

ARLINGTON

Family Service, Inc. Addiction Services
401 West Sanford Street
Suite 2600
Arlington, TX 76011

Green Oaks Mental Health Services
3150 Matlock Road, Suite 409
Arlington, TX 76015

Tarrant Community Outreach, Inc.
711 East Lamar Boulevard
Suite 205
Arlington, TX 76011

Urban Behavioral Health Care Systems, Ltd.
711 East Lamar Street, Suite 112
Arlington, TX 76011

AUSTIN

Aeschbach and Associates Substance Abuse Services
2005 East Riverside Drive
Austin, TX 78741

American Institute for Learning Outpatient Program
422 Congress Avenue
Austin, TX 78701

Austin Family House, Inc.
2604 Paramount Avenue
Austin, TX 78704

Austin Recovery Center
1900 Rio Grande Street
Austin, TX 78705

Adolescent Outpatient
1900 East Oltorf Street, Suite 102
Austin, TX 78741

Men's Program Level II
1808 West Avenue
Austin, TX 78701

Outpatient Adult Center
1900 East Oltorf Street
Suites 102-3
Austin, TX 78705

Recovery Lodge/Girls Residential and Day Treatment
3207 Slaughter Lane
Austin, TX 78748-5707

Women's Program Level II
1900 Rio Grande Street Annex
Austin, TX 78705

Austin/Travis County MH/MR Center
Methadone Maintenance
1631-A East 2 Street
Austin, TX 78702

Oak Springs Treatment Center
3000 Oak Springs Drive
Austin, TX 78702

Charter Behavioral Health Systems
8402 Cross Park Drive
Austin, TX 78754

Cornerstone Counseling
2417 Ashdale Drive
Austin, TX 78757

Counseling Network
809 North Cuernavaca Drive
Austin, TX 78733-3217

La Haciendas Solutions
10435 Burnet Street, Suite 114
Austin, TX 78758

Northwest Counseling and Wellness Center
13740 Research Boulevard
Building 4
Austin, TX 78750

Phoenix House Academy
400 West Live Oak Street
Austin, TX 78704

Phoenix House Council for Drug Education
611 South Congress Avenue
Suite 225
Austin, TX 78704

Push-Up Foundations, Inc.
1700 East 2nd Street
Austin, TX 78702

Saint David's Pavilion Chemical Dependency Partial Program
1025 East 32nd Street
Austin, TX 78705

Teen and Family Counseling Center
3536 Bee Caves Road
Suite 100
Austin, TX 78746

Travis County Community Justice Center Wakenhut Corrections Corporation
8101 FM 969
Austin, TX 78724

Trinity Therapeutic Options
1709 San Antonio Street
Austin, TX 78701-1224

Up to Me, Inc.
6222 North Lamar Street
Austin, TX 78752

BASTROP

Bastrop Behavioral Health Center
106 Loop 150 West
Bastrop, TX 78602

Bastrop Recovery Center
1106 College Street, Suite B
Bastrop, TX 78602

BAYTOWN

Community Council on Drugs and Alcohol Just for You Program
616 Park Street
Baytown, TX 77520

BEAUMONT

Beaumont Transitional Treatment Center
2495 Gulf Street
Beaumont, TX 77703

Columbia Behavioral Health Center Pinebrook Center
3250 Fannin Street
Beaumont, TX 77701

Franklin House/North
5670 Concord Street
Beaumont, TX 77708

Jefferson County COADA
700 North Street
Beaumont, TX 77701

Drug/Alcohol Abuse Recovery Center (DAARC)
2235 South Street
Beaumont, TX 77701

Land Manor, Inc.
Adams House/Adolescent Residential
1970 Franklin Street
Beaumont, TX 77701

Graham House
1635 Avenue A
Beaumont, TX 77701

Melton Center
1785 Washington Boulevard
Beaumont, TX 77705

Life Resource Substance Abuse Program
2750 South 8 Street
Beaumont, TX 77701

New View Partial Hospitalization Center
4310 Dowlen Road, Suite 13
Beaumont, TX 77706

Texas Youth Commission Chemical Dependency Treatment Programs
Jefferson County State School
3890 FM 3514
Beaumont, TX 77705

BEDFORD

Harris Methodist Springwood Hospital Addiction Treatment Center
1608 Hospital Parkway
Bedford, TX 76022

BELTON

Christian Farms/Treehouse, Inc. Christian Farms Men's Center
Route 3 Box 3852
Belton, TX 76513

DAIRE Information Referral Educational Services
306 East Avenue C
Belton, TX 76513

BIG LAKE

Permian Basin Rehabilitation House, Inc. Clover House Circuit Rider Annex
3rd and Plaza Street
Big Lake, TX 76932

BIG SPRING

Veterans' Affairs Medical Center Substance Abuse Treatment Program
300 Veterans Boulevard
Big Spring, TX 79720

BONHAM

Northeast Texas Council on Alcohol and Drug Abuse (NETCADA)
107 East 16th Street
Bonham, TX 75418

Sam Rayburn Memorial Veterans Center Domiciliary Substance Abuse Program
9th and Lipscomb Streets
Bonham, TX 75418

BRADY

West Texas Recovery Center
116 West Main Street
Brady, TX 76825

BRECKENRIDGE

Gateway Foundation Walker Sayle Unit Breckenridge SAFP Facility
4176 Fm 1800
Breckenridge, TX 76424

BROWNSVILLE

Cameron County Housing Authority Recovery Center
65 Castellano Circle
Brownsville, TX 78520

Tropical Texas Center for MH/MR Services
5 Boca Chica Street
Suite 5
Brownsville, TX 78520

BROWNWOOD

Mid-Texas Council on Alcohol and Drug Abuse
901 Avenue B
Brownwood, TX 76801

Thomas R Havins Substance Abuse Felony Punishment Facility
500 FM 45 East
Brownwood, TX 76801

BRYAN

Brazos Valley Council on Alcohol Substance Abuse Adolescent Treatment
1103 Turkey Creek
Bryan, TX 77805

Mental Health/Mental Retardation Authority of Brazos Valley
Dual Diagnosis Treatment
804 South Texas Avenue
Bryan, TX 77805

Saint Joseph Adolescent Substance Abuse Program
2010 East Villa Maria Road
Bryan, TX 77802

Twin City Mission TTC
500 North Main Street
Bryan, TX 77803-3322

BUDA

Austin Recovery Center
1888 Wright Road
Buda, TX 78610

BUFFALO GAP

Shades of Hope Treatment Center, Inc.
402 A Mulberry Street
Buffalo Gap, TX 79508

BURLESON

Abide Inc
6436 Mark Drive
Burleson, TX 76028

BURNET

Gateway Foundation Burnet Substance Abuse Facility
800 Ellen Halbert Drive
Burnet, TX 78611

CANTON

Andrews Center
575 West Highway 243
Canton, TX 75103

Sundown Ranch, Inc.
Route 4
Canton, TX 75103

CARRIZO SPRINGS

South Texas Rural Health Services, Inc. Substance Abuse Program
709 North 3rd Street
Carrizo Springs, TX 78834

CARROLLTON

North Dallas Drug Rehabilitation Center
1606 South I-35
Suite 101
Carrollton, TX 75006

CENTER

Alcohol and Drug Abuse Council of Deep East Texas
114 Hurst Street
Center, TX 75935

CENTER CITY

Starlite Village Hospital Substance Abuse Services
Elm Pass Road
Center Point, TX 78010

CHILTON

Chilton House
4006 Street
Chilton, TX 76632

CLARKSVILLE

Northeast Texas Council on Alcohol and Drug Abuse
200 Walnut Road
Clarksville, TX 75426

CLEBURNE

Helping Open Peoples Eyes, Inc.
1800 Ridgemar Street
Cleburne, TX 76031

Outpatient
619 North Main Street
Cleburne, TX 76031-0162

Huguley Psychotherapy Clinic
214 North Caddo Street
Cleburne, TX 76031

Johnson/Ellis/Navarro MH/MR Services
1601 North Anglin Street
Cleburne, TX 76031

COLLEGE STATION

Scott and White Regional Clinic Alcohol and Drug Dependency Outpatient Treatment Program
702University Drive
Suite 100 D
College Station, TX 77840

COLORADO CITY

Permian Basin Rehabilitation House, Inc. Clover House Circuit Rider
Mitchell County Courthouse
349 Oak Street
Colorado City, TX 79512

COLUMBUS

Colorado County Youth/Family Servs Inc
1336 Fannin Street
Columbus, TX 78934

CONROE

Continuum Health Care Systems Texas Serenity Counseling Service
240 South Main Street
Conroe, TX 77301

Texas Serenity Whitehouse
3201 North Frazier Street
Conroe, TX 77301

CORPUS CHRISTI

Bay Area Health Care Group, Ltd. Columbia Counseling Center
6629 Woolridge Street
Corpus Christi, TX 78414

Charter Behavioral Health Systems
3126 Rodd Field Road
Corpus Christi, TX 78414

Coastal Bend Alcohol/Drug Rehab Center
25 North Country Club Place
Corpus Christi, TX 78407

Henderson House
38 North Country Club Place
Corpus Christi, TX 78407

Ivy House
41 North Country Club Place
Corpus Christi, TX 78407

Coastal Bend Outpatient Services
1201 Agnes Road
Corpus Christi, TX 78401

Corpus Christi Regional Center for
Addictions, Inc. Bay Area Care
5230 Kostoryz Road, Suite 5B
Corpus Christi, TX 78415

Counseling and Assistance Center
Naval Hospital
10651 East Street
Corpus Christi, TX 78419

Spohn Memorial Hospital Behavioral Medicine Department
2606 Hospital Boulevard
Corpus Christi, TX 78405-1818

Council on Alcohol and Drug Abuse/Coastal Bend
1201 3rd Street
Corpus Christi, TX 78404

CORSICANA

Corsicana State Home
West 2 Avenue
Corsicana, TX 75110

Helping Open Peoples Eyes, Inc.(HOPE)
300 West 3rd Street
Corsicana, TX 75110

Johnson/Ellis/Navarro MH/MR Services
800 North Main Street
Corsicana, TX 75110

COTULLA

South Texas Rural Health Services, Inc.
304 Nueces Street
Cotulla, TX 78014

CROCKETT

Community Rehabilitation Professional Services, Inc.
110 North 2nd Street
County Courthouse Annex
Crockett, TX 75835

DALHART

69th Judicial District CSCD
5th and Denver Street
Courthouse Annex, Suite 5
Dalhart, TX 79022

DALLAS

Adapt Behavioral Healthcare
4225 Office Parkway
Dallas, TX 75204

Addicare Group of Texas Zenith Program
4300 North Central Expressway
Suite G-100
Dallas, TX 75206

Addiction Counseling Associates
6220 Gaston Avenue, Suite 405
Dallas, TX 75214

Alameda Heights Outreach Center
2721 Lyola Street
Dallas, TX 75241

Alliance Life Centers
13999 Goldmark Street, Suite 343
Dallas, TX 75240

Baylor University Medical Center Baylor Center for Addictive Diseases
3500 Gaston Avenue
Collias Hospital
Dallas, TX 75246

Catholic Charities Adolescent and Family Services
325 West 12th Street
Dallas, TX 75208

Community Alcohol/Drug Aftercare Housing Program
3200 S Lancaster
Kiest Shopping Center, Suite 509
Dallas, TX 75216

Cornell Corrections
3606 Maple Avenue
Dallas, TX 75219

D/Boy Counseling Center
5215 Lawnview Avenue
Dallas, TX 75227

Dallas County Juvenile Department
2600 Lone Star Drive
Dallas, TX 75212

Dallas Inter/Tribal Center
209 East Jefferson Boulevard
Dallas, TX 75203

Daytop Dallas Drug Treatment Program
2345 Reagan Street
Dallas, TX 75219

East Dallas Counseling Center
4306 Bryan Street
Dallas, TX 75204

Ethel Daniels Foundation, Inc.
Outpatient Services
1900 North Prairie Street
Dallas, TX 75206

First Step Counseling Center
13610 Midway Road
Suite 421
Dallas, TX 75208

Gateway Foundation Help Is Possible Project
723 South Peak Street
Dallas, TX 75223

Green Oaks at Medical City
7808 Clodus Fields Drive
Dallas, TX 75251

Holmes Street Foundation, Inc.
Adolescent Residential
2719 Holmes Street
Dallas, TX 75209

Outpatient Program
2606 Martin Luther King Jr. Boulevard
Suite 202
Dallas, TX 75215

Homeward Bound, Inc. Trinity Recovery Center
233 West 10th Street
Dallas, TX 75215

La Sima Foundation
777 R. L. Thornton Freeway
Suite 106
Dallas, TX 75209

Miracle Network, Inc.
1266 East Ledbetter Drive
Dallas, TX 75216

New Place, The
4301 Bryan Street
Suite 120
Dallas, TX 75204

Nexus Recovery Center
Adult and Adolescent Specialized Female Residential Program
Women and Children Residential Program
8733 La Prada Drive
Dallas, TX 75228

Nexus Outreach Center
2519 Oaklawn Avenue
Dallas, TX 75219

North Texas Health Care System
4500 South Lancaster Road
Dallas, TX 75216

Oak Lawn Community Services
4300 MacArthur Street
Dallas, TX 75209

One Day At A Time Ministries Outpatient Counseling Center
2702 South Buckner Boulevard
Dallas, TX 75227

Our Brothers Keeper/NDUGU
4200 South Fitzhugh Street
Dallas, TX 75210

Permanente Medical Association of Texas Kaiser Permanente Chemical Dependency Treatment Program
9250 Amberton Parkway
Dallas, TX 75243

Phoenix Project, Inc.
201 South Tyler Street
Dallas, TX 75208

Recovery First
9202-B Markville Drive
Dallas, TX 75243

Recovery Healthcare Corporation
2530 Electronic Lane, Suite 707
Dallas, TX 75220

Right Alternatives for People, Inc.
401 Wynnewood Village
Suite 104
Dallas, TX 75224

Road to Recovery Chemical Dependency Program
8350 Meadow Road, Suite 268
Dallas, TX 75231

Saint Paul Medical Center Chemical Dependency Recovery Services
5909 Harry Hines Boulevard
Dallas, TX 75235

Salud Counseling Services
2760 West Davis Street
Dallas, TX 75211

Salvation Army Social Service Center Substance Abuse Services Program
5302 Harry Hines Boulevard
Dallas, TX 75235

Step Med
1705 Martin Luther King Jr. Boulevard
Suite E
Dallas, TX 75215-3222

Timberlawn Mental Health Systems
4600 Samuell Boulevard
Dallas, TX 75227

Turtle Creek Manor, Inc.
2707 Routh Street
Dallas, TX 75201

Outpatient Services
2506 Cedar Springs Street
Dallas, TX 75201

Welcome House, Inc.
921 North Peak Street
Dallas, TX 75204

DAYTON

Key Program Lonestar/Dempsey Henley Unit
Highway 321
5 Miles North of Dayton Street
Dayton, TX 77535

DEER PARK

Cenikor Foundation, Inc. Substance Abuse Program
4525 Glenwood Avenue
Deer Park, TX 77536

DEL RIO

Alliance Behavioral Health Services, Inc. Excel Adolescent Program
902 South Main Street, Suite G
Del Rio, TX 78840

DENISON

Drug Recovery Center
330 Highway 69-E
Denison, TX 75021

DENTON

Denton County MH/MR Center Intensive Outpatient Substance Abuse Treatment Program
2519 Scripture Street
Denton, TX 76201

Starting Over, Inc.
531 Londonderry Lane Suite 100
Denton, TX 76205

DE SOTO

Haven Behavioral Health System
800 Kirnwood Drive

Lakeview Southwest, Inc. DBA Cedars Hospital
2000 North Old Hickory Trail
De Soto, TX 75115

DICKINSON

Bay Area Recovery Center
4316 Washington Street
Dickinson, TX 77539

1807 Pine Drive
Dickinson, TX 77539

Omega/Alpha House Women's Center
1122 Farm Market Road
Suite 517
Dickinson, TX 77539

DRISCOLL

Coastal Bend Youth City Substance Abuse Program
2547 U.S. Highway 77
Driscoll, TX 78351

DUMAS

69th Judicial District CSCD
810 South Dumas Avenue
Suite 416
Dumas, TX 79029

DYESS AFB

Dyess Air Force Base Substance Abuse Program
7 MDOS/SGOMH
597 Hospital Loop
Dyess AFB, TX 79607-1442

EAGLE PASS

Alliance Behavioral Health Services, Inc. Excel Adolescent Program
2315 Hillcrest Street
Eagle Pass, TX 78852

EDINBURG

Areas Management Information Systems Amistad Alcohol and Drug Treatment
1401 South 9th Street
Edinburg, TX 78540

Rio Grande Valley Council on Alcohol and Drug Abuse
3511 West Alberta Street
Edinburg, TX 78539

Tropical Texas Center for MH/MR Outpatient Substance Abuse Services
1901 South 24 Street
Edinburg, TX 78539

ELGIN

Twin Oaks Adolescent Center, Inc.
701 North Highway 95
Elgin, TX 78621

EL PASO

Aliviane NO/AD, Inc.
7722 North Loop Street
El Paso, TX 79915

Adolescent Day Treatment
7580 Alameda Street
Building 1 Space 3
El Paso, TX 79915

Inner Resources Women's/ Children's Residential Center
11960 Golden Gate Road
El Paso, TX 79936

Inner Resources Recovery Center
10690 Socorro Road
El Paso, TX 79927

Outpatient Clinic
5160B El Paso Drive
El Paso, TX 79905

Alliance Behavioral Health Services, Inc. Excel Adolescent Program
4919 Hondo Pass
El Paso, TX 79903

Drug Abuse Service Center
5160 El Paso Drive
El Paso, TX 79905

El Paso Methadone Maintenance and Detox Treatment Center
5004 Alameda Avenue
El Paso, TX 79905

El Paso Psychiatric Association Alternatives Center
5001 Alabama Street
El Paso, TX 79930

Life Management Center for MH/MR Services
Casa Blanca Therapeutic Communities
600 Newman Street
El Paso, TX 79902

Ocotillo
5304 El Paso Drive
El Paso, TX 79905

Serenity Outpatient Services, Inc.
4625 Alabama Street
El Paso, TX 79930

Tigua Indian Reservation Ysleta del Sur Pueblo Substance Abuse Program
119 South Old Pueblo Drive
El Paso, TX 79907

Veterans' Affairs Substance Abuse Treatment Program
5001 North Piedras Street
El Paso, TX 79925

William Beaumont Army Medical Center Residential Treatment Facility
10 West 5005 Piedras Street
El Paso, TX 79920-5001

EULESS

American Indian Center
2219 West Euless Boulevard
Euless, TX 76040

FALFURRIAS

Alice Counseling Center Rural Youth Treatment
217 East Miller Street
Falfurrias, TX 78355

FLORESVILLE

Brush Country COADA Supportive Outpatient Program
3190 State Highway 97 East
Floresville, TX 78114

FORT BLISS

Alcohol and Drug Abuse and Control Program
1733 Pleasanton Road
Fort Bliss, TX 79916-6816

FORT DAVIS

Clover House, Inc. Circuit Rider
Jeff Davis County Courthouse
Court and Main Street
Fort Davis, TX 79734

FORT WORTH

All Saints Episcopal Hospital Recovery Place
1400 8th Avenue
Fort Worth, TX 76104

Cenikor Foundation, Inc. North Texas Facility
2209 South Main Street
Fort Worth, TX 76110

Family Service, Inc. Substance Abuse Treatment
1424 Hemphill Street
Fort Worth, TX 76104

North Texas Addiction Counseling, Inc.
909 West Magnolia Street, Suite 2
Fort Worth, TX 76104

Permanente Medical Association of Texas Kaiser Permanente Chemical Dependency Treatment Program
1300 South University Drive
Fort Worth, TX 76104

Phoenix Associates Counseling Services
3001-A West 5th Street
Fort Worth, TX 76107

Salvation Army
First Choice Program
2110 Hemphill Street
Fort Worth, TX 76110

Santa Fe Counseling Center, Inc. Adolescent Services
3122 East Rosedale Street
Fort Worth, TX 76105

Tarrant County Medical Education and Research Foundation
Outpatient
904 Southland Avenue
Fort Worth, TX 76104

Volunteers of America Northern Texas, Inc.
2710 Avenue J
Fort Worth, TX 76105
Gemini House South
4700 South Riverside Street
Fort Worth, TX 76119

Western Clinical Health Services Pennsylvania Avenue Clinic
514 Pennsylvania Avenue
Fort Worth, TX 76104

FREEPORT

Brazoria County Alcohol Recovery Center Brazos Place
11034 North Avenue H
Freeport, TX 77541

GAINESVILLE

Cooke County Mental Health Center
301 West Main Street
Gainesville, TX 76240

Texas Youth Commission
Gainesville State School
4701 East Farm Road
Suite 678
Gainesville, TX 76240

GALVESTON

Alcohol and Drug Abuse Women's Center, Inc.
201 1st Street
Galveston, TX 77550

Dual Recovery
Galveston, TX 77550

Family Opportunity Resources
6000 Broadway Street Suite 106-R
Galveston, TX 77551

Galveston Recovery Program
123 Rosenberg Street
Galveston, TX 77550

Gulf Coast Center
123 Rosenberg Street, Suite 6
Galveston, TX 77550

New Horizons Center
728 Church Street
Galveston, TX 77550

Recovery Campuses of Texas, Inc.
2216 Avenue O
Galveston, TX 77550

Recovery Center, Inc.
3205 Avenue O
Galveston, TX 77550-6861

Turning Point
801 37th Street
Galveston, TX 77550

GARLAND

D. Gonzalez and Associates
2848 West Kingsley Road
Suite B
Garland, TX 75041

Garland Community Hospital
Behavioral Medicine Services
2696 West Walnut Street
Garland, TX 75042

Wellness Center
2636 West Walnut Street
Garland, TX 75042

Garland Treatment Center
6246 Broadway Street, Suite 102
Garland, TX 75043

GATESVILLE

Gateway Foundation Texas Hackberry SAFP Facility
1401 State School Road
Gatesville, TX 76528

GEORGETOWN

Center for Addiction Recovery and Education
107 Halmar Cove
Georgetown, TX 78628

Cornerstone Counseling
504-B Leander Road
Georgetown, TX 78628

GOODFELLOW AFB

Goodfellow Air Force Base Substance Abuse Program
17 MDOS/SGOKB
143 Ft Lancaster Avenue
Building 143
Goodfellow AFB, TX 76908

GRAPEVINE

Charter Grapevine Behavioral Health System
2300 William D Tate Street
Grapevine, TX 76051

GREENVILLE

Glen Oaks Hospital
301 East Division Street
Greenville, TX 75401

Green Villa
733 IH 30 East
Greenville, TX 75401

Tarrant Community Outreach, Inc. Faces of Reality Counseling Center
2901 Lee Street
Greenville, TX 75403-1097

HARLINGEN

Rio Grande Valley Council on Alcohol and Drug Abuse
2308 South 77 Sunshine Strip
Harlingen, TX 78550

Tropical Texas Center for Mental Health/Mental Retardation Services
1242 North 77 Sunshine Strip
Harlingen, TX 78550

HEBRONVILLE

Stop Child Abuse and Neglect, Inc. Stand Outpatient Program
707 South Smith Street
Hebbronville, TX 78361

HEMPSTEAD

Brazos Valley Council on Alcohol and Drug Abuse
Walker County Corrections
925 5th Street
Hempstead, TX 77445

HENDERSON

Sabine Valley Center
Regional Substance Abuse Recovery Center
209 North Main Street
Henderson, TX 75652

HOUSTON

Association for the Advancement of Mexican Americans (AAMA)
Region 6 Youth COADA
6001 Gulf Freeway Building C-1
Houston, TX 77023

AAMA/Campus
4514 Lyons Avenue
Houston, TX 77020

Better Way Inc
4802 Caroline Street
Houston, TX 77004

Bay Area Council on Drugs and Alcohol
1300 Bay Area Boulevard
Houston, TX 77058

Bayon City Medical Center South Campus Psychiatric Services
6700 Bellaire Boulevard
Houston, TX 77074

Best Recovery Health Care
9211 South Main Street
Houston, TX 77025

Blues Management, Inc. Dapa Recovery Program
7447 Harwin Street, Suite 212-B
Houston, TX 77036

Boundaries Counseling Center
9725 1/2 Lou Edd Street
Houston, TX 77070

Browns Education and Recovery
9000 West Bellfort Street
Suite 325
Houston, TX 77031

Career and Recovery Alternative Drug Abuse Treatment Program
2525 San Jacinto Street
Houston, TX 77002

Center for Recovering Families
2620 Fountain View Drive
Suite 480
Houston, TX 77057-7621

Cheyenne Center
9100 Dodson Street
Houston, TX 77093-7148

Chicano Family Center Substance Abuse Program
7524 Avenue E
Houston, TX 77012

Child and Adolescent Development, Inc. Adolescent Residential
2505 Southmore Street
Houston, TX 77004

Clear Lake Counseling Services
17000 El Camino Real Street
Suite 104-C
Houston, TX 77058

Cornell Corrections Ben A. Reid Facility
10950 Beaumont Highway
Houston, TX 77078

Cypress Creek Hospital Substance Abuse Services
17750 Cali Street
Houston, TX 77090

De Pelchin Children's Center
Montgomery County Satellite
100 Sandman Street
Houston, TX 77007

Outpatient Services
3214 Austin Street
Houston, TX 77004

Door to Recovery
4910 Dacoma Street
Houston, TX 77092

2005 Jacquelyn Drive
Houston, TX 77055

7605 Denton Street
Houston, TX 77028

Intensive Residential Unit
638 Harbor Road
Houston, TX 77092

Dr. Crismon and Associates, Inc.
4625 North Freeway, Suite 150
Houston, TX 77022-2913

Easy Does It, Inc.
6630 Harwin Street, Suite 225
Houston, TX 77036

Extended Aftercare, Inc.
5002 North Shepard Street
Houston, TX 77018

Families Under Urban and Social Attack
2206 Dowling Street Suite 201
Houston, TX 77288-0107

6719 West Montgomery Street
Houston, TX 77091

Family Service Center
4615 Lillian Street
Suite 101
Houston, TX 77087

Forest Springs Residential Treatment Center
1120 Cypress Station
Houston, TX 77090

Fulfillment Foundation Prospect House
309 West 27th Street
Houston, TX 77008

Gulf Shores Academy, Inc.
11300 South Post Oak Street
Suite 1
Houston, TX 77038

Harris County Dual Disorders Project
2627 Caroline Street
Houston, TX 77004

New Directions Women's Program
2502 Fannin Street
Houston, TX 77002

Houston Aftercare, Inc.
407 Welch Street
Houston, TX 77006-1307

2004 Crocker Street
Houston, TX 77006

Houston Maintenance Clinic, Inc.
4900 Fannin Street, Suite 201
Houston, TX 77004

Houston New Start Inc
9219 Katy Freeway Suit 291
Houston, TX 77024

Houston Substance Abuse Clinic
7428 Park Place Boulevard
Houston, TX 77087

Journey Program Inc
9219 Katy Freeway, Suite 291
Houston, TX 77024

Lifeway
6251 Corporate Drive
Houston, TX 77036-3411

Mac House, Inc.
3903 Hartsdale Street
Houston, TX 77063

Make Ready, Inc.
2405 Smith Street
Houston, TX 77006

Memorial Hospital Southwest Parkside Recovery Center
7600 Beechnut Street, 10th Floor
Houston, TX 77074

Montrose Counseling Center
701 Richmond Avenue
Houston, TX 77006

Narcotics Withdrawal Center
4800 West 34th Street
Suite B3
Houston, TX 77092

New Directions Club, Inc.
607 Thornton Street
Houston, TX 77018

Odyssey House Texas, Inc.
5629 Grapevine Street
Houston, TX 77085

Oxford Counseling Center
4101 North Freeway
Suite 100
Houston, TX 77022

Pain Care Center
4543 Post Oak Place, Suite 106
Houston, TX 77027

Passages, Inc.
7722 Westview Drive
Houston, TX 77055

Pollux House Addictions Foundation, Inc.
4728 Gunter Street
Houston, TX 77020

Recovery Foundation, Inc.
4312 Crane Street
Houston, TX 77026-4802

Recovery Houston Institute Choices Program
10525 Eastex Freeway
Houston, TX 77093

Rehab Mission, Inc.
1701 Jacquelyn Street
Houston, TX 77055

Riverside Campus
4514 Lyons Avenue
Houston, TX 77020-5237

Riverside General Hospital
2905 Elgin Street
Houston, TX 77004

Jones Healthcare Center
7655 Bellfort Street
Houston, TX 77061

Total Care/Detox Unit
3204 Ennis Street
Houston, TX 77004

S and S Counseling Services and Associates Incorporated
9000 West Bellfort Street Suite 570
Houston, TX 77031

Santa Maria Hostel, Inc.
807 Paschall Street
Houston, TX 77009

Set Free DAT Center
3333 Fannin Street, Suite 106
Houston, TX 77004

Sunrise Recovery Program
2611 Fm 1960 West
Houston, TX 77090

Texas Alcoholism Foundation, Inc.
Texas House Treatment Program
2208 West 34 Street
Houston, TX 77213

Texas Clinic/Fulton Street
6311 Fulton Street
Houston, TX 77022

Westview Drive
9320 Westview Drive
Suite 10
Houston, TX 77055

Texas Serenity Counseling Service
250 Meadow Fern Road Suite 100
Houston, TX 77067

Texas Treatment Center
4800 West 34th Street Suite B-3
Houston, TX 77092

Texas Treatment Center South
1050 Edgebrook Drive Suite 2
Houston, TX 77034

Toxicology Associates
530 North Belt Street
Suite 311
Houston, TX 77060

Turning Point
3600 South Gessner Street
Suite 248
Houston, TX 77063

University of Texas Health Science Center
Houston Recovery Campus
4514 Lyons Avenue
Houston, TX 77020

Substance Abuse Research Center
1300 Moursund Street
Houston, TX 77030

Unlimited Visions Aftercare, Inc.
5528 Lawndale Street
Houston, TX 77023

Veterans' Affairs Substance Abuse Program
2002 Holcombe Boulevard
Houston, TX 77030

Volunteers of America
Rogers Street Recovery Center
308 East Rogers Street
Houston, TX 77022

2141 Bingle Street
Houston, TX 77001

West Oaks Hospital, Inc. Chemical Dependency Services
6500 Hornwood Drive
Houston, TX 77074

HUMBLE

Door to Recovery Montgomery County
1220 Stone Hollow Drive
Humble, TX 77339

HUNT

La Hacienda Treatment Center
FM 1340
Hunt, TX 78024

HUNTSVILLE

Dual Diagnosis Treatment Program
21016 South Sam Houston Street
Huntsville, TX 77340

Gateway Foundation
Estelle Unit/SAFP Facility
Huntsville, TX 77340

Jester Unit 1
1600 Financial Plaza, Suite 370
Huntsville, TX 77340

Hunstville Alcohol/Drug Abuse Program
115 North Highway 75
Huntsville, TX 77340

Hunstville Clinic, Inc.
829 10th Street
Huntsville, TX 77340

Montgomery/Walker County COADA Right Start Youth
526 11th Street
Huntsville, TX 77340

HURST

Tarrant County MH/MR Services Addiction Recovery Center
129 Harmon Road
Hurst, TX 76053

HUTCHINS

Volunteers of America Northern Texas, Inc. Perry F Bradley Center
800 West Wintergreen Road
Hutchins, TX 75141

IRVING

New Vision Teen Center
220 West Irving Boulevard
Irving, TX 75060

Irving Christian Counseling Centers
2621 West Airport Freeway
Suite 124
Irving, TX 75062-6069

Remedy Addictions Counselors (RAC), Inc.
317 East Airport Freeway
Irving, TX 75062

West Texas Counseling of Irving
2001 West Airport Freeway
Suite 113
Irving, TX 75062

JACKSONVILLE

Community Rehabilitation Professional Services, Inc.
514 East Commerce Street
Jacksonville, TX 75766

Sabine Valley Center The Beginning/Regional Substance Abuse Recovery Center
903 South Jackson Street
Jacksonville, TX 75766

KELLY AFB

Alcohol and Drug Abuse Prevention and Treatment Program
76 AMDS/SGPH
1014 Billy Mitchell Boulevard
Suite 2
Kelley AFB, TX 78241-5604

Kelly Air Force Base Substance Abuse Program
76 MDOS-SGOMH
144 Armistad Circle, Suite 2
Kelly AFB, TX 78241-5846

KERRVILLE

Hill Country Independence House
976 Barnett Street
Kerrville, TX 78028

Kimberlite Cottage
324 Clay Street
Kerrville, TX 78028

South Texas Veterans Health Care System Substance Abuse Treatment
3600 Memorial Boulevard
Kerrville, TX 78028

Treatment Associates
712 Barnett Street
Kerrville, TX 78028-4520

KINGWOOD

Charter Hospital of Kingwood Inpatient Unit
2001 Ladbrook Drive
Kingwood, TX 77339

KOUNTZE

Life Resource/Hardin County
Highway 326
Kountze, TX 77625

KYLE

Wackenhut Corrections Corporation New Vision
701 South IH 35
Kyle, TX 78640

LACKLAND AFB

Lackland AFB Alcohol and Drug Abuse Prevention and Treatment Program
59 MDW/MMCNS
2220 Berguist Drive, Suite 1
Lackland AFB, TX 78236-5300

Wilford Hall Medical Center Substance Abuse Services/ MMPWS
2289 McChord Street
Building 1355
Lackland AFB, TX 78236-5300

LAKE JACKSON

Brazosport Memorial Hospital Alpha Center
100 Medical Drive
Lake Jackson, TX 77566

LA MARQUE

Bay Area Council on Drugs and Alcohol, Inc.
1101 Delmar Street, Suite 9
La Marque, TX 77568

Toxicology Associates
2411 Franklin Street
La Marque, TX 77568

LAMESA

Permian Basin Rehabilitation House, Inc. Clover House Circuit Rider
609 North 1st Street
Lamesa, TX 79331

LAREDO

Association for the Advancement of Mexican Americans
Buena Salud
2305 Ventura Street
Laredo, TX 78040

Concilio Hispano Libre
1205 East Hillside Street
Laredo, TX 78041

Mi Tierra South Texas Council on Alcohol/Drug Abuse
2520 Lane Street
Laredo, TX 78040

South Texas COADA
1502 Laredo Street, Suite 2
Laredo, TX 78040

Stop Child Abuse and Neglect, Inc.
Raices Residential Program
4600 South Zapata Highway
Laredo, TX 78042

Stand Outpatient Program
1901 La Pita Mangana Road
Laredo, TX 78043

2387 East Sanders Street
Laredo, TX 78041-5434

LAUGHLIN AFB

Laughlin Air Force Base Substance Abuse Program
47 MDOS/SGOMH
590 Mitchell Boulevard
Laughlin AFB, TX 78840-5244

LEAGUE CITY

Devereux Texas Treatment Network
Devereux Intensive Outpatient Chemical Dependency Program
1150 Devereux Drive
League City, TX 77573

Neurobehavioral Institute of Texas
Helena House
2605 Austin Street
League City, TX 77573

LEWISVILLE

Medical City Dallas, Inc. Columbia Green Oaks Behavioral Health Services
475 West Elm Street Suite 100
Lewisville, TX 75057

LIVERPOOL

Door to Recovery III on the Bayou
638 Harbor Road
Liverpool, TX 77577

LIVINGSTON

Alcohol and Drug Abuse Council of Deep East Texas
Courthouse
Livingston, TX 77351

LOCKHART

Hayes Caldwell Council on ADA
216 West San Antonio Street
Lockhart, TX 78644-2807

LONGVIEW

East Texas Clinic
201 Pine Tree Road
Longview, TX 75604

Kirkpatrick Family Center
1411 North 10th Street, Suite 1
Longview, TX 75601

Woodbine Treatment Center
9111 Pegues Place
Longview, TX 75608

LUBBOCK

Canyon Lakes Residential Treatment Center, Inc. Supportive Adolescent Services
2402 Canyon Lake Drive
Lubbock, TX 79415

Charter Plains Behavioral Health Services
801 North Quaker Avenue
Lubbock, TX 79416

Lubbock Faith Center, Inc.
Center Recovery Program
2809 Clovis Road
Lubbock, TX 79415

Lubbock Regional MH/MR Center
Billy Meeks Addiction Center
1601 Vanda Avenue
Lubbock, TX 79401

Lubbock Regional MH/MR Center Project Hope
1202 Main Street
Lubbock, TX 79401

Methadone Clinic
14 Briercroft Office Park
Lubbock, TX 79402

The Ranch
3201 East Kent Street
Lubbock, TX 79403

Managed Care Center for Addictive and Other Disorders
1705 North Farm Market Road 179
Lubbock, TX 79416

1926 34th Street
Lubbock, TX 79411

Texas Tech University Health Sciences Center Southwest Institute for Addictive Diseases
Department of Psychiatry
3601 4th Street
Lubbock, TX 79410

Walker House
1614 Avenue K
Lubbock, TX 79401

LUFKIN

Alcohol and Drug Abuse Council of Deep East Texas
304 North Raguet Street
Lufkin, TX 75901

Burke Center
Peavy Switch Recovery Center
Route 5
Lufkin, TX 75901

Adolescent Center
2303 North Raguet Street
Lufkin, TX 75901

LYTLE

Las Manos Community Mental Health Center
18325 IH-35 South
Lytle, TX 78052

MARSHALL

Azleway, Inc.
Azleway Boys Ranch
411 West Burleson Street
Marshall, TX 75670

Choices Adolescent Treatment Center
4521 Karnack Highway
Marshall, TX 75670

Grove/Moore Center
401 North Grove Street
Marshall, TX 75670

Oak Haven Recovery Center
Highway 154
Marshall, TX 75670

MCALLEN

Charter Palms Behavioral Health Services
1421 East Jackson Avenue
McAllen, TX 78503

Kids in Development Services, Inc. Pasos at Taylor Ranch
4 1/2 Miles North Taylor Road
McAllen, TX 78501

Rio Grande Valley Family Recovery Center
5401 North 10th Street, Suite 128
McAllen, TX 78504-2759

Treatment Associates
805 East Esperanza Street
McAllen, TX 78501

MCKINNEY

Collin County MH/MR Center
825 North McDonald Street
McKinney, TX 75069

MENARD

West Texas Recovery Center Menard County Courthouse
210 East San Saba Street
Menard, TX 76859

MIDLAND

Court Residential Treatment Center
215 West Industrial Avenue
Midland, TX 79702

Desert Springs Medical Center
3300 South FM 1788
Midland, TX 79711

Palmer Drug Abuse Program
413 North Baird Street
Midland, TX 79701

Permian Basin Community Centers for Mental Health/ Mental Retardation
Project Proud
606 North Weatherford Street
Midland, TX 79701

West Texas Counseling and Rehabilitation Program
1802 West Wall Street
Midland, TX 79701

MINEOLA

Andrews Center Substance Abuse Services
703 West Patton Street
Mineola, TX 75773

MINERAL WELLS

Helping Open Peoples Eyes, Inc.
319 North Oak Street
Mineral Wells, TX 76068

MOUNT PLEASANT

Sabine Valley Center The Beginning/Regional Substance Abuse Recovery Center
107 East 11th Street
Mount Pleasant, TX 75455

NACOGDOCHES

Alcohol and Drug Abuse Council of Deep East Texas
1329 North University Drive
Nacogdoches, TX 75961

Community Rehabilitation Professional Services, Inc.
206 West Pillar Street
Nacogdoches, TX 75961

ODESSA

Permian Basin Rehab
406 North Texas Street
Odessa, TX 79761

700 North Dixie Street
Odessa, TX 79761

Project Elizabeth
620 South Grant Street
Odessa, TX 79761

TTC Circuit Rider
300 North Jackson Street
Odessa, TX 79761

Turning Point
2000 Maurice Road
Odessa, TX 79763

ORANGE

Life Resource/A CMHC Substance Abuse Services
4303 North Tejas Parkway
Orange, TX 77630

PALESTINE

Daytop Pine Mountain Residential
Route 3
Palestine, TX 75801

PALO PINTO

Helping Open Peoples Eyes, Inc.
503 Oak Street
Palo Pinto, TX 76484

PAMPA

Genesis House, Inc.
Administrative Unit
615 West Buckler Street
Pampa, TX 79066

Genesis House for Boys
600 West Browning Street
Pampa, TX 79065

Genesis House for Girls
420 North Ward Street
Pampa, TX 79066

PARIS

Northeast Texas Council on Alcohol and Drug Abuse Bright Futures Integrated Treatment Program
136 Grand Avenue
Paris, TX 75460

Saint Josephs Hospital and Health Center Behavioral Medicine Services
820 Clarksville Street
Paris, TX 75460

PASADENA

Bay Area Council on Drugs and Alcohol
1149 West Elsworth Street
Suite 145
Pasadena, TX 77501

Houston Substance Abuse Clinic
5825 Spencer Highway
Pasadena, TX 77505

PHARR

Self and Family Empowerment Zone
1899 North Cage Street, Suite B-2
Pharr, TX 78577

PLAINVIEW

Central Plains Center for MH/MR and Substance Abuse
2700 Yonkers Street
Plainview, TX 79072

Institute for Adolescent Addictions
404 Floydada Street
Plainview, TX 79072

Plainview Women's Center
700 Borger Street
Plainview, TX 79072

W. W. Allen Treatment Center
715 Houston Street
Plainview, TX 79072

Methodist Hospital Plainview Lonetree Recovery Center
2601 Dimmitt Road
Plainview, TX 79072

Serenity Center, Inc.
806 El Paso Street
Plainview, TX 79072

PLANO

Collin County MH/MR Center Plano Clinic
3920 Alma Drive
Plano, TX 75023

Green Oaks Behavior Healthcare
3801 West 15th Street, Suite 320
Plano, TX 75075

New Place, The
221 West Parker Road, Suite 510
Plano, TX 75023

Presbyterian Hospital of Plano
Seay Behavioral Healthcare Center
6110 West Parker Road
Plano, TX 75093

PLEASANTON

Brush Country COADA Supportive Outpatient Program
1085 FM 3006
Pleasanton, TX 78064

PORT ARTHUR

Best Recovery Health Care, Inc.
509 9th Avenue
Port Arthur, TX 77642

Life Resource South County Alcohol and Drug Treatment
3401 57th Street
Port Arthur, TX 77640

PORT NECHES

Patch of Jefferson County, Inc.
1227 Dallas Street
Port Neches, TX 77651

PRESIDIO

Clover House, Inc. Circuit Rider
Court House Annex
O Riley Street
Presidio, TX 79845

RANDOLPH AFB

Randolph Air Force Base ADAPT Program
12 MDOS/SGOMH
221 3rd Street West
Randolph AFB, TX 78150

RANKIN

Permian Basin Rehabilitation House, Inc. Clover House
205 East 10th Street
Rankin, TX 79778

RICHARDSON

Baylor/Richardson Medical Center Mental Health Services
401 West Campbell Road
Richardson, TX 75080

Paul Meier New Life Clinic
2071 North Collins Boulevard
Richardson, TX 75080

Turning Point Counseling Center
1701 North Greenville Avenue
Suite 701
Richardson, TX 75081-1852

RIO GRANDE CITY

STCADA Outpatient Level IV
105 Lopez Street
Rio Grande City, TX 78582

Stop Child Abuse and Neglect, Inc. Stand Outpatient Program
102 North Lopez Street
Rio Grande City, TX 78582

SAN ANGELO

Court Residential Treatment Center
3398 McGill Street
San Angelo, TX 76905

River Crest Hospital
1636 Hunters Glen Street
San Angelo, TX 76901

Shannon Medical Center
2018 Pulliam Street
San Angelo, TX 76905

West Texas Counseling and Rehabilitation Program
601 South Irving Street, Suite 4
San Angelo, TX 76903

West Texas Recovery Center
232 West Beauregard Road
Suite 101
San Angelo, TX 76903

SAN ANTONIO

Alamo Area Dual Diagnosis Expansion Project The Center for Health Care Services
3031 IH 10 West
San Antonio, TX 78201

Alamo Mental Health Group, Inc. Spectrum
5115 Medical Drive
Building G
San Antonio, TX 78229

Alamo Recovery Center
1018 Grayson Street
San Antonio, TX 78208

2821 Guadalupe Street, Suite 108
San Antonio, TX 78207

Alpha Home, Inc.
300 East Mulberry Avenue
San Antonio, TX 78212

Baptist Medical Center Baptist Recovery Center
111 Dallas Street
San Antonio, TX 78205

Brooks Air Force Base Counseling Services
8005 Lindbergh Drive
San Antonio, TX 78235

Center for Health Care Services
IH 10 West Unit
3031 IH 10 West
San Antonio, TX 78201

Charter Behavioral Health System
8550 Huebner Road
San Antonio, TX 78240

City of San Antonio Metropolitan Health District
332 West Commerce Street
San Antonio, TX 78210-3845

Community Counseling Center
MCHE 5YA Building
142 Stanley Road
San Antonio, TX 78234-6327

Drug Dependence Associates
3701 West Commerce Street
San Antonio, TX 78207

Inman Christian Center
Residential Treatment Center
18952 Redland Road
San Antonio, TX 78259

Youth Counseling Center
1014 South San Jacinto Street
San Antonio, TX 78207

Lackland Air Force Base Substance Abuse Clinic
Building 1355
San Antonio, TX 78236

Mission Vista Behavioral Health System
14747 Jones Maltsberger Street
San Antonio, TX 78247-3713

Patrician Movement
263 Felisa Street
San Antonio, TX 78210

Site 1/Residential
222 East Mitchell Street
San Antonio, TX 78210

Site 3/Outpatient Treatment Program
215 Claudia Street
San Antonio, TX 78210

Site 5/Outpatient Treatment Program
528 South Polaris Street
San Antonio, TX 78203

River City Rehabilitation Center, Inc.
680 Stonewall Street
San Antonio, TX 78214

San Antonio Regional Hospital Chemical Dependency Program
8026 Floyd Curl Drive
San Antonio, TX 78229

South Texas Veterans Health Care System Substance Abuse Treatment
7400 Merton Minter Boulevard
San Antonio, TX 78284

Southwest Mental Health Center
2939 West Woodlawn Avenue
San Antonio, TX 78228

Tejas Recovery and Counseling Services
7418 West Military Drive
San Antonio, TX 78227-2949

Treatment Associates of San Antonio
410 South Main Street, Suite 202
San Antonio, TX 78204

SAN DIEGO

Key Program Lonestar/ Glossbrenner Unit
623 South Fm 1329
San Diego, TX 78384

SAN MARCOS

Counseling Network
174 South Guadalupe Street
Suite 200
San Marcos, TX 78666

Hays Caldwell Council on Alcohol and Drug Abuse
101 Uhland Road, Suite 113
San Marcos, TX 78666

SEGUIN

Guadalupe Valley Hospital Teddy Buerger Center for Alcohol/Drug Abuse
1215 East Court Street
Seguin, TX 78155

Treatment Associates of Seguin
504 North River Street
Seguin, TX 78155-4739

SHEPPARD AFB

Sheppard Air Force Base Substance Abuse Program
82 MDOS/SGOHA
149 Hart Street, Suite 5
Sheppard AFB, TX 76311-3482

SHERMAN

Alliance Life Centers
209 South Travis Street
Sherman, TX 75090

SINTON

Coastal Bend Regional Substance Abuse Treatment Facility
800 North Vineyard Street
Sinton, TX 78387

SMITHVILLE

Austin Recovery Center
Park Road Suite 1-C
Smithville, TX 78957

SPRING

Jamies House, Inc.
15919 Stuebner Airline Street
Spring, TX 77379

SPUR

Permian Basin Rehabilitation House, Inc.
Clover House White River Lindsey Place
HCR 2, Box 123
White River Lake
Spur, TX 79370

STAFFORD

Depelchin Children's Center
10435 Greenbough Street
Building 200
Stafford, TX 77477

STEPHENVILLE

Helping Open Peoples Eyes, Inc. (HOPE)
586 East Washington Street
Stephenville, TX 76402

Summer Sky, Inc. Chemical Dependency Treatment Center
1100 McCart Street
Stephenville, TX 76401

SULPHUR SPRINGS

Northeast Texas Council on Alcohol and Drug Abuse Outpatient Unit
954 Main Street
Sulphur Springs, TX 75482

TAFT

Shoreline, Inc.
1220 Gregory Street
Taft, TX 78390

TEMPLE

CEN/TEX Alcoholic Rehabilitation Center
2500 South General Bruce Drive
Temple, TX 76504

Central Texas Veterans Healthcare System Psychiatry Service
1901 South First Street
Temple, TX 76504

Christian Farms/Treehouse, Inc. Treehouse Women's Center
3804 Riverside Trail
Temple, TX 76502

Scott and White Santa Fe Center Alcohol and Drug Dependence Treatment Program
600 South 25 Street
Temple, TX 76503

TERRELL

Alliance Life Centers
809 West Nash Street
Terrell, TX 75160

Training andDevelopment Center of Terrell Employee Support Systems Company of Texas
211 East Moore Street
Terrell, TX 75160

TEXARKANA

Bowie County Addiction Counseling
1414 New Boston Road, Suite 101
Texarkana, TX 75501

Edge of Texas Recovery Center
519 Oak Street
Texarkana, TX 75501

Hazel Street Recovery Center
1217 Hazel Street
Texarkana, TX 75501

Red River Council on Alcohol and Drug Abuse
Dowd House
2101 Dudley Avenue
Texarkana, TX 75502

Sabine Valley Center The Beginning/Regional Substance Abuse Recovery Center
911 North Bishop Street
Texarkana, TX 75501

TEXAS CITY

Alcohol Drug Abuse Women's Center, Inc.
712 5 Avenue North
Texas City, TX 77590

Gulf Coast Center Mainland Recovery Program
8900 Emmett Lowry Expressway
Suite 103
Texas City, TX 77591-2103

TOMBALL

Tomball College Counseling Institute Substance Abuse Services
30555 Tomball Parkway
Tomball, TX 77375

TULIA

Driskill Halfway House
1202 Highway 87 North
Tulia, TX 79088

TYLER

Azleway Inc.
1203 North Broadway
Tyler, TX 75702

Azleway Boys Ranch
15892 County Road 26
Tyler, TX 75707

Beginning, The
4717 Troup Highway
Tyler, TX 75701

East Texas Medical Center Behavioral Health Center
4101 University Boulevard
Tyler, TX 75701

Fister Counseling Services First Step Recovery Program
215 Winchester Drive
Tyler, TX 75701

UNIVERSAL CITY

Behavorial Health Clinic ADAPT Program
1985 First Street West, Suite 1
Universal City, TX 78150

UVALDE

South Texas Rural Health Services, Inc.
1024 Garner Field Road
Uvalde, TX 78801

VAN HORN

Aliviane NO/AD, Inc. Project Ahora
1801 West Broadway, Suite 105
Van Horn, TX 79855

VERNON

Vernon State Hospital Adolescent Forensics
4730 College Drive
Vernon, TX 76384

VICTORIA

Bay Area Health Care Group, Ltd. Columbia Counseling Center
1403 North Wheeler Street
Victoria, TX 77901

Best Recovery Health Care, Inc.
1708 Laurent Street
Victoria, TX 77901

Columbia Counseling Center
2001 Sabine Street Suite 104
Victoria, TX 77901-5953

Steps to Recovery
1402-B Villagee Drive
Victoria, TX 77901

Treatment Associates
107 Cozzi Circle
Victoria, TX 77901

VINTON

Alliance Behavioral Health Services Excel Adolescent Program
431-B East Vinton Road
Vinton, TX 79821

WACO

Lake Shore Center for Psychological Services Better Way Chemical Dependency Treatment Program
4555 Lake Shore Drive
Waco, TX 76710

Manna House
926 North 14th Street
Waco, TX 76707

Freeman Center
Dear Unit
1619 Washington Avenue
Waco, TX 76703

Doris Goodrich Jones House
326 North 14th Street
Waco, TX 76703

Men's Residential
1401 Columbus Avenue
Waco, TX 76701

Outpatient Unit
2505 Washington Avenue
Waco, TX 76703

Residential Unit
1515 Columbus Avenue
Waco, TX 76701

Women's Residential
1425 Columbus Avenue
Waco, TX 76703

Washington House
2200 Washington Avenue
Waco, TX 76708

WAXAHACHIE

Alliance Life Centers
201 East Franklin Street
Waxahachie, TX 75165

Johnson/Ellis/Navarro MH/MR Services
116 North Rogers Street
Waxahachie, TX 75165

WESLACO

Texson Management Group, Inc. Valley Transitional Treatment Center
617 1/2 South International Street
Weslaco, TX 78596

WHITE OAK

Sabine Valley Center Dear Recovery Center
2000 U.S. Highway 80
White Oak, TX 75693

WICHITA FALLS

Red River Detox and Recovery Center
4411 Henry S Grace Freeway
Wichita Falls, TX 76302

Red River Drug and Alcohol Treatment Center Adolescent Inpatient Program
1505 8th Street
Wichita Falls, TX 76301

Rose Street Clinics
1800 Rose Street
Wichita Falls, TX 76301

Serenity Foundation of Texas Intensive Outpatient/ Outpatient
3100 5th Street, Suite 12
Wichita Falls, TX 76309

510 Lamar Street
Wichita Falls, TX 76301

WILMER

Cornell Corrections
200 Greene Road
Wilmer, TX 75172

WOODVILLE

Alcohol and Drug Abuse Council of Deep East Texas
100 Courthouse Street, Room 303
Woodville, TX 75979

Stop Child Abuse and Neglect, Inc. Stand Outpatient Program
800 Block Highway 83
Zapata, TX 78076

UTAH

BEAVER

Southwest Center
757 North Main Street
Beaver, UT 84713

BLANDING

San Juan Substance Abuse Services
356 East Main Street
Blanding, UT 84511

BOUNTIFUL

Bountiful Outpatient
470 East Medical Drive
Bountiful, UT 84010

Columbia Lake View Hospital Behavioral Medicine Unit
630 East Medical Drive
Bountiful, UT 84010

Utahs, Inc. of Davis County
48 East 400 South, Suite C
Bountiful, UT 84010

BRIGHAM CITY

New Choices Substance Abuse Treatment
245 West 1100 South
Brigham City, UT 84302

CASTLE DALE

Four Corners Mental Health Center
45 East 100 South
Castle Dale, UT 84513

CEDAR CITY

Paiute Tribe Behavioral Health Dept
600 North 100 East Paiute Drive
Cedar City, UT 84720

Southwest Center
91 North 1850 West
Cedar City, UT 84720

Horizon House Chemical Dependency Center
54 North 200 East
Cedar City, UT 84720

CLEARFIELD

Davis County Mental Health
Addictions Treatment Unit
904-A South State Street
Clearfield, UT 84015

Alcohol and Drug Center
860 South State Street
Clearfield, UT 84015

Women's Recovery Center
904-B South State Street
Clearfield, UT 84015

DELTA

Central Utah Counseling Center
51 North Center Street
Delta, UT 84624

DUCHESNE

Northeastern Counseling Center
27 South 100 West
Duchesne, UT 84021

DUGWAY

Alcohol and Drug Abuse Prevention and Control Program
Dugway Proving Ground
Building 5124, Room 210
Dugway, UT 84022

EAST CARBON

Four Corners Mental Health Center
305 Center Street
East Carbon, UT 84520

EPHRAIM

Central Utah Counseling Center
390 West 100 North
Ephraim, UT 84627

ESCALANTE

Southwest Center
100 East 100 North
Escalante, UT 84726

FARMINGTON

Davis County Mental Health Center
291 South 200 West
Farmington, UT 84025

FILLMORE

Central Utah Counseling Center
65 West Center
Fillmore, UT 84631

FORT DUCHESNE

Ute Indian Tribe Adult Alcohol Program
550 South 6777 East
Fort Duchesne, UT 84026

GREEN RIVER

Four Corners Mental Health Center
110 Medical Drive
Green River, UT 84525

HEBER CITY

Wasatch County Alcohol and Drug Treatment and Prevention Program
32 West 200 South
Heber City, UT 84032

HILL AFB

75 Medical Group/SGOHS Substance Abuse Program
6068 Aspen Avenue
Bldg 1295, Room 8
Hill AFB, UT 84056-5401

HURRICANE

Southwest Center
25 South Main Street
Hurricane, UT 84737

KANAB

Southwest Center
310 South 100 East, Suite 11
Kanab, UT 84741

KOOSHAREM

Sorenson's Ranch School, Inc.
410 North 100 East
Koosharem, UT 84744

LOGAN

Bear River Health Department New Choices Substance Abuse Treatment Program
95 South 100 West, Suite 300
Logan, UT 84321

Logan Regional Hospital Dayspring
1400 North 500 East
Logan, UT 84321

MIDVALE

Family Counseling Center
46 East 7200 South
Midvale, UT 84047

MOAB

Four Corners Mental Health Center MOAB Clinic
198 East Center
Moab, UT 84532

MOUNT PLEASANT

Central Utah Counseling Center
125 South State Street
Mount Pleasant, UT 84647

MATR
Mount Pleasant, UT 84647

NEPHI

Central Utah Counseling Center
656 North Main Street
Nephi, UT 84648

NORTH SALT LAKE

Life Line Inc
1130 West Center Street
North Salt Lake, UT 84054

OGDEN

Blue Skies Recovery Center, Inc.
727 24th Street
Ogden, UT 84102

Columbia Ogden Regional Medical Center
5475 South 500 East
Ogden, UT 84405

McKay/Dee Hospital Dayspring Chemical Dependency Unit
5030 Harrison Boulevard
Ogden, UT 84403

New Horizons Education Treatment and Consulting
205 26th Street, Suite 14
Ogden, UT 84401

Professional Services Corporation
533 26 Street
Suite 100
Ogden, UT 84401

Rocky Mountain Consultants
727 24th Street
Ogden, UT 84401

Weber Human Services
2650 Lincoln Avenue
Ogden, UT 84401

ORDERVILLE

Southwest Center
425 East State Street, Suite 11
Orderville, UT 84758

OREM

Addiction and Psychological Services
224 North Orem Boulevard
Orem, UT 84057

Assessment and Psychotherapy Assoc Inc
1411 North State Street, Suite 7
Orem, UT 84058

Utah County Council on Drug Abuse Rehabilitation (UCCODAR)
251 East 1200 South
Orem, UT 84058

PANGUITCH

Southwest Center
609 North Main Street
Panguitch, UT 84759

PARK CITY

Aspen Therapy Center
700 Bitner Road
Park City, UT 84098

Valley Mental Health Summit County Unit
1753 Sidewinder Drive
Park City, UT 84060

PAYSON

Columbia Mountain View Hospital Pavilion
1000 East Highway 6
Payson, UT 84651

PRICE

Four Corners Mental Health Center
276 South Carbon Avenue
Price, UT 84501

Price Clinic
575 East 100 South
Price, UT 84501

PROVO

Affiliated Family Treatment Center
1675 North Freedom Boulevard
Provo, UT 84604

Project Reality Utah County Site
150 East Center Street, Suite 1100
Provo, UT 84606

Provo Canyon School Substance Abuse Services
4501 North University Avenue
Provo, UT 84603

Heritage Center
5600 North Heritage School Drive
Provo, UT 84604

Utah County Human Services
1726 South Buckley Lane
Provo, UT 84606

RICHFIELD

Central Utah Counseling Substance Abuse Center
255 South Main Street
Richfield, UT 84701

Sevier County Alcohol and Drug Program
835 East 300 North
Richfield, UT 84701

ROOSEVELT

Northeastern Counseling Center
510 West 200 North
Roosevelt, UT 84066

SAINT GEORGE

Brightway at Saint George
115 West 1470 South
Saint George, UT 84770

Kolob Therapeutic Services, Inc.
437 South Bluff Street, Suite 202
Saint George, UT 84770

Southwest Center
354 East 600 South
Suite 202
Saint George, UT 84770

Reach Alcohol and Drug Outpatient
321 North Mall Drive, Suite 101
Saint George, UT 84770

Youth Services
628 South 300 East
Saint George, UT 84770

SALT LAKE CITY

Asian Association of Utah
1588 South Major Street 30 East
Salt Lake City, UT 84115

Assessment and Psychotherapy Association, Inc.
2114 East Fort Union Boulevard
Salt Lake City, UT 84121

Catholic Community Services
2570 West 1700 South
Salt Lake City, UT 84104

Center for Behavioral Health
1073 East 3300 South
Salt Lake City, UT 84106

Cornerstone Counseling Center
660 South 200 East
Suite 308
Salt Lake City, UT 84111

Drug Free Community
3646 South Redwood Road
Suite 1-A
Salt Lake City, UT 84119

England and Associates
5821 South Beaumont Drive
Salt Lake City, UT 84121

Family Counseling Center
807 East South Temple Street
Suite 350
Salt Lake City, UT 84102

First Step House
411 North Grant Street
Salt Lake City, UT 84116

Gateway to Recovery
320 West 200 South, Suite 230-B
Salt Lake City, UT 84101

Haven, The
974 East South Temple
Salt Lake City, UT 84102

Highland Ridge Hospital Substance Abuse Services
4578 Highland Drive
Salt Lake City, UT 84117

Latter-Day Saints Hospital Intermountain Health Care Dayspring Program
C Street and 8 Avenue
Salt Lake City, UT 84143

Neo Genesis
744 South 500 East
Salt Lake City, UT 84102

Northwest Passage, Inc.
432 North 300 West
Salt Lake City, UT 84103

Odyssey House, Inc.
Adolescent Facility
607 East 200 South
Salt Lake City, UT 84102

Adult Treatment Program
68 South 600 East
Salt Lake City, UT 84102

Intensive Outpatient Program
623 South 200 East
Salt Lake City, UT 84102

Women and Children's Program
42 South 500 East
Salt Lake City, UT 84102

Parents Helping Parents DBA Turnabout
2738 South 2000 East
Salt Lake City, UT 84109

Positive Adjustments Corporation
2480 South Main Street, Suite 108
Salt Lake City, UT 84115

Professional Services Corporation Substance Abuse Services
4667 South Halladay Boulevard
Salt Lake City, UT 84117

Project Reality
150 East 700 South
Salt Lake City, UT 84111

Residential Unit
1416 South State Street
Salt Lake City, UT 84115

Rocky Mountain Consultants
5278 Pinemount Drive
Suite A-120
Salt Lake City, UT 84107

Saint Mary's Home for Men
1206 West 200 South
Salt Lake City, UT 84104

Salt Lake County Division of Youth Services
177 West Price Avenue
Salt Lake City, UT 84115

Salvation Army Alcohol Rehabilitation Program
252 South 500 East
Salt Lake City, UT 84102

Sequoia Counseling Center
20738 South 2000 East, Suite B
Salt Lake City, UT 84109

University of Utah Alcohol and Drug Abuse Clinic
50 North Medical Drive
Room 1R52
Salt Lake City, UT 84132

University of Utah Neuropsychiatric Institute
501 Chipeta Way
Salt Lake City, UT 84108

Utah Alcoholism Foundation Combined Facilities
2880 South Main Street
Suite 210
Salt Lake City, UT 84115

House of Hope
1006 East 100 South, Suite 210
Salt Lake City, UT 84102

Progress Home
21 I Street
Salt Lake City, UT 84103

Utah Child and Youth Guidance Center
1414 East 4500 South, Suite 4
Salt Lake City, UT 84117

Valley Mental Health
East Valley Unit
1141 East 3900 South
Suite A-160
Salt Lake City, UT 84124

Forensic Unit
530 East 500 South, Suite 10
Salt Lake City, UT 84102

Alcohol and Drug Treatment Unit
5965 South 900 East, Suite 240
Salt Lake City, UT 84121

Veterans' Affairs Medical Center Substance Abuse Treatment Units
500 East Foothill Boulevard
Salt Lake City, UT 84148

Volunteers of America Alcohol and Drug Detoxification Center
252 West Brooklyn Avenue
Salt Lake City, UT 84101

Wasatch Canyons
Intermountain Health Care
5770 South 1500 West
Salt Lake City, UT 84123

Wasatch Youth Support Systems
3392 West 3500 South
Salt Lake City, UT 84120

SANDY

Positive Adjustments Corporation
870 East 9400 South, Suite 103-C
Sandy, UT 84094

SAINT GEORGE

Desert Hills Therapeutic Services, Inc.
1240 East 100 South, Suite 18-B
St George, UT 84790

TOOELE

Valley Mental Health Center
305 North Main Street
Tooele, UT 84074

TREMONTON

New Choices SAT Program
125 South 100 West
Tremonton, UT 84337

VERNAL

Mountain Valley Counseling
365 West 50 North, Suite W-1
Vernal, UT 84078

Uintah Basin Counseling Vernal Office
559 North 1700 West
Vernal, UT 84078

WASHINGTON

Counseling Services of Southern Utah
293 East Telegraph Road
Suite 35
Washington, UT 84780

WOODS CROSS

Benchmark Behavioral Health Services
592 West 1350 South
Woods Cross, UT 84087

VERMONT

BELLOWS FALLS

Healthcare and Rehab Services of Southeast Vermont
1 Hospital Court, Suite 410
Bellows Falls, VT 05101

BENNINGTON

United Counseling Service of Bennington County, Inc.
Ledge Hill Drive
Bennington, VT 05201

BRATTLEBORO

Alcohol/Drug Treatment
5 Fairview Street
Brattleboro, VT 05301

Brattleboro Retreat Adult Alcohol and Substance Abuse Program
75 Linden Street
Brattleboro, VT 05301

Families in Recovery
75 High Street
Brattleboro, VT 05301

Marathon Behavioral Treatment Services
101 Western Avenue
Brattleboro, VT 05301

Youth Services Incorporated
11 Walnut Street
Brattleboro, VT 05301

BURLINGTON

Act One
184 Pearl Street
Burlington, VT 05401

Champlain Drug and Alcohol Services
45 Clarke Street
Burlington, VT 05401

Howard Center for Human Services Pine Street Counseling Center
855 Pine Street
Burlington, VT 05401

Spectrum Youth and Family Services
31 Elmwood Avenue
Burlington, VT 05401

HUNTINGTON

Marathon, Inc. Mountain View Treatment Center
609 Delfrate Road
Huntington, VT 05462

MIDDLEBURY

Counseling Service of Addison County Substance Abuse Treatment Unit
89 Main Street
Middlebury, VT 05753

MONTPELIER

Dawnland Center
119 Barre Street
Montpelier, VT 05601

Washington County Youth Service Bureau
38 Elm Street
Montpelier, VT 05602

MORRISVILLE

Lamoille County Mental Health Services Substance Abuse Treatment Unit
520 Washington Highway
Morrisville, VT 05661

NEWPORT

Northeast Kingdom Mental Health Tri-County Substance Abuse Services
343 Main Street
Newport, VT 05855

RANDOLPH

Clara Martin Center Substance Abuse Treatment Unit
11 Main Street
Randolph, VT 05060

RUTLAND

Rutland Mental Health Service Evergreen Center for Alcohol/Drug Services
7 Court Square
Rutland, VT 05701

SAINT ALBANS

Northwestern Counseling and Support Services, Inc. Outpatient Treatment Unit
8 Ferris Street
Saint Albans, VT 05478

SAINT JOHNSBURY

Tri-County Substance Abuse Services
297 Summer Street
Saint Johnsbury, VT 05819-1605

SOUTH BURLINGTON

Adolescent Family Services
595 Dorset Street, Suite 6
South Burlington, VT 05403

Fletcher Allen Day One
200 Twin Oaks Terrace, Suite 6
South Burlington, VT 05403

UNDERHILL

Maple Leaf Farm Associates, Inc.
10 Maple Leaf Road
Underhill, VT 05489

WALLINGFORD

Recovery House
12 Church Street
Wallingford, VT 05773

WHITE RIVER JUNCTION

Health Rehabilitation Services of Southeastern Vermont
195 North Main Street, Suite 2
White River Junction, VT 05001-7044

Veterans Affairs Medical Center Substance Abuse Treatment Services
215 North Main Street
White River Junction, VT 05009

WILDER

Quitting Time
Depot Street
Wilder, VT 05088

WINOOSKI

Centerpoint
81 West Canal Street
Winooski, VT 05404-2111

VIRGINIA

ABINGDON

Highlands Community Services Substance Abuse Intensive Treatment Program
432 East Main Street, Suite A
Abingdon, VA 24210

ALEXANDRIA

Alexandria Community Services Board Substance Abuse Services
2355-A Mill Road
Alexandria, VA 22314

Franconia Road Treatment Center
6015 Bush Hill Drive
Alexandria, VA 22310

Living Free Alcohol and Chemical Dependence Program
6391 Little River Turnpike
Alexandria, VA 22312

Second Genesis, Inc.
1001 King Street
Alexandria, VA 22314

ANANDALE

Fairfax Methadone Treatment Center
7008-G Little River Turnpike
Annandale, VA 22003

ARLINGTON

Arlington County Alcohol and Drug Program
1725 North George Mason Drive
Arlington, VA 22205

Columbia Arlington Hospital
Addiction Treatment Program
1701 North George Mason Drive
Arlington, VA 22205

Northern Virginia Community Hospital
601 South Carlin Springs Road
Arlington, VA 22204

The Women's Home, Inc.
1628 North George Mason Drive
Arlington, VA 22205

Vanguard Services, Ltd. Phoenix Program
506 North Pollard Street
Arlington, VA 22203

ASHLAND

Hanover County Community Services Board
12300 Washington Highway
Ashland, VA 23005

BLACKSBURG

New River Valley Community Services Montgomery Clinic
700 University City Boulevard
Blacksburg, VA 24060-2706

CARTERSVILLE

Human Resources, Inc. Willow Oaks
2123 Cartersville Road
Cartersville, VA 23027

CEDAR BLUFF

Cumberland Mental Health Center Substance Abuse Services
Route 19
Cedar Bluff, VA 24609

CHARLOTTESVILLE

University of Virginia Hospitals North Ridge Hosp Addictions Treatment Program
2955 Ivy Road, Suite 210
Charlottesville, VA 22903

CHESAPEAKE

Chesapeake Substance Abuse Program
524 Albermarle Drive
Chesapeake, VA 23320

Virginia Beach Group at Chesapeake
300 Medical Parkway Suite 306
Chesapeake, VA 23320-4985

CHESTERFIELD

Chesterfield Substance Abuse Services
6801 Lucy Corr Court
Rogers Building
Chesterfield, VA 23832

CLINTWOOD

Dickenson County Community Services Substance Abuse Services
McClure Avenue Clinical Services Building
Clintwood, VA 24228

COLONIAL HEIGHTS

Behavior and Stress Management
3236 B Boulevard
Colonial Heights, VA 23834

COVINGTON

Allegheny Highlands Community Services Board Substance Abuse Services
305 South Monroe Avenue
Covington, VA 24426

CULPEPER

Pinebrook Psychiatric Center Substance Abuse Services
501 Sunset Lane
Culpeper, VA 22701

DANVILLE

Alcoholic Counseling Center, Inc. Hope Harbor
1021 Main Street
Danville, VA 24541

Associates in Mental Health Services
108 Holbrook Street, Suite 203
Danville, VA 24541

Interventions Counseling and Consulting Services
105 South Union Street, Suite 800
Danville, VA 24541

FAIRFAX

Dominion Hospital
11200 Waples Mill Road, Suite 100
Fairfax, VA 22030

Fairfax/Falls Church Community Services Board Alcohol and Drug Services
3900 Jermantown Road
Suite 200
Fairfax, VA 22030

Life Line Addictions Program
10565 Lee Highway
Suite 100
Fairfax, VA 22030

FALLS CHURCH

Ethos Foundation, Inc.
5201 Leesburg Pike
Suite 100
Falls Church, VA 22041

Innova Comprehensive Addiction Treatment Services (CATS)
3300 Gallows Road
Falls Church, VA 22046

FARMVILLE

Crossroads Community Services Board
Highway 460
Farmville, VA 23901

FISHERSVILLE

Recovery Choice Program at Augusta Medical Center
96 Medical Center Way
Fishersville, VA 22939

FORT EUSTIS

Fort Eustis Community Counseling Center Army Substance Abuse Program
515 Sternberg Avenue
Fort Eustis, VA 23604-5548

FORT LEE

Fort Lee Alcohol and Drug Community Counseling Center
Kenner Clinic
Building 12000
Fort Lee, VA 23801

FORT MONROE

Community Counseling Center Alcohol and Drug Prevention Control Office
Building T-194
Fort Monroe, VA 23651

FREDERICKSBURG

Rappahannock Area Community Services Board Alcohol and Drug Outpatient Services
600 Jackson Street
Fredericksburg, VA 22401

Serenity Home, Inc. Substance Abuse ICF and Halfway Treatment Services
514 Wolfe Street
Fredericksburg, VA 22401

Snowden at Fredericksburg
1200 Sam Perry Boulevard
Fredericksburg, VA 22401

FRONT ROYAL

Northwestern Community Services Board Substance Abuse Service
209 West Criser Road
Front Royal, VA 22630

GALAX

Galax Treatment Center, Inc. Life Center of Galax
112 Painter Street
Galax, VA 24333

GLEN ALLEN

Henrico Area MH/MR Services
10299 Woodman Road
Glen Allen, VA 23060

GLOUCESTER

Middle Peninsula/Northern Neck Counseling Center
9228 George Washington Memorial Highway
Gloucester, VA 23061

GOLDVEIN

Deep Run Lodge
13259 Blackwells Mill Road
Goldvein, VA 22720

GOOCHLAND

Goochland/Powhatan Community Services
3058 River Road West
Goochland, VA 23063

HAMPTON

Hampton Roads Clinic
2236 West Queen Street
Hampton, VA 23666

Langley Air Force Base Substance Abuse Program
Building 74
Hampton, VA 23665

Peninsula Behavioral Health Center
2244 Executive Drive
Hampton, VA 23666

Substance Abuse Treatment Program
100 Emancipation Road
Suite 116-A
Hampton, VA 23667

HARRISONBURG

Family Life Resource Center
250 East Elizabeth Street, Suite 102
Harrisonburg, VA 22801

Harrisonburg/Rockingham Community Services Board
1241 North Main Street
Harrisonburg, VA 22801

HOPEWELL

Columbia John Randolph Behavioral Health Center
504 North 3rd Street
Hopewell, VA 23860

LEESBURG

Graydon Manor Psychiatric Hospital
801 Childrens Center Road
Leesburg, VA 20175

Loudoun County Mental Health Center Substance Abuse Program
102 Heritage Way NE, Suite 302
Leesburg, VA 20176

LYNCHBURG

Community Services Board of Central Virginia Substance Abuse Services
2235 Landover Place
Lynchburg, VA 24501

Virginia Baptist Hospital Pathways Treatment Center
3300 Rivermont Avenue
Lynchburg, VA 24503

MANASSAS

Center for Psychiatric and Addiction Treatment
8700 Sudley Road
Manassas, VA 22110

MARION

Mount Rogers Transitions Substance Abuse Services
115 North Church Street
Marion, VA 24354

Southwestern Virginia Mental Health Institute Medical Detox Unit
502 East Main Street
Marion, VA 24354

MARTINSVILLE

Memorial Hospital of Martinsville and Henry County Psychiatric Services
320 Hospital Drive
Martinsville, VA 24115-4788

Passages
817 Starling Avenue
Martinsville, VA 24112

Patrick Henry Drug and Alcohol Council Crossroads/Intensive Outpatient Program
24 Clay Street
Martinsville, VA 24114

MECHANICSVILLE

Hanover Community Service Board Gail Taylor/LCSW
8157 Old Calvary Drive, Suite 102
Mechanicsville, VA 23111

MIDLOTHIAN

Rockwood Counseling Associates
10128 Hull Street Road
Midlothian, VA 23112

NEW KENT

Cumberland Hospital for Children and Adolescents
9407 Cumberland Road
New Kent, VA 23124

NEWPORT NEWS

CAPO Center Detox
2351 Terminal Avenue
Newport News, VA 23607

Comprehensive Outpatient Services Peninsula Alcoholism Services
11832 Canon Boulevard, Suite C
Newport News, VA 23606

Riverside New Foundations
610 Thimble Shoals Boulevard
Building 5, Suite 100-A
Newport News, VA 23606

Woodside Hospital LLC
17579 Warwick Boulevard
Newport News, VA 23603

NORFOLK

ARD/CAAC NAB LCREK AMPHIB Base
Building 3007
Norfolk, VA 23521-5000

Naval Alcohol Rehabilitation Center
1650 Gilbert Street
Norfolk, VA 23511

New Bridges
Bridges Outpatient Rehabilitation Center
6330 Newtown Road, Suite 200
Norfolk, VA 23502

Norfolk Community Services Board Substance Abuse Services
1150 East Little Creek Road
Suite 302
Norfolk, VA 23518

Rehabilitation Services, Inc.
300 West 20 Street
Norfolk, VA 23517

Sentara Norfolk General Hospital
600 Gresham Drive
Norfolk, VA 23507

NORTON

Saint Mary's Family Center
910 Virginia Avenue
Norton, VA 24273

PETERSBURG

District 19 Substance Abuse Services
20 West Bank Street
Petersburg, VA 23803

Poplar Springs Hospital Chemical Dependency Program
350 Poplar Drive
Petersburg, VA 23805

PILOT

Serenity House
Fisher's View
Pilot, VA 24138

PORTSMOUTH

T. W. Neumann and Associates
720 Rodman Avenue
Portsmouth, VA 23707

QUANTICO

Consolidated Substance Abuse Counseling Center
Marine Corps Base Quantico
2034 Barnett Avenue
Quantico, VA 22134-5012

RADFORD

Saint Albans Psychiatric Hospital Substance Abuse Services
Route 11 West
Radford, VA 24143

RICHMOND

Chippenham Medical Center
Johnston Willis Hospital
Tucker Pavilion
7101 Jahnke Road
Richmond, VA 23225

Deaf and Hard of Hearing Community Counseling Services
8917 Fargo Road
Richmond, VA 23229

Human Resources' Inc.
Division of Addiction Services/ Drug Free Unit
2926 West Marshall Street
Richmond, VA 23230

Outpatient Methadone Program
15 West Cary Street
Richmond, VA 23220

MCC Behavioral Care
7501 Boulders View, Suite 400
Richmond, VA 23225

Richmond Aftercare, Inc. Men's and Women's Program
1109 Bainbridge Street
Richmond, VA 23224

Saint Marys Hospital
Outpatient Services
2006 Bremo Road, Suite 102-A
Richmond, VA 23226

Psychiatric Unit Substance Abuse Services
5801 Bremo Road, 7th Floor
Richmond, VA 23226

Veterans' Affairs Medical Center Substance Abuse Treatment Program
1201 Broad Rock Boulevard
Richmond, VA 23249

Virginia Health Center
2203 East Broad Street
Richmond, VA 23223

Williamsburg Place of Richmond
10049 Midlothian Turnpike, Suite B-2
Richmond, VA 23235

ROANOKE

Bethany Hall Women's Recovery Home Chemical Dependency Treatment
1109 Franklin Road SW
Roanoke, VA 24016

Walnut Avenue Clinic
16 Walnut Avenue SW
Roanoke, VA 24016

SALEM

Lewis Gale Medical Center Center for Recovery
1902 Braeburn Drive
Salem, VA 24153

Veterans Affairs Medical Center Substance Abuse Treatment Program
1970 Roanoke Boulevard
Psychiatry 116A-4
Salem, VA 24153

SOUTH BOSTON

Southside Community Services Board Substance Abuse Treatment Services
424 Hamilton Boulevard
South Boston, VA 24592

STAUNTON

Shenandoah Counseling Associates
1048 West Beverley Street
Staunton, VA 24401

Valley Alcohol Safety Action Program
Holiday Court
Suite B
Staunton, VA 24401

STEPHENSON

Shalom Et Benedictus, Inc.
1160 Jordan Springs Road
Stephenson, VA 22656

SUFFOLK

Psychiatric Care Center Dr. Richard Key
1900 North Main Street, Suite 207
Suffolk, VA 23434

Western Tidewater Mental Health Center Substance Abuse Department
157 North Main Street
Suffolk, VA 23434

SURRY

District 19 MH/MR Substance Abuse Services Surry Counseling Service
474 Colonial Trail West
Surry, VA 23883

VIRGINIA BEACH

Addiction Rehabilitation Department Counseling and Assistance Center
Building 531 NAS Oceana
Virginia Beach, VA 23460

Atlantic Psychiatric Services
780 Lynnhaven Parkway, Suite 220
Virginia Beach, VA 23452

Crisis Intervention Home
811 13th Street
Virginia Beach, VA 23451

First Hospital Corp Recovery Place and Serenity Lodge at the Beach
1100 First Colonial Road
Virginia Beach, VA 23454

Virginia Beach Substance Abuse Services
Pembroke Six Street
Suite 126
Virginia Beach, VA 23462

WARRENTON

Family Focus Counseling Service
20-B John Marshall Street
Warrenton, VA 20186

Rappahannock/Rapidan CSB
Fauquier Family Guidance
340 Hospital Drive
Warrenton, VA 20186

WILLIAMSBURG

Bacon Street, Inc.
247 McLaws Circle
Williamsburg, VA 23185

Colonial Services Board Substance Abuse Services
1657 Merrimac Trail
Williamsburg, VA 23185

Williamsburg Place
5447 Mooretown Road
Williamsburg, VA 23185

WINCHESTER

First Step
129 Youth Develpment Court
Winchester, VA 22602

Lord Fairfax Community, Inc. Council on Alcoholism
512 South Braddock Street
Winchester, VA 22601

Winchester Medical Center Choices Detox
1890 Amherst Street
Winchester, VA 22601

WOODSTOCK

Northwestern Community Services Board
441 North Main Street
Woodstock, VA 22664

WASHINGTON

ABERDEEN

Grays Harbor Community Hospital Eastcenter Recovery
1006 H Street
Aberdeen, WA 98520

Social Treatment Opportunity Programs (STOP)
2700 Simpson Avenue
Aberdeen, WA 98520

ANACORTES

Follman Agency
1004 7th Street, Suite 207
Anacortes, WA 98221

SKAGIT Recovery Center
1010-A 6th Street
Anacortes, WA 98221

ARLINGTON

Focus
436 West Avenue North
Arlington, WA 98223

M K Standish and Associates, Inc.
16404 Smokey Point Boulevard
Suite 109
Arlington, WA 98223

Stillaguamish Tribe Substance Abuse Services
3439 Stoluckguamish Lane
Arlington, WA 98223

AUBURN

Future Visions (FVP Enterprises) DBA Social Treatment Opportunity Programs
620 M Street NE
Auburn, WA 98002

Lakeside Milam Recovery Centers
1833 Auburn Way North, Suite A
Auburn, WA 98002

Muckleshoot Tribal Alcohol and Drug Program
39015 172nd Avenue South East
Auburn, WA 98092-9763

BAINBRIDGE ISLAND

Bainbridge Island Recovery Center, Inc.
600 Winslow Way East, Suite 135
Bainbridge Island, WA 98110

BELLEVUE

C and P Counseling
1200 112th Avenue NE
Suite C-179
Bellevue, WA 98004

Coastal Treatment Services
12443 Bel Red Road
Building 300, Suite 320
Bellevue, WA 98005

Group Health Behavioral Health Services
13451 SE 36th Street
Bellevue, WA 98006

Open Door Behavioral Health Services
2840 Northup Way
Bellevue, WA 98004

Youth Eastside Services (YES)
16150 NE 8th Street
Bellevue, WA 98008

BELLINGHAM

Belair Clinic
1130 North State Street
Bellingham, WA 98225

Chambers and Wells Counseling
1130 North State Street
Bellingham, WA 98225

Lummi Care Program
1790 Bayon Road
Bellingham, WA 98226

Pacific Recovery Healing Center
2502 Cedarwood Avenue, Suite 3
Bellingham, WA 98225-1464

Saint Joseph Hospital Recovery Center Inpatient and Outpatient
809 East Chestnut Street
Bellingham, WA 98225-5298

Saint Josephs Recovery House
1209 Girard Street
Bellingham, WA 98225

Sea Mar Substance Abuse Program
2209 Elm Street, Suite AZC
Bellingham, WA 98225

Sehome Behavioral Health, Inc.
1116 Key Street
Bellingham, WA 98225-5224

BOTHELL

Alpha Center for Treatment, Inc.
10614 Beardslee Boulevard
Suite D
Bothell, WA 98011

Residence XII
14506 Juanita Drive NE
Bothell, WA 98011

BREMERTON

Agape Unlimited
5464 Kitsap Way
Bremerton, WA 98312

Group Health Adapt/Bremerton
5002 Kitsap Way
Suite 202
Bremerton, WA 98312

Kitsap Mental Health Services Youth MICA Program
5455 Almira Drive
Bremerton, WA 98312

Kitsap Recovery Center
1975 NE Fuson Road
Bremerton, WA 98310

Navy Alcohol Treatment Department
1400 Farragut Avenue
Building 491, 2nd Floor
Bremerton, WA 98314-5001

Olympic Educational Services District 114/Youth Recovery Program
105 National Avenue North
Bremerton, WA 98312

Right Choice Counseling Service
1740 Northeast Riddell Road
Suite 314
Bremerton, WA 98310

Tara Counseling Center, Inc.
3627 Wheaton Way, Suite F
Bremerton, WA 98310

BURIEN

South King County Recovery Centers
15025 4th Avenue SW
Burien, WA 98166-2301

BURLINGTON

Follman Agency
127 South Spruce Street
Burlington, WA 98233

CASTLE ROCK

Drug Abuse Prevention Center
2232 South Silverlake Road
Castle Rock, WA 98611

CATHLAMET

Wahkiakum Chemical Dependency Services
42 Elochoman Valley Road
Cathlamet, WA 98612

CENTRALIA

New Directions Counseling
1000 Kresky Road, Suite G
Centralia, WA 98531

Omni Program
20311 Old Highway 9 SW
Centralia, WA 98531-9699

CHEHALIS

Eugenia Center
249 NW Chehalis Avenue
Chehalis, WA 98532-1371

Green Hill School Day Treatment Program
375 SW 11th Street
M-S S21-5
Chehalis, WA 98532

Right Step, Inc.
118 North Market Boulevard
Chehalis, WA 98532

CHELAN

Riverview Recovery
219 West Gibson Avenue
Chelan, WA 98816

CHEWELAH

Stevens County Counseling Services
East 301 Clay Street, Room 210
Chewelah, WA 99109

COLVILLE

Stevens County Counseling Services
165 East Hawthorne Avenue
Colville, WA 99114

CONCRETE

Sunlight Again, Inc.
310 Dillard Avenue
Concrete, WA 98237-9643

DAVENPORT

Lincoln County Alcohol/Drug Center
518 Morgan Street
Davenport, WA 99122

DAYTON

Columbia County Services Substance Abuse Program
221 East Washington Street
Dayton, WA 99328

DEER PARK

Deer Park Recovery
South 22 Vernon Street, Suite 4
Deer Park, WA 99006

Salvation Army Drug Abuse Outpatient
West 110 Crawford Street
Deer Park, WA 99006

DES MOINES

Sea Mar Treatment Center
24215 Pacific Highway South
Des Moines, WA 98198

EDMONDS

New Spirit Recovery Program
22617 76th Avenue W, Suite 1001
Edmonds, WA 98026

Lakeside Milam Recovery North
7935 Lake Ballinger Way
Edmonds, WA 98026

ELLENSBURG

Alcohol/Drug Dependency Service
507 Nanum Street
Room 111
Ellensburg, WA 98926

Kittitas Valley Recovery Services
103 East 4th Street, Suite 204
Ellensburg, WA 98926

Parke Creek Chemical Dependency Program
11042 Parke Creek Road
Ellensburg, WA 98926

ELMA

Northwest Indian Treatment Center
Elma, WA 98541

ENUMCLAW

Dotters Counseling Service, Inc.
847 Blake Street
Enumclaw, WA 98022

South King County Recovery Centers
1325 Cole Street
Enumclaw, WA 98022

EVERETT

Catholic Community Services Lifeline Recovery Program
1918 Everett Avenue
Everett, WA 98201

Everett Treatment Services
7207 Evergreen Way, Suite M
Everett, WA 98203

Evergreen Manor, Inc.
Outpatient Services
Recovery House/Detox Services
2601 Summit Avenue
Everett, WA 98201

Family Counseling DBA Northwest Alternatives
9930 Evergreen Way
Everett, WA 98208

Focus
909 SE Everett Mall Way
Suite C-364
Everett, WA 98204

Lakeside/Milam Recovery Centers, Inc.
2731 Wetmore Avenue, Suite 402
Everett, WA 98201

North Sound Assesment and Counseling Service
1316 Wall Street, Suite 1-B
Everett, WA 98201

Pacific Treatment Alternatives
1114 Pacific Avenue
Everett, WA 98201

Sea Mar Community Health Center
8625 Evergreen Way, Suite 255
Everett, WA 98201

EVERSON

Nooksack Tribes Genesis II
6750 Mission Road
Everson, WA 98247

FAIRCHILD AIR FORCE BASE

Fairchild Air Force Base Mental Health Services
200 North Chennault Street
Fairchild AFB, WA 99011

FEDERAL WAY

Federal Way Youth and Family Services
1411 Dash Point Road SW
Federal Way, WA 98023

Intercept Associates
30620 Pacific Highway South
Suites 107
Federal Way, WA 98003

Lakeside Milam Recovery Centers
28621 Pacific Highway South
Federal Way, WA 98003

Sundown M Ranch
720 South 333rd Street, Suite 105
Federal Way, WA 98003-6399

Valley Cities Counseling and Consultation
33301 1st Way South, Suite C-115
Federal Way, WA 98003

Western Clinical Health Services
2025 South 341st Place
Federal Way, WA 98003

FERNDALE

Jerry F. Starr Memorial Foundation Avalon Counseling and Treatment Services
5778 2nd Avenue, Suite B
Ferndale, WA 98248

FORKS

West End Outreach Services Forks Community Hospital
550 5th Street
Forks, WA 98331

FORT LEWIS

Alcohol and Drug Abuse Prevention and Control Program
HQ I Corps/Fort Lewis
Building 2006 Room 206
Fort Lewis, WA 98433

FRIDAY HARBOR

San Juan Community Alcoholism Center
955 Guard Street
Friday Harbor, WA 98250

GIG HARBOR

Center Peninsula
7116 Pioneer Way
Gig Harbor, WA 98335

Gig Harbor Counseling and Recovery Center
5112 Olympic Drive NW
Gig Harbor, WA 98335

GOLDENDALE

Goldendale Branch White Salmon Counseling
777 East Broadway Street, Suite 1
Goldendale, WA 98620

Kick/It Counseling
104 East Main Street
Goldendale, WA 98620-9005

GRANDVIEW

Phoenix Addiction Counseling Services
242 Division Street
Grandview, WA 98930

HOQUIAM

Evergreen Chemical Dependency Program
804 Levee Street
Hoquiam, WA 98550

Grays Harbor Crisis Clinic Detox Unit
615 8th Street
Hoquiam, WA 98550

ISSAQUAH

Friends of Youth Issaquah
414 Front Street
Issaquah, WA 98027

Lakeside/Milam Issaquah Outpatient
98 North East Gilman Street
Suite 200
Issaquah, WA 98027

KELSO

Drug Abuse Prevention Center
214 North Pacific Avenue
Kelso, WA 98626

First Place Inc
309 Oak Street
Kelso, WA 98626

KENMORE

Residence XII
14506 Juanita Drive NE
Kenmore, WA 98028

KENNEWICK

Action Chemical Dependency Center
552 North Colorado Street
Suite 114
Kennewick, WA 99336

Advocates for Wellness
120 Vista Way
Kennewick, WA 99336

Discovery Substance Abuse Services
5219 West Clearwater Avenue
Suite 9
Kennewick, WA 99336

Life Changes Chemical Dependency Agency
313 North Morain Street
Kennewick, WA 99336

KENT

Comprehensive Alcohol Services
1609 South Central Avenue
Suite 1
Kent, WA 98032

Hope Recovery Services
10820 South Kent-Kangley Road
Kent, WA 98031

Kent Youth and Family Services
232 South 2 Avenue
Suite 201
Kent, WA 98032

South King County Recovery Centers
505 South Washington Avenue
Kent, WA 98032

KINGSTON

Port Gamble Klallam Recovery Center
32272 Little Boston Road NE
Kingston, WA 98346

KIRKLAND

Lakeside Milam Recovery Centers
10422 NE 37th Circle, Suite B
Kirkland, WA 98033

10322 NE 132nd Street
Kirkland, WA 98034

McClure and Associates Counseling
11416 Slater Avenue NE, Suite 202
Kirkland, WA 98033

Youth Eastside Services Lake Washington
13009 85th Street
Kirkland, WA 98033

LAKEWOOD

Moms and Women's Recovery Center
9609 Bristol Street
Lakewood, WA 98499

LA PUSH

Quileute Family and Health Services
560 Quileute Heights Road
La Push, WA 98350

LONGVIEW

Chance for Change
828 12th Avenue, Suite B
Longview, WA 98632

Starting Point, Inc.
1315 Hemlock Street
Longview, WA 98632

LYNDEN

The Center
310 5th Street
Lynden, WA 98264-1911

LYNNWOOD

Crosby Enterprises, Inc.
3924 204 Street SW
Lynnwood, WA 98036

Family Counseling Service DBA Northwest Alternatives
4230 198th Street SW, Suite 100
Lynnwood, WA 98036-672

Options Treatment and Evaluations
15620 Highway 99, Suite 10
Lynnwood, WA 98037

Pacific Treatment Alternatives
19324 40 Avenue West
Suite A
Lynnwood, WA 98036

MAPLE VALLEY

Cedar Hills Treatment Center
15900 227 Avenue SE
Maple Valley, WA 98038

MARYSVILLE

Northwest Alternatives
1410 7th Street
Marysville, WA 98270

Tulalip Tribal Alcoholism Program
6700 Todum Road
Marysville, WA 98271

Tulalip Tribes Recovery Home
2821 Mission Hill Road
Marysville, WA 98271

MCCHORD AIR FORCE BASE

62 MDG/SGOHA Alcohol and Drug Abuse Prevention and Treatment
Building 100, Room 3012
McChord AFB, WA 98438

MEDICAL LAKE

Pine Lodge Pre-Release
751 Pine Street
Medical Lake, WA 99022

MONROE

Alpha Center for Treatment, Inc.
18962 South Route 2, Suite A
Monroe, WA 98272

Drug Abuse Council of Snohomish County
909 West Main Street, Suite 9
Monroe, WA 98272

Family Counseling DBA Northwest Alternatives
18962 State Road 2, Suite A
Monroe, WA 98272

Valley General Hospital Alcohol and Drug Recovery Center
14701 179 Street SE
Monroe, WA 98272

MONTESANO

Healthy Risk Counseling Center
330 Pioneer Avenue West
Montesano, WA 98563

MOSES LAKE

Grant County Alcohol and Drug Center
510 West Broadway
Moses Lake, WA 98837

MOUNT VERNON

Skagit Community Mental Health Center
916 South 3rd Street
Mount Vernon, WA 98273

Skagit Recovery Center John King Recovery House
1905 Continental Place
Mount Vernon, WA 98273

NASELLE

Naselle Youth Camp Bridge
11 Youth Camp Lane
Naselle, WA 98638

NESPELEM

Colville Tribal Alcohol Drug Program
Confederated Tribes of Colville Street
Nespelem, WA 99155

NEWPORT

Pend Oreille County Mental Health
325 South Spokane Street
Newport, WA 99156

OAK HARBOR

Recovery Center Island County
231 SE Barrington Drive
Suite 209
Oak Harbor, WA 98277

OAKVILLE

Tsapowum Chehalis Tribal Chemical Dependency Program
420 Howanut Drive
Oakville, WA 98568

OLALLA

Olalla Recovery Center
12851 Lala Cove Lane SE
Olalla, WA 98359

OLYMPIA

BHR Recovery Services
317 East 4th Avenue
Olympia, WA 98501

Group Health Cooperative Behavioral Health Services
700 North Lilly Road NE
Olympia, WA 98506

Northwest Resources
2742 Pacific Avenue
Olympia, WA 98506

Olympic Counseling Services/ Tamarc
1625 Mottman Road SW
Olympia, WA 98502

Recovery Associates
317 Fourth Avenue East
Olympia, WA 98501

Right Step, Inc.
3929 Martin Way East
Suites A and B
Olympia, WA 98506

OMAK

Okanogan County Counseling Services Chemical Dependency Programs
307 South Main Street
Omak, WA 98841

OTHELLO

Adams County Community Counseling Service Alcohol/ Drug Abuse Program
165 North First Street
Suite 120
Othello, WA 99334

PARKLAND

Moms and Women's Recovery Center
12108 Pacific Avenue
Parkland, WA 98444

PASCO

Benton Franklin Detox Services
1020 South 7th Street
Pasco, WA 99301

Unity Counseling Services
303 North 20 Street
Pasco, WA 99301

POMEROY

Rogers Counseling Center
856 Main Street
Pomeroy, WA 99347

PORT ANGELES

Healthy Families of Clallam County
1914 West 18th Street
Port Angeles, WA 98363

Lower Elwha Chemical Dependency Program
22 Kwitsen Drive
Port Angeles, WA 98362

Peninsula Community Mental Health Center Substance Abuse Services
118 East 8 Street
Port Angeles, WA 98362

Woodlands
1225 East Front Street
Port Angeles, WA 98362

PORT ORCHARDS

Olympic Educational Services/ District 114
1962 Hoover Avenue SE
Port Orchard, WA 98366

Port Orchard Counseling Recovery Center
1950 Pottery Avenue
Port Orchard, WA 98366

West Sound Treatment Center
120 Bethel Avenue
Port Orchard, WA 98366

PORT TOWNSEND

Jefferson County Community Recovery Center
1200 Sims Way
Port Townsend, WA 98368

Safe Harbor Recovery Center
686 Lake Street, Suite 400
Port Townsend, WA 98368-2272

PULLMAN

Abstemious Outpatient Clinic
10525 East Main Street, Suite P
Pullman, WA 99206

Whitman County Alcohol Center
NE 340 Maple Street
Room 2
Pullman, WA 99163

PUYALLUP

Counselor
315 39 Avenue SW
Suite 11
Puyallup, WA 98373

Horizon Treatment Services
11212 94th Avenue East, Suite B
Puyallup, WA 98373-3656

Lakeside Recovery Center
12812 101st Avenue, Suite 103
Puyallup, WA 98373

Shared Health Services
10116 116th Street East
Suite 202 and 102
Puyallup, WA 98373

REDMOND

Group Health Cooperative Alcohol and Drug Abuse Unit
2700 152 Avenue NE
Redmond, WA 98052

Square One Redmond
7811 159th Place NE
Redmond, WA 98052-7301

RENTON

Lakeside/Milam Recovery Centers, Inc. South
1000 SW 7th Street
Renton, WA 98055

Renton Area Youth Services (RAYS)
1025 South 3 Street
Renton, WA 98055

Valley Medical Recovery Center
400 South 43 Street
Renton, WA 98055

REPUBLIC

Change Point of Ferry County Community Services
42 North Klondike Road
Republic, WA 99166

RICHLAND

Carondelet/Lourdes ADTP
1175 Carondelet Drive
Richland, WA 99352

Choices and Changes, Inc.
1236 Columbia Drive SE
Richland, WA 99352

SEATTLE

Addiction Recovery Systems
720 Broadway
Seattle, WA 98125

Alternatives
1530 Eastlake Avenue East
Suite 305
Seattle, WA 98109

Associated Behavioral Health
120 Northgate Plaza
Northgate Medical Building
Suite 355
Seattle, WA 98125

Bissell Institute
22620 7th Avenue South
Seattle, WA 98198

Catholic Community Services
100 23rd Avenue South
Seattle, WA 98144

Central Seattle Recovery Center
464 12th Avenue Suite 300
Seattle, WA 98122

Detoxification Unit
1309 Summit Avenue
Seattle, WA 98101

Central Youth and Family Services
1901 Martin Luther King Jr. Way South
Seattle, WA 98144

Chrysalis Recovery Inc
816 North 38th Street
Seattle, WA 98103

Circle of Recovery
1207 North 200th Street
Seattle, WA 98133

Cocaine Outreach and Recovery Programs
1509 East Madison Street, Suite 101
Seattle, WA 98122

Consejo Counseling and Referral Services
3808 South Angeline Street
Seattle, WA 98118

Dykeman, Ruth Youth and Family Service
15001 8 Avenue SW
Seattle, WA 98166

Evergreen Treatment Services
1700 Airport Way South
Seattle, WA 98134-1618

1740 Airport Way South
Seattle, WA 98134-1618

Genesis House
621 34 Avenue
Seattle, WA 98122

Group Health/Behavioral Health
1730 Minor Avenue
Suite 1400
Seattle, WA 98101

Guardian Recovery Program
4812 Aurora Avenue North
Seattle, WA 98121

Highline West
2600 SW Holden Street
Seattle, WA 98126-3505

Iwasil Youth Program
102 Prefontaine Place
Seattle, WA 98104

Milam Recovery Program
12845 Ambaum Boulevard SW
Seattle, WA 98146

Perinatal Treatment Services
1005 East Jefferson Street
Seattle, WA 98122

Praxis
1319 Dexter Avenue North, Suite 290
Seattle, WA 98109

2825 Eastlake Avenue East
Suite 305
Seattle, WA 98102

Professional Health Associates
610 NW 44th Street
Seattle, WA 98107-4431

Recovery Options Northwest
2150 North 107th Street
Suite 200
Seattle, WA 98133-9009

Ryther Child Center
Adolescent Alcohol and Substance Abuse Program
2400 NE 95th Street
Seattle, WA 98115-2499

Safeco Safe House
11729 1/2 36th Avenue NE
Seattle, WA 98125

Schick Shadel Hospital Substance Abuse Program
12101 Ambaum Boulevard SW
Seattle, WA 98146

Seadrunar
Phase I/Georgetown
976 South Harney Street
Seattle, WA 98108

Queenanne
200 West Comstock Street
Seattle, WA 98119

Seattle Indian Health Board Alcohol/Drug Outpatient
611 12 Avenue South
Seattle, WA 98144

Shamrock Group, Inc.
8535 Phinney Avenue North
Seattle, WA 98103

Shared Health Services
14900 Interurban Avenue South
Suite 215
Seattle, WA 98168

Stonewall Recovery Services
430 Broadway East
Seattle, WA 98107

Sunrise Centers
12650 First Avenue South
Seattle, WA 98168

Swedish Medical Center
Addiction Recovery Program
5300 Tallman Street NW
Seattle, WA 98104

Therapeutic Health Services, Inc. Midvale Treatment Center
1116 Summit Avenue
Seattle, WA 98101

Thunderbird Treatment Center
9236 Renton Avenue South
Seattle, WA 98118

Trexam Program
1530 Eastlake Avenue East
Suite 203
Seattle, WA 98102

Veterans' Affairs Medical Center Addiction Treatment Center
1660 Columbian Way South
Seattle, WA 98108

Virginia Mason Chemical Dependency Program
1100 Olive Way
Metro Park West Tower
Suite 1000
Seattle, WA 98101

Washington Asian Pacific Islander Families
606 Maynard Avenue South
Suite 106
Seattle, WA 98104-2957

Women's Recovery Center
4649 Sunnyside Avenue North
Suite 200
Seattle, WA 98103

SEDRO WOOLLEY

Pioneer Center North
2275 Thompson Drive
Sedro Woolley, WA 98284

Safe Passage NPO
2268 Hub Drive
Sedro Woolley, WA 98284

United Northwest Recovery Center, Inc.
605 B Sunset Park Drive
Sedro Woolley, WA 98284-1578

SEQUIM

Jamestown S. Klallam Chemical Dependency Program
1032 Old Blyn Highway
Sequim, WA 98382

Safe Harbor Recovery Center
271 South 7th Avenue, Suite 23
Sequim, WA 98382-3633

SHELTON

Olympic Counseling Services/ Tamarc
615 Alder Street
Shelton, WA 98584

Recovery Associates
110 West K Street
Shelton, WA 98584

Skokomish Tribe Alcohol/Drug Program Hope
North 80 Tribal Center Road
Shelton, WA 98584

Squaxin Island Health Clinic
70 Squaxin Lane SE
Shelton, WA 98584

Right Step, Inc.
111 East Railroad Avenue
Shelton, WA 98584

SHORELINE

Center for Human Services
17018 15th Avenue NE
Shoreline, WA 98155

Therapeutic Health Services
17962 Midvale Avenue North, Suite 150
Shoreline, WA 98133-4922

SILVERDALE

Cascade Recovery Center Silverdale
9095 McConnell Avenue
Silverdale, WA 98383

SNOQUALMIE

Echo Glen Children's Center Exodus
33010 SE 99 Street
Snoqualmie, WA 98065

SPOKANE

Abstemious Outpatient Clinic, Inc.
1007 West Francis Avenue
Spokane, WA 99205

Addiction Recovery Systems, Inc.
West 601 Francis Avenue
Spokane, WA 99205

American Behavioral Health Systems, Inc.
3400 West Garland Street
Spokane, WA 99205

Behavioral Health Services
2703 North Pittsburgh Street
Spokane, WA 99209

Colonial Clinic
N 910 Washington Street
Suite 210
Spokane, WA 99204

Community Detox Services of Spokane
165 South Howard Street
Spokane, WA 99204

Daybreak of Spokane
11707 East Sprague, Suite D4
Spokane, WA 99206

Intensive Inpatient Program for Youth
Outpatient Treatment
628 South Cowley
Spokane, WA 99223

Deaconess Medical Center Chemical Dependency Unit
800 West 5th Avenue
Spokane, WA 99210

Group Health Northwest Chemical Dependency Program
322 West North River Drive
Spokane, WA 99201

Healing Lodge of The Seven Nations Youth Treatment Center
5600 East 8th Avenue
Spokane, WA 99212

Isabella House
West 2308 3 Avenue
Spokane, WA 99204

Lakeside Recovery Centers
601 West Mallon Avenue, Suite C
Spokane, WA 99201

Native Project
1803 West Maxwell Street
Spokane, WA 99201

New Horizon Counseling Services
West 2317 3 Avenue
Spokane, WA 99204

Spokane Addiction Recovery Centers (SPARC)
1509 West 8 Avenue
Spokane, WA 99204

1508 West 6th Avenue
Spokane, WA 99204

West 1403 7th Avenue
Spokane, WA 99204

Spokane Regional Health District
1101 West College Avenue
Spokane, WA 99201

Stepps/YFA Connections
901 East 2nd Avenue Suite 100
Spokane, WA 99202-2257

Sun Ray Court
518 South Browne Street
Spokane, WA 99202

Veterans' Affairs Medical Center Substance Abuse Treatment Program
4815 North Assembly Street
Spokane, WA 99205

STEVENSON

Skamania County Counseling Center
683 SW Rock Creek Drive
Stevenson, WA 98648

SUMNER

Moms and Women's Recovery Center
930 Alder Street
Sumner, WA 98390

Prosperity Counseling and Treatment Services, Inc.
1723 Bonney Avenue, Suite A
Sumner, WA 98390

The Center East
1110 Fryar Avenue
Sumner, WA 98390

SUNNYSIDE

Merit Resource Services
702 East Franklin Street
Sunnyside, WA 98944

SUQUAMISH

Suquamish Wellness Program
18465-A Augusta Avenue
Suquamish, WA 98392

TACOMA

Action Association Counseling Services
923 Martin Luther King Jr. Way
Tacoma, WA 98405-4149

Affirmation Counseling Services
4301 South Pine Street
Suite 30
Tacoma, WA 98409

Crossroads Treatment Center
6403 Lakewood Drive West
Tacoma, WA 98467

Griffin and Griffin EAP, Inc.
4218 South Steele Street
Suite 304
Tacoma, WA 98409

Health Department Methadone Treatment Program
3629 South D Street MS-049
Tacoma, WA 98408

Horizon Treatment Services
2607 Bridgeport Way West
Suite 2-J
Tacoma, WA 98466

Lakeside/Milam Recovery Centers, Inc.
535 Dock Street, Suite 104
Tacoma, WA 98402

Moms and Women's Recovery Center
2367 Tacoma Avenue South
Tacoma, WA 98402

Pierce County Alliance
510 Tacoma Avenue South
Tacoma, WA 98402

Puyallup Tribal Treatment Center
2209 East 32 Street
Tacoma, WA 98402

Reflections Recovery and Learning Center
8907-C Gravelly Lake Drive SW
Tacoma, WA 98499-3109

Remann Hall Alcohol and Drug Development Program (RHADD)
5501 6th Avenue
Tacoma, WA 98406-2697

Serenity Counseling Services
4410 East 20th Street
Tacoma, WA 98424

Shared Health Services
9112 Lakewood Drive SW
Suite 208
Tacoma, WA 98499

Social Treatment Opportunity Programs (STOP)
4301 South Pine Street, Suite 112
Tacoma, WA 98409

Tacoma Detoxification Center
721 Fawcett Avenue
Room 100
Tacoma, WA 98402

The Center MDC
721 South Fawcett Street
Suite 203
Tacoma, WA 98402

The Center South
10510 Gravelly Lake Drive SW
Tacoma, WA 98498

Transitions Limited
1004 72nd Street
Tacoma, WA 98445

Upper Tacoma Treatment Service
2367 South Tacoma Avenue
Tacoma, WA 98402

Western Washington Alcohol Center, Inc.
504 South 112th Street
Suite 214
Tacoma, WA 98409

TAHOLAH

Quinault Indian Nation Alcoholism Treatment Program
116 Quinault Street
Taholah, WA 98587

TOKELAND

Shoalwater Bay Tribe Counseling Chemical Dependency Program
4138 Shoalwater Bay Drive
Tokeland, WA 98590

TOPPENISH

Merit Resource Services
307 Asotin Street
Toppenish, WA 98948

Phoenix Support Services, Inc.
304 Monroe Street
Toppenish, WA 98948

USK

Kalispel Tribe Social Services Alcohol Program
Usk, WA 99180

VANCOUVER

Clark County Council on Alcohol and Drugs 8th Street Branch
509 West 8 Street
Vancouver, WA 98660

Columbia Treatment Services
7017 NE Highway 99, Suite 114
Vancouver, WA 98665

John Owen Recovery House
1950 Fort Vancouver Way
Vancouver, WA 98663

Rivercrest Treatment Center
1815 D Street
Vancouver, WA 98663

Starting Point
2703 East Mill Plain Boulevard
Vancouver, WA 98661

Western Psychological and Counseling Services
5305 East 18th Street
Suite A-East
Vancouver, WA 98661-6582

WALLA WALLA

Chemical Dependency Treatment Program DVAMC
77 Wainwright Drive
Suite 112-MH
Walla Walla, WA 99362

WAPATO

Merit Resource Services
312 West 2 Street
Wapato, WA 98951

WELLPINIT

Spokane Tribe of Indians Tribal Alcoholism and Drug Abuse Program
Old School Lane
Wellpinit, WA 99040

WENATCHEE

Center for Alcohol and Drug Treatment Casa, Inc.
327 Okanogan Avenue
Wenatchee, WA 98801

Olympic Counseling Services
766 South Mission Street
Wenatchee, WA 98801-3052

Quality Resources
6 First Street, Suite 6
Wenatchee, WA 98801

WHITE SALMON

White Salmon Counseling
1000 Jewett Boulevard, Suite 4
White Salmon, WA 98672

WOODINVILLE

Motivations
17311 135 Avenue NE
Suite C-400
Woodinville, WA 98072

YAKIMA

A J Alcohol and Drug Services
32 North 3 Street
Room 310
Yakima, WA 98901

Barth Clinic
414 North 2nd Street
Suite 2
Yakima, WA 98901

Central Washington Comprehensive Mental Health Drug Program
321 East Yakima Avenue
Yakima, WA 98901

James Oldham Treatment Center
308 North 4th Street
Yakima, WA 98907

Riel House
1408 West Yakima Avenue
Yakima, WA 98902

Sundown M Ranch
2280 SR 821
Yakima, WA 98901

Triumph Treatment Services, Inc. Community Drug and Alcohol Center
102 South Naches Avenue
Yakima, WA 98901

Yakima Human Services DBA Dependency Health Services
315 Holton Avenue, Suite B-1
Yakima, WA 98902

Detox Unit
401 South 5th Avenue
Yakima, WA 98902

YELM

Resolution A Counseling Service
10501 Creek Street SE
Suite 4
Yelm, WA 98597

WEST VIRGINIA

BARBOURSVILLE

Cedar Ridge Group Home
55 Bass Avenue
Barboursville, WV 25504

BECKLEY

FMRS Mental Health Council, Inc.
Public Inebriate Shelter
101 South Eisenhower Drive
Beckley, WV 25801

Southern West Virginia Fellowship Home, Inc.
201 Woodlawn Avenue
Beckley, WV 25801

Veterans' Affairs Medical Center Substance Abuse Treatment Program
200 Veterans Avenue
Beckley, WV 25801

BERKELEY SPRINGS

Eastridge Health Systems
404 South Green Street
Berkeley Springs, WV 25411

BUCKHANNON

Appalachian Community Health Center, Inc. Upshur County Office
27 South Kanawha Street
Buckhannon, WV 26201

Saint Josephs Hospital Center Behavioral Health Unit
Amalia Drive
Buckhannon, WV 26201

CHARLESTON

Alliance Behavioral Services, Inc.
3508 Staunton Avenue, Suite 300
Charleston, WV 25304-1477

Behavioral Health Services Charleston Area Medical Center
Brooks and Morris Streets
Charleston, WV 25302

Behavioral Health Services Substance Abuse Services
501 Morris Street
Charleston, WV 25301

Hopemont
State Route 1 Box 223
Charleston, WV 25304

PEERS
600 Broad Street
Charleston, WV 25301

Shawnee Hills, Inc.
DUI Safety Treatment Program
Adult Outpatient Services
600 North Broad Street, 2nd Floor
Charleston, WV 25301

Southway Treatment Center
4605 Maccorkle Avenue SW
Charleston, WV 25309

CHARLES TOWN

Eastridge Health Systems
114 West Liberty Street
Charles Town, WV 25414

CHATTAROY

Logan/Mingo Area Mental Health, Inc. Mingo County Office
Buffalo Creek Road
Chattaroy, WV 25667

CLARKSBURG

United Summit Center Adult Intensive Outpatient Program
6 Hospital Plaza
Clarksburg, WV 26301

Veterans' Affairs Medical Center Substance Abuse Services
Medical Center Drive
Clarksburg, WV 26301

CROSS LANES

Viewpoint
5405 Alpine Drive
Cross Lanes, WV 25313

DANVILLE

Shawnee Hills, Inc.
DUI Safety Treatment Program
Adult Outpatient Services
2 Human Services Complex
Danville, WV 25053

ELKINS

Appalachian Community Health Center
725 Yokum Street
Elkins, WV 26241

Public Inebriate/Detainee Shelter
Gorman and Main Streets
Elkins, WV 26241

FAIRMONT

Fairmont General Hospital Addiction Treatment
1325 Locust Avenue
Fairmont, WV 26554

Valley Comprehensive Community Mental Health Center, Inc.
Alpha Chemical Dependency Treatment Unit
100 Crosswind Drive
Fairmont, WV 26554

Marion County Office
28 Oakwood Road
Fairmont, WV 26554

New Beginnings Program for Women
202 Columbia Street
Fairmont, WV 26554

Crossroads Treatment Program for Adolescents
28 Oakwood Road
Fairmont, WV 26554

FAYETTEVILLE

FMRS Mental Health Council Fayette County Office
209 West Maple Avenue
Fayetteville, WV 25840

GRAFTON

Valley Comprehensive Community Mental Health Center Taylor County Office
501 North Pike Street
Grafton, WV 26354

GYPSY

Rainbow House
158 Main Street
Gypsy, WV 26361

HARRISVILLE

Westbrook Health Services
605 North Street
Harrisville, WV 26362-1205

HUNTINGTON

Area Psychiatric and Psychotherapy Group
1326 6th Avenue
Huntington, WV 25701-2100

Columbia Riverpark Hospital
1230 6th Avenue
Huntington, WV 25701

Innerchange/Prestera River Park Hospital
1230 6th Avenue
Huntington, WV 25701

Laurelwood
432 6th Avenue
Huntington, WV 25701

PARC Way Assessment Center
1530 Norway Avenue
Huntington, WV 25709

PARC West
318 West 14th Street
Huntington, WV 25701

Prestera Center for Mental Health Services, Inc.
1420 Washington Avenue
Huntington, WV 25705

Saint Mary's Hospital Substance Abuse Unit
2900 First Avenue
Huntington, WV 25701

Veterans Affairs Medical Center Outpatient Treatment Program
1540 Spring Valley Drive
Huntington, WV 25704

KINGWOOD

Olympic Center/Preston Adolescent Treatment Program
Route 7
Kingwood, WV 26537

Preston Addiction Treatment Center
300 South Price Street
Kingwood, WV 26537

Valley Comprehensive Community Mental Health Center, Inc. Preston County Office
202 Tunnelton Street Garden Towers
Kingwood, WV 26537

LOGAN

Logan/Mingo Area Mental Health Substance Abuse Services
Route 10
3 Mile Curve
Logan, WV 25601

MARLINGTON

Seneca MH/MR Council, Inc. Pocahontas County Office
704 3rd Avenue
Marlinton, WV 24954

MARTINSBURG

CAT 5/Substance Abuse Services
Route 9 South
Martinsburg, WV 25401

City Hospital, Inc. Gateway Behavioral Health Services
Dry Run Road
Martinsburg, WV 25401

Eastridge Health Systems Eastridge Addiction Treatment Center
125 West Martin Street
Martinsburg, WV 25401

MORGANTOWN

Valley Comprehensive CMHC Main Unit
301 Scott Avenue
Morgantown, WV 26505

MOUNDSVILLE

Northwood Health Systems
10 Ash Avenue
Moundsville, WV 26041

MULLENS

Southern Highlands Community Mental Health Center, Inc.
Wyoming County Office/Mullens Clinic
102 Howard Avenue
Mullens, WV 25882

NEW MARTINSVILLE

Evergreen Behavioral Health
240 North Street
New Martinsville, WV 26155-0247

Northwood Health Systems Wetzel County Office
747 2nd Street
New Martinsville, WV 26155

PARKERSBURG

Saint Josephs Hospital Center for Behavioral Medicine
1824 Murdoch Avenue
Parkersburg, WV 26101

Westbrook Health Services Amity Center
1011 Mission Drive
Parkersburg, WV 26101

Worthington Center, Inc.
3199 Core Road
Parkersburg, WV 26104

PARSONS

Appalachian Community Health Center, Inc. Tucker County Office
601 Walnut Street
Parsons, WV 26287

PETERSBURG

Potomac Highlands Guild, Inc.
1 Virginia Avenue
Petersburg, WV 26847

PHILIPPI

Appalachian Community Health Center, Inc. Barbour County Office
227 Garnett Avenue
Philippi, WV 26416

POINT PLEASANT

Prestera Center for Mental Health Services, Inc. Mason County Office
715 Main Street
Point Pleasant, WV 25550

PRINCETON

Mercer/McDowell/Wyoming Mental Health Council, Inc. Mentoring Program
200 12th Street Extension
Princeton, WV 24740

Southern Highlands Community Mental Health Center, Inc.
200 12 Street Extension
Princeton, WV 24740

RAINELLE

Seneca MH/MR Council, Inc.
645 Kanawha Avenue Suite A
Rainelle, WV 25962

RIPLEY

Westbrook Health Services Jackson County Office
6003 Church Street
Ripley, WV 25271

SOUTH CHARLESTON

Thomas Memorial Hospital Southway Outpatient Program
4825 MacCorkle Avenue SW
Suite B
South Charleston, WV 25309

SPENCER

Westbrook Health Services Roane County Office
227 Clay Road
Spencer, WV 25276

SUMMERSVILLE

Seneca MH/MR Council, Inc.
1 Stevens Road
Summersville, WV 26651

SUTTON

Braxton County Fellowship Home
72 South Stone Wall Street
Suite 2
Sutton, WV 26601

TERRA ALTA

Shawnee Hills, Inc.
Rehabilitation Unit
Substance Abuse Treatment
State Route 1
Terra Alta, WV 26764

UNION

FMRS Mental Health Council Monroe County Office
Monroe County Health Center
Union, WV 24983

VIENNA

Westbrook Health Services
1907 Grand Central Avenue
Lower Level
Vienna, WV 26105

WAYNE

Prestera Center for Mental Health Services, Inc.
145 Kenova Avenue
Wayne, WV 25570

WEBSTER SPRINGS

Seneca MH/MR Council, Inc. Webster County Office
70 Parcoal Road
Webster Springs, WV 26288

WEIRTON

Healthways, Inc.
501 Colliers Way
Weirton, WV 26062

WELCH

Southern Highlands Community Mental Health Center, Inc. McDowell County Office
787 Virginia Avenue
Welch, WV 24801

WHEELING

Northwood Health Systems First Step Program
111 19th Street
Wheeling, WV 26003

New Hope
304 North East Street
Wheeling, WV 26003

WISCONSIN

ALGOMA

Kewaunee County Community Programs Alcohol and Drug Abuse Treatment Program
522 4 Street
Algoma, WI 54201

ALMA

Gundersen Lutheran
Alma, WI 54610

AMERY

Cottonwood Group Homes, Ltd.
773 Rustic Road
Amery, WI 54001

ANTIGO

Langlade Health Care Center
1225 Langlade Road
Antigo, WI 54409

APPLETON

Casa Clare, Inc.
310 North Durkee Street
Appleton, WI 54911

Health Assessment and Counseling Services
1531 South Madison Street
Madison Center, Suite 530
Appleton, WI 54915

Lutheran Social Services
1412 North Rankin Street
Appleton, WI 54913

Meridian House
1308 North Leona Street
Appleton, WI 54913

Saint Elizabeth Hospital
Alcohol/Drug Program (ADP)
1506 South Oneida Street
Appleton, WI 54915

The Mooring Halfway House, Inc.
607 West 7th Street
Appleton, WI 54911

ARCADIA

Franciscan Skemp Healthcare Emergency Room Substance Abuse Services
464 South Saint Joseph Avenue
Arcadia, WI 54612

ASHLAND

Ashland County Information and Referral Center
206 6th Avenue West
Room 213
Ashland, WI 54806

Memorial Medical Center, Inc. Behavioral Health Services
1635 Maple Lane
Ashland, WI 54806

New Horizons North Community Support Services
511 West Main Street Suite 1
Ashland, WI 54806

BARABOO

Saint Clare Hospital Saint Clare Center
707 14th Street
Baraboo, WI 53913

Sauk County Dept of Human Services Alcoholism and Drug Abuse Outpatient Services
505 Broadway Street
Baraboo, WI 53913

BAYFIELD

Red Cliff Tribe AODA Program
Old Dump Road Off Blueberry Road
Bayfield, WI 54814

BEAVER DAM

Psychiatric Associates
200 Front Street
Beaver Dam, WI 53916

BELOIT

Beloit Inner City Council Substance Abuse Services
1435 Wisconsin Avenue
Beloit, WI 53511

Mercy Options Addiction Treatment Services
2825 Prairie Avenue
Beloit, WI 53511

BERLIN

Berlin Memorial Hospital Emergency Detoxification
225 Memorial Drive
Berlin, WI 54923

BLACK RIVER FALLS

Franciscan Skemp Behavioral Health
208 Main Street
Black River Falls, WI 54615-1747

Ho Chunk Nation Dept of Social Services Alcohol/Drug Program Services
1 North 2nd Street
Black River Falls, WI 54615

Kruhn Clinic
610 West Adams Street
Black River Falls, WI 54615

BOSCOBEL

Memorial Hospital of Boscobel Substance Abuse Services
205 Parker Street
Boscobel, WI 53805

BOWLER

Stockbridge/Munsee Health Center Tribal Alcoholism Treatment Program
N8705 Moh-He-Con-Nuck Road
Bowler, WI 54416

BROOKFIELD

Elmbrook Memorial Hospital Alcohol and Drug Treatment Center
19333 West North Avenue
Brookfield, WI 53045

BURLINGTON

Transition House
501 McHenry Street
Burlington, WI 53105

CHILTON

Calumet County Human Service Dept. Alcohol and Other Drug Abuse Unit
206 Court Street
Courthouse
Chilton, WI 53014

CHIPPEWA FALLS

L. E. Phillips Libertas Center for the Chemically Dependent
2661 County Road I
Chippewa Falls, WI 54729

Serenity House, Inc. Transitional Living Program
205 East Grand Avenue
Chippewa Falls, WI 54729

Transitus House
1830 Wheaton Street
Chippewa Falls, WI 54729

CRANDON

Forest County Potawatomi Alcohol and Drug Program
Crandon, WI 54520

Koller Behavioral Health Services
213 East Madison Street
Crandon, WI 54520

CUMBERLAND

Cumberland Memorial Hospital Emergency Detoxification
1110 7 Avenue
Cumberland, WI 54829

Northern Pines Unified Service Center Board Chemical Dependency Service
1066 8 Avenue
Cumberland, WI 54829

DARLINGTON

Lafayette County Department of Human Services/AODA Program
627 Main Street
Darlington, WI 53530

Memorial Hospital of Lafayette County
800 Clay Street
Darlington, WI 53530

DODGEVILLE

Unified Counseling Services Dodgeville Outpatient Clinic
410 North Union Street
Dodgeville, WI 53533

EAGLE RIVER

Eagle River Memorial Hospital Emergency Room Substance Abuse Services
201 Hospital Road
Eagle River, WI 54521

Koller Behavioral Health Services
150 Hospital Road
Eagle River, WI 54521

EAU CLAIRE

Eau Claire Academy
550 North Dewey Street
Eau Claire, WI 54701

Fahrman Center
3136 Craig Road
Eau Claire, WI 54701

First Things First Counseling and Consulting, Ltd.
2125 Heights Drive, Suite 2-D
Eau Claire, WI 54701

Luther Midelfort Behavioral Health
1221 Whipple Street
Eau Claire, WI 54702

Lutheran Social Services
3136 Craig Road
Eau Claire, WI 54701

Sacred Heart Hospital Substance Abuse Services
900 West Clairemont Avenue
Eau Claire, WI 54701

Triniteam, Inc. Treatment Alternative Program
202 Graham Avenue
Eau Claire, WI 54701

ELKHORN

Walworth County Department of Human Services Center
County Highway NN
Elkhorn, WI 53121

ELLSWORTH

Pierce County Dept. of Human Services Alcohol and Other Drug Abuse Services
412 West Kinne Street
Ellsworth, WI 54011

ELROY

Franciscan Skemp Healthcare Community Services Pinecrest Center
1510 Academy Street
Elroy, WI 53929

FOND DU LAC

Beacon House
166 South Park Avenue
Fond Du Lac, WI 54935

Blandine House, Inc.
25 North Park Avenue
Fond du Lac, WI 54935

Robert E. Berry Halfway House
178 6th Street
Fond du Lac, WI 54935

Saint Agnes Hospital Behavioral Health Services
430 East Division Street
Fond du Lac, WI 54935

FORT ATKINSON

Fort Atkinson Memorial Health Services
611 East Sherman Avenue
Fort Atkinson, WI 53538

FRIENDSHIP

Adams County Dept. of Community Programs
108 East North Street
Friendship, WI 53934

GREEN BAY

Brown County Mental Health Center Alcohol and Other Drug Abuse Services
2900 Saint Anthony Drive
Green Bay, WI 54311

Family Service Association Outpatient AODA Program
300 Crooks Street
Green Bay, WI 54301

Jackie Nitschke Center, Inc.
630 Cherry Street
Green Bay, WI 54301

Libertas Treatment Center
1701 Dousman Street
Green Bay, WI 54303

Oneida Tribal Social Services Counseling Services
2640 West Point Road
Green Bay, WI 54304

GREEN LAKE

Green Lake County Human Services Dept. Community Services Unit
500 Lake Steel Street
Green Lake, WI 54941

GRESHAM

Maehnowesekiyah Treatment Program
N 4587 County Highway G
Gresham, WI 54128

HALES CORNERS

Cedar Creek Family Counseling
9415 West Forest Home Avenue, Suite 108
Hales Corners, WI 53130

HAYWARD

Hayward Area Memorial Hospital Substance Abuse Services
Route 3
Hayward, WI 54843

Lac Courte Oreilles Alcohol/Drug and Mental Health Program
Route 2
Hayward, WI 54843

NOO/JII/MOO/WII/MIES Halfway House
Round Lake Township
Hayward, WI 54843

Sawyer County Council on AODA Hill House
County Hill Road
Hayward, WI 54843

Sawyer County Information and Referral Center on Alcohol and Other Drug Abuse
105 East 4th Street
Hayward, WI 54843

HERTEL

Saint Croix Family Resource Center
Hertel, WI 54845

HUDSON

Burkwood Residence
615 Old Mill Road
Hudson, WI 54016

Hudson Medical Center Chemical Health Recovery Center
400 Wisconsin Street
Hudson, WI 54016

HURLEY

Iron County Council on Alcohol and Drug Abuse
408 Silver Street
Hurley, WI 54534

JANESVILLE

Alcohab, Inc.
New Dawn Residential Primary Treatment
430 North Jackson Street
Janesville, WI 53545

River Commons
786 South Main Street
Janesville, WI 53545

Associates in Psychotherapy Affected Family Member Program
1519 Primrose Lane
Janesville, WI 53545

Crossroads Counseling Center
301 East Milwaukee Street
Janesville, WI 53545

Lutheran Social Services/Rock County Alcohol and Drug Treatment Unit
205 North Main Street
Suite 102
Janesville, WI 53545

Mercy Options Addiction Treatment Services
1000 Mineral Point
Janesville, WI 53545

Rock County Psychiatric Hospital Substance Abuse Services
3530 North County Trunk Street
Suite F
Janesville, WI 53545

Rock Valley Community Program, Inc. Treatment Alternative Program
203 West Sunny Lane Road
Janesville, WI 53546

JUNEAU

Dodge County Dept. of Human Services Chemical Dependency Services
199 Home Road
Juneau, WI 53039

KENOSHA

Addiction Consulting Associates
611 56th Street
Kenosha, WI 53140

Alcohol and Drug Consultants
7543 17 Avenue
Kenosha, WI 53143

Covenant Behavioral Health
6021 56th Avenue, Suite 6
Kenosha, WI 53144

Gateway House Group Home
460 56th Avenue
Kenosha, WI 53144

Interventions
6755 14th Avenue
Kenosha, WI 53140

Oakwood Clinical Associates, Ltd.
4109 67th Street
Kenosha, WI 53140

Professional Services Group Inc
6233 39th Avenue
Kenosha, WI 53142

Saint Catherine's Hospital Behavioral Services
3556 7th Avenue
Kenosha, WI 53140

3734 7th Avenue Dominican Building, Suite 3
Kenosha, WI 53140

KESHENA

Menominee County Human Services Dept. Alcohol and Other Drug Abuse Program
Highway 55 and 47
Keshena, WI 54135

KEWASKUM

Exodus Transitional Care Facility, Inc.
1421 Fond Du Lac Avenue
Kewaskum, WI 53040

LAC DU FLAMBEAU

Family Resource Center Chippewa Health Center
450 Old Abe Road
Lac du Flambeau, WI 54538

LA CROSSE

Coulee Youth Center, Inc.
231 Copeland Avenue
La Crosse, WI 54602

La Crosse County Human Service Dept. Clinical Services Section
300 North 4th Street
La Crosse, WI 54601

Farrell and Wissing Alternatives
505 King Street, Suite 38
La Crosse, WI 54601

Franciscan Skemp Behavioral Healthcare
212 South 11th Street
La Crosse, WI 54601

Intensive Residential Adult Chemical Dependency Program
620 South 11th Street
La Crosse, WI 54601

FSH Behavioral Health Residential Services
LAAR House
1022 Division Street
La Crosse, WI 54601

Scarseth House
535 South 17th Street
La Crosse, WI 54601

Gundersen Lutheran
1312 5th Avenue
La Crosse, WI 54601

Unity for Women
1922 Miller Street
La Crosse, WI 54601

Hoffe/Cassel Counseling Services LLC
La Crosse, WI 54601

LADYSMITH

Rusk County Memorial Hospital Substance Abuse Services
900 College Avenue West
Ladysmith, WI 54848

LANCASTER

Unified Counseling Services
210 South Washington Street
Lancaster, WI 53813

MADISON

ARC Community Services, Inc.
Arc House
202 North Paterson Street
Madison, WI 53703

ARC Center for Women and Children
1409 Emil Street
Madison, WI 53713

Capitol Square Associates
660 West Washington Avenue, Suite 305
Madison, WI 53703

Hope Haven, Inc.
Colvin Manor
425 West Johnson Street
Madison, WI 53703

North Bay Lodge
3602 Memorial Drive
Madison, WI 53704

Lutheran Social Services
5 Odana Court
Madison, WI 53713

Mendota Mental Health Institute Substance Abuse Services
301 Troy Drive
Madison, WI 53704

Mental Health Center of Dane County Alcohol and Drug Treatment Unit
625 West Washington Avenue
Madison, WI 53703

Meriter Hospital/New Start
New Start East
1310 Mendota Street
Suite 110
Madison, WI 53714

New Start West
1015 Gammon Lane
Madison, WI 53719

Washington Avenue Unit
309 West Washington Avenue
Madison, WI 53703

Schwert AODA Treatment Center
3501 Kipling Drive
Madison, WI 53704

Tellurian UCAN, Inc.
Adult Residential Program (ARP)
300 Femrite Drive
Madison, WI 53716

Day Treatment Program
1250 Fermrite Drive
Madison, WI 53716

Detoxification Unit
2914 Industrial Drive
Madison, WI 53713

Thoreau House
1102 Spaight Street
Madison, WI 53703

MANITOWOC

Holy Family Memorial Medical Center
2300 Western Avenue
Manitowoc, WI 54220

Manitowoc County Human Services Dept. Counseling Center
927 South 8th Street
Manitowoc, WI 54220

Marco
1114 South 11th Street
Manitowoc, WI 54220

MARINETTE

Bay Area Medical Center
3100 Shore Drive
Marinette, WI 54143

Marinette County Human Services Adapt
2400 Hall Avenue
Marinette, WI 54143

MAUSTON

Juneau County Human Service Center
220 East La Crosse Street
Mauston, WI 53948

MEDFORD

Taylor County Human Services Department
540 East College Street
Medford, WI 54451

MENASHA

Family Service Association of Fox Valley
1488 Kenwood Center
Menasha, WI 54952

Theda Clark Center for Recovery
324 Nicolet Boulevard
Menasha, WI 54952

MENOMONIE

Arbor Place Alcohol and Other Drug Abuse Program
320 21st Street North
Menomonie, WI 54751

Saint Mary's Behavioral Medicine
13111 North Port Washington Road
Mequon, WI 53097

MERRILL

Lincoln Health Care Center Merrill Office
503 South Center Avenue
Merrill, WI 54452

Sacred Heart Outpatient Clinic Oasis Recovery Program
807 East 1st Street
Merrill, WI 54452

MILWAUKEE

American Indian Council on Alcoholism
2240 West National Avenue
Milwaukee, WI 53204

Aro Counseling Center, Inc.
4325 South 60th Street, Suite 3
Milwaukee, WI 53220-3508

Cedar Creek Counseling Center
6815 West Capitol Drive, Suite 301
Milwaukee, WI 53216-2070

Children and Family Service, Inc.
4365 North 27th Avenue
Milwaukee, WI 53218

Council for the Spanish Speaking Salud de la Familia
614 West National Street
Milwaukee, WI 53204

Genesis Behavioral Services, Inc.
Milwaukee Outpatient Clinics
2040 West Wisconsin Avenue, Suite 560
Milwaukee, WI 53201

Genesis Detoxification Center
1218 West Highland Boulevard
Milwaukee, WI 53233

Holton Youth Center AODA YMCA
510 East Burleigh Street
Milwaukee, WI 53202

Horizon House
2511 West Vine Street
Milwaukee, WI 53205

Ivanhoe Treatment, Inc.
2203 East Ivanhoe Place
Milwaukee, WI 53202

Lutheran Social Services of Wisconsin and Upper Michigan, Inc.
6101 West Vliet Street, Suite 100
Milwaukee, WI 53213

10401 West Lincoln Avenue
Suite 209
Milwaukee, WI 53227

Matt Talbot Recovery Center
2613 West North Avenue
Milwaukee, WI 53205

Milwaukee Health Services System
4383 North 27 Street
Milwaukee, WI 53216

2778 South 35th Street
Milwaukee, WI 53215

Milwaukee Women's Center Behavioral Health Clinic
611 North Broadway
Suite 230
Milwaukee, WI 53202

Multi-Cultural Counseling Services DBA Renew Counseling Services
1225 West Mitchell Street
Suite 223
Milwaukee, WI 53204

2014 West North Avenue
Milwaukee, WI 53233

Northwest General Hospital Substance Abuse Services
5310 West Capitol Drive
Milwaukee, WI 53216

Pathways Counseling Center
2645 North Mayfair Road
Suite 230
Milwaukee, WI 53226

Reach, Inc. Comprehensive Mental Health Clinic Substance Abuse
6001 West Center Street
Suite 9711
Milwaukee, WI 53210

Relapse Prevention Service
8112 West Bluemound Road
Suite 106
Milwaukee, WI 53213

Riverwest North Meta House
2626 North Bremen Street
Milwaukee, WI 53212

SAFE Group Services, Inc.
3500 North Sherman Boulevard
Suite 302
Milwaukee, WI 53216

Saint Michael's Hospital Mental Health Center
2400 West Villard Avenue
Milwaukee, WI 53209

Sinai Samaritan Medical Center Substance Abuse Services
2000 West Kilbourn Avenue
Milwaukee, WI 53233

United Community Center New Beginning Clinic
1028 South 9 Street
Milwaukee, WI 53204

Wisconsin Cipe, Inc.
1915 West Hampton Avenue
Milwaukee, WI 53209

Wisconsin Correctional Service (WCS)
152 West Wisconsin Avenue
Milwaukee, WI 53203

Wisconsin Midwest Clinical Services
2200 North Mayfair Road
Milwaukee, WI 53226

Zablocki VA Medical Center
5000 West National Avenue
Milwaukee, WI 53295

MINOCQUA

Koller Behavioral Health Services
415 Menominee Street
Minocqua, WI 54548

MONROE

Green County Human Services Alcohol/Other Drug Abuse Services
N3152 State Highway 81
Monroe, WI 53566

MONTELLO

Marquette Chemical Dependency Service
Highway 22 South
Montello, WI 53949

MUKWONAGO

Norris Adolescent Center
Center Drive
Route 5 W247 S10395
Mukwonago, WI 53149

NEILLSVILLE

Clark County Community Services Alcohol and Other Drug Abuse Program
517 Court Street
Neillsville, WI 54456

NEW LONDON

New London Family Medical Center Emergency Room Substance Abuse Services
1405 Mill Street
New London, WI 54961

NEW RICHMOND

Saint Croix County Health and Human Services
1445 North 4th Street
New Richmond, WI 54017

NIAGARA

Adapt Human Services
1201 Jackson Street
Niagara, WI 54151

OCONTO

Oconto County Dept. of Human Services Clinical Services Division/Substance Abuse Unit
501 Park Avenue
Oconto, WI 54153

ODANAH

Bad River Alcohol/Drug Program
Bad River Community Center
Odanah, WI 54861

ONEIDA

Oneida Group Homes Kuthani Yosta
453 Country Court
Oneida, WI 54155

OSHKOSH

Mercy Medical Center Counseling Service
515 Washburn Boulevard
Oshkosh, WI 54901

Nexus House Lutheran Social Services
2002 Algoma Boulevard
Oshkosh, WI 54901

Summit House
2501 Harrison Street
Oshkosh, WI 54901

Nova Treatment Center
Horizon House
111 Josslyn Street
Oshkosh, WI 54901

Terra House
105 Josslyn Street
Oshkosh, WI 54901

United Behavioral Health Services
1750 West Pointe Drive
Oshkosh, WI 54901

PHILLIPS

Counseling and Personal Development AODA Treatment Program
171 Chestnut Street
Phillips, WI 54555

PLATTEVILLE

Unified Counseling Services Platteville Outpatient Clinic
6057 South Chestnut Street
Platteville, WI 53818

PLYMOUTH

Sheboygan Counseling and Development Center
710 Eastern Avenue
Plymouth, WI 53073

PORTAGE

Divine Savior Hospital Emergency Room Substance Abuse Detox Services
1015 West Pleasant Street
Portage, WI 53901

Pauquette Center for Psychological Services
304 West Cook Street
Portage, WI 53901

PORT EDWARDS

Entrance/Exit Program
1351 Wisconsin River Drive
Port Edwards, WI 54469

PORT WASHINGTON

Ozaukee County Dept. of Community Programs Ozaukee County Counseling Center
121 West Main Street
Port Washington, WI 53074

POYNETTE

Poynette Counseling and Psychotherapy Associates, Inc.
415 North Main Street
Poynette, WI 53955

PRAIRIE DU CHIEN

FSH Behavioral Health Residential Services Villa Succes
121 South Prairie Street
Prairie du Chien, WI 53831

PRAIRIE DU SAC

Pathway Clinic
50 Prairie Avenue
Prairie Du Sac, WI 53578

RACINE

All Saints Behavioral Health Services
1320 Wisconsin Avenue
Racine, WI 53403

Charter Counseling Center
6021 Durand Avenue
Racine, WI 53406

Covenant Behavioral Health
1055 Prairie Drive
Racine, WI 53406

Crisis Center of Racine, Inc.
1925 Washington Street
Racine, WI 53403

Genesis Behavioral Services
5200 Washington Avenue
Suite 105
Racine, WI 53406

Durand Home
4606 Durand Avenue
Racine, WI 53406

Saint Clair House
4107-4109 Saint Clair Street
Racine, WI 53402

Spring Place Manor Residential Facility
1725-27 Spring Place
Racine, WI 53401

Racine Psychological Services
840 Lake Avenue
Racine, WI 53403

RHINELANDER

Koinonia Residential Treatment Center
1991 Winnebago Drive
Rhinelander, WI 54501

Koller Behavioral Health Services
622 Mason Street
Rhinelander, WI 54501

RICE LAKE

Parkview Center
1107 East Orchard Beach Lane
Rice Lake, WI 54868

RICHLAND CENTER

Richland County Community Programs
1000 Highway 14 West
Richland Center, WI 53581

RIVER FALLS

Kinnic Falls Alcohol and Drug Abuse Services
900 South Orange Street
River Falls, WI 54022

SAINT CROIX FALLS

Saint Croix Valley Memorial Hospital Chemical Dependency Center
204 South Adams Street
Saint Croix Falls, WI 54024

SHAWANO

Shawano County Department of Community Programs
504 Lakeland Road
Shawano, WI 54166

Shawano Medical Center
309 North Bartlette Street
Shawano, WI 54166

Shawano County Community Programs Professional Services Center
125 North Main Street
Shawano, WI 54166

SHEBOYGAN

Counseling and Development Center
2205 Erie Avenue
Sheboygan, WI 53081

Kettle Moraine/Genesis
503 Wisconsin Avenue
Sheboygan, WI 53081

Rebos Manor
908 Jefferson Street
Sheboygan, WI 53081

Sheboygan County Human Services Outpatient Services
1011 North 8th Street
Sheboygan, WI 53081

Sheboygan Memorial Medical Center Chemical Dependency Services
2629 North 7th Street
Sheboygan, WI 53083

SHELL LAKE

Indianhead Residential Care Facility, Inc.
122 5th Avenue West
Shell Lake, WI 54871

SPARTA

Monroe County Department Human Service
14301 County Highway B, Box 19
Sparta, WI 54656-4509

SPOONER

Spooner Community Memorial Hospital Substance Abuse Detox
819 Ash Street
Spooner, WI 54801

STEVENS POINT

Community Alcohol and Drug Abuse Center
209 Prentice Street North
Stevens Point, WI 54481

Oakside Residential Living Facility
201 North Prentice Street
Stevens Point, WI 54481

Saint Michael's Hospital Emergency Room Substance Abuse Services
900 Illinois Avenue
Stevens Point, WI 54481

STOUGHTON

Lutheran Social Services Home Programs/Serenity Unit
209 North Division Street
Stoughton, WI 53589

STURGEON BAY

Door County Department of Community Programs
421 Nebraska Street
Sturgeon Bay, WI 54235

SUPERIOR

Recovery Center, Inc.
2231 Catlin Avenue
Suite 2 East
Superior, WI 54880

TOMAH

Gundersen Lutheran
321 Butts Avenue
Tomah, WI 54660

Veterans' Affairs Medical Center Alcohol/Drug Dependence Treatment Program
500 East Veterans Street
Tomah, WI 54660

TOMAHAWK

Lincoln Health Care Center Tomahawk Office/Substance Abuse Services
310 West Wisconsin Avenue
Tomahawk, WI 54487

Oasis Recovery Program
216 North 7th Street
Tomahawk, WI 54487

VIROQUA

Pierzina Counseling Services
210 Airport Road, Suite 103-B
Viroqua, WI 54665-1160

Vernon Memorial Hospital Emergency Detoxification
507 South Main Street
Viroqua, WI 54665

WASHBURN

Lutheran Social Services
320 Superior Avenue
Washburn, WI 54891

WATERTOWN

Directions Counseling Center
129 Hospital Drive
Watertown, WI 53094

WAUKESHA

Aro Counseling Center Incorporated
400 West Moreland Boulevard
Waukesha, WI 53188

Century House
1130 Northview Road
Waukesha, WI 53188

Genesis House
1002 Motor Avenue
Waukesha, WI 53188

La Casa de Esperanza AODA Prevention/Education
410 Arcadian Avenue
Waukesha, WI 53186

Lutheran Social Services
325 Sentinel Drive
Waukesha, WI 53186

Southeastern Youth and Family Services NOAH House
West 222 South 3210 Racine Avenue
Waukesha, WI 53186

WAUPACA

Waupaca County Dept. of Human Services Outpatient Treatment Services
811 Harding Street
Waupaca, WI 54981

WAUSAU

Center for Well Being, Inc.
2801 North Seventh Street
Suite 400
Wausau, WI 54401

North Central Health Care Facilities
1100 Lake View Drive
Wausau, WI 54401

WAUTOMA

Alcoholism and Drug Abuse Services of Waushara County
310 South Scott Street
Wautoma, WI 54982

WAUWATOSA

Associated Women Psychotherapist
10625 West North Avenue, Suite 208
Wauwatosa, WI 53226

Milwaukee Psychiatric Hospital Chemical Dependency Services
1220 Dewey Avenue
Wauwatosa, WI 53213

WEST ALLIS

Charter Counseling Center
2323 South 109th Street
Suite 175
West Allis, WI 53227

Genesis Behavioral Services, Inc.
1126 South 70th Street
West Allis, WI 53214

WINNEBAGO

Anchorage
Winnebago, WI 54985

Winnebago Mental Health Institute Gemini
Main Butler Avenue
Winnebago, WI 54985

WISCONSIN RAPIDS

Riverview Hospital Emergency Inpatient Detox
410 Dewey Street
Wisconsin Rapids, WI 54494

Wood County Unified Services
2611 South 12th Street
Wisconsin Rapids, WI 54494

WYOMING

BASIN

Big Horn County Counseling
220 South 4 Street
Basin, WY 82410

BUFFALO

Northern Wyoming Mental Health Center Substance Abuse Services
521 West Lott Street
Buffalo, WY 82834

CASPER

Casper Psychological Services
136 South Washington Street
Casper, WY 82601

Sunrise Recovery Center Wyoming Medical Center
255 South Jackson Street
Casper, WY 82601

The Prairie Institute, Inc.
309 North McKinley Street
Casper, WY 82601

Wyoming Behavioral Institute
2521 East 15th Street
Casper, WY 82609

CHEYENNE

Behavioral Health Services
United Medical Center East Building
2600 East 18th Street
Cheyenne, WY 82001

Cheyenne Community Drug Abuse Treatment Council, Inc. Pathfinder
121 west Carlson Street
Cheyenne, WY 82001

Southeast Wyoming Mental Health Center
Chemical Health Services
2526 Seymour Avenue
Cheyenne, WY 82003

Cheyenne Alcohol Receiving Center
Halfway House
Transitions Residential Program
2310 East 8th Street
Cheyenne, WY 82001

Veterans Affairs Medical Center Substance Abuse Treatment Program
2360 East Pershing Boulevard
Cheyenne, WY 82001

CODY

Cedar Mountain Center at West Park Hospital
707 Sheridan Avenue
Cody, WY 82414

DOUGLAS

Eastern Wyoming Mental Health Center Substance Abuse Services
1841 Madora Avenue
Douglas, WY 82633

EVANSTON

Cornerstone
195 Featherway Street, Suite 1
Evanston, WY 82930

Wyoming State Hospital
831 Highway 150
Evanston, WY 82930

GILLETTE

Powder River Chemical Dependency, Inc.
400 South Kendrick Avenue
Suite 101
Gillette, WY 82716

Wyoming Regional Counseling Center
900 West 6th Street
Gillette, WY 82716

GLENROCK

Eastern Wyoming Mental Health Center
925 West Birch Street
Glenrock, WY 82637

JACKSON

Curran/Seeley Foundation
610 West Broadway
Suite L-1
Jackson, WY 83001

LARAMIE

Ivinson Memorial Hospital
255 North 30th Street
Laramie, WY 82072

Southeast Wyoming Mental Health Center Substance Abuse Services
710 Garfield Street
Suite 320
Laramie, WY 82070

Wyoming Counseling and Outreach Services
901 South 3rd Street
Laramie, WY 82070

LOVELL

Big Horn County Counseling
441 Montana Avenue
Lovell, WY 82431

LUSK

Eastern Wyoming Mental Health Center Substance Abuse Services
905 South Main Street
Lusk, WY 82225

NEWCASTLE

Northern Wyoming Mental Health Center Substance Abuse Services
420 Deanne Avenue
Newcastle, WY 82701

PINEDALE

Sublette Community Counseling Services
41 1/2 South Franklin Street
Pinedale, WY 82941

RAWLINS

Carbon County Counseling Center
721 West Maple Street
Rawlins, WY 82301

RIVERTON

Fremont Counseling Service
511 North 12th West
Riverton, WY 82501

ROCK SPRINGS

Southwest Counseling Service
1414 North 12th Street East
Rock Springs, WY 82901

SHERIDAN

Northern Wyoming Mental Health Center Substance Abuse Services
1221 West 5 Street
Sheridan, WY 82801

Piedmont Psychological Practice
425 West Loucks Street
Sheridan, WY 82801

Sheridan House, Inc.
1003 Saberton Street
Sheridan, WY 82801

Veterans' Affairs Medical Center Substance Abuse Treatment Program
1898 Fort Road
Sheridan, WY 82801

THERMOPOLIS

Hot Springs County Counseling Service
121 South 4th Street
Thermopolis, WY 82443

TORRINGTON

Southeast Wyoming Mental Health Center Substance Abuse Services
1942 East D Street
Torrington, WY 82240

WHEATLAND

Southeast Wyoming Mental Health Center Substance Abuse Services
103 Park Avenue
Wheatland, WY 82201

WORLAND

Washakie County Mental Health Services
509 Big Horn Avenue
Worland, WY 82401

U.S Territories and Affiliated States

FEDERATED STATES OF MICRONESIA

POHNPEI

Department of Health Services Community Mental Health Center
Pohnpei, FM 96941

YAP

Yap Memorial Hospital Department of Health Services
Yap, FM 96943

GUAM

TAMUNING

Department of Mental Health and Substance Abuse
Substance Abuse Drug and Alcohol Prevention Program
790 Governor Carlos G. Camacho Road
Tamuning, GU 96911

PUERTO RICO

AGUADILLA

Centro de Salud Mental (SITA)
First Floor Hospital Regional
Aguadilla, PR 00605

Hogar Crea Aguadilla
Carretera 2 Interior 110 Km 1180
Barrio Ceiba Baja
Aguadilla, PR 00605

Teen Challenge de Aguadilla
Carretera 107 KM 3.5
Sector Playuela Barrio Borinquen
Aguadilla, PR 00603

AIBONITO

Hogar Crea Aibonito Adolescentes
Calle Alfredo Marrero 68
Aibonito, PR 00705

ANASCO

Hogar Crea Anasco Varones
Carretera 109 Kilometro 42 Barrio Espino
Anasco, PR 00610

Hogar Jesus Inc
Street 406 Kilometro 22 Barrio Casey
Anasco, PR 00610

ARECIBO

Centro de Tratamiento de Menores Libre de Drogas de Arecibo
Carretera 129 Arecibo Alares Antiguo Hospital de Distrito
Arecibo, PR 00612

Centro de Tratamiento Para Adultos de Arecibo
Antiguo Hospital de Distrito
Arecibo, PR 00612

Hogar Crea Arecibo Adolescentes
Carretera 682 Kilometro 59
Barrio Garrochales
Arecibo, PR 00612

Hogar Crea Arecibo Adultos
Carretera 129 Kilometro 412
Barrio Hato Arriba
Arecibo, PR 00612

Mision Rescate Drug Abuse Treatment Rehabilitation Center
Carretera 651 Km 27
Int Hato Arriba Sector Combate
Arecibo, PR 00612

BARRANQUITAS

Hogar Crea Barranquitas
Calle Principal 14
Barranquitas, PR 00794

BAYAMON

Hogar Crea Vista Alegre
Calle La Liga Esquina C
Barriada Vista Alegre
Bayamon, PR 00959

Hogar Crea Bayamon Adolescentes
Calle la Liga Esquina Barriada Vista Alegre
Bayamon, PR 00959

Hogar Crea Districto de Bayamon
Carretera 852 Kilometro 02
Bayamon, PR 00961

Ministerios Jehova/Justicia Nuestra
Calle A-CC-16 Bayamon Gardens
Bayamon, PR 00957

New Life for Girls de Puerto Rico
Carretera 830 Km 57 Barrio Santa Olaya Sector Los Llanos
Bayamon, PR 00956

Renovados en Cristo
Carr 812 KM 6.4 Camino Los Ponos
Bo Guaraguao Sector La Pena
Bayamon, PR 00956

Teen Challenge of Puerto Rico Inc
Carretera 2 Kilometro 77 Barrio Juan Domingo
Bayamon, PR 00957

CAGUAS

Hogar Crea Cabo Rojo
Carr 311 Kilometro 31
Interior Camino Los Ascencios
Cabo Rojo, PR 00623

Adm Servicios Salud Mental y Contra Adicion Centro deTratamiento Menores
Centro Tratamiento a Menores
Apartado 9150
Caguas, PR 00726

Caguas Substance Abuse Treatment Center
Calle Gautier Benitez 162
Edificio Angora San Alfonso Plaza
Caguas, PR 00725

Hogar Crea De Caguas
Calle Padia Final Barrio
Bairoa La 25 Carretera 796
Caguas, PR 00725

Hogar Resurreccion
Carretera 175 KM 3 HM O
Bo San Antonio
Caguas, PR 00725

Hogar Crea Canovanas
Carretera 188 Kilometro 13
Barrio San Isidro
Canovanas, PR 00729

Hogar Crea La Central
Barrio Torrecilla Alta
Canovanas, PR 00729

CAROLINA

Hogar Crea Carolina
Carretera 887 Kilometro 14
Barrio Martin Gonzalez
Carolina, PR 00987

Hogar El Buen Samaritano, Inc.
Unit 2
Carr 857 KM 9.5
Barrio Carruzo Sector Filipinas
Carolina, PR 00628

CAYEY

Hogar Crea Cayey
Avenida Antonio R. Barzelo
al lado del Cuartel de la Policia
Cayey, PR 00737

CIDRA

First Hospital Panamerica
Carretera 787 Kilometro 15
Cidra, PR 00739

COMERIO

Hogar Crea Comerio Adolescente Barrio Palomas Abajo
Sector El 26
Carretera 156 Kilometro 33 Hm 8
Comerio, PR 00782s

COROZAL

Hogar Crea Corozal
Hectometro 02 Barrio Dos Bocas
Carretera 159 Km 124
Corozal, PR 00783

DORADO

Hogar Crea Dorado
Calle A Bloque C-48
Costa de Oro
Dorado, PR 00646

EL SENORIAL/RIO PIEDRAS

Puerto Rico Addiction Medical Services
6 Street South 7-2
Villas de Parana
El Senorial/Rio Piedras, PR 00960

FAJARDO

Hogar Crea Arturo Nieves
Calle 3 Barrio Jerusalem
Fajardo, PR 00738

GUANICA

Hogar Crea Guanica
Carretera Ochoa Km 19 Bda Esperanza
Finca 5 Hermanos
Guanica, PR 00653

GUAYAMA

Hogar Crea Guayama
Barrio Linea
Capo 13 Carretera 15
Guayama, PR 00785

Hogar Nuevo Camino
Sector Villodas
Carretera 713 Kilometro 0.3
Guayama, PR 00784

GUAYNABO

Centro Renancer, Inc.
Carretera 834 Km 42
Barrio Sonadora Sector Las Parcelas
Guaynabo, PR 00970

Hogar Crea Guaynabo Adolescentes
Calle Vanda Numero 1
Urbanizacion Torrimar
Guaynabo, PR 00966

Hogar Crea Guaynabo Adultos
Calle Union 3 Sector Montalvo
Camino Alejandrino Kilometro 05
Guaynabo, PR 00965

Hogar Crea Sabana
Calle Maritima 410
Barrio Sabana
Guaynabo, PR 00965

Hogar de Ayuda El Refugio, Inc.
Avenida Ponce de Leon
Esquina Santa Rosa De Lima 17A
Guaynabo, PR 00965

GURABO

Hogar El Buen Samaritano, Inc.
Carretera 941 KM 5 HM 0
Barrio Jaguas
Gurabo, PR 00778

Hogar Intermedio De Dama En Gurabo
Calle Santiago Final
Carretera 943 Km2
Gurabo, PR 00778

Hogar Nueva Vida
Carretera 181 Ramal 944
Bo Celada
Gurabo, PR 00778

Hogar Nueva Vida Oseli
Barrio Calabasas, Carretera 182
Gurabo, PR 00778

HATO REY

Hogar Crea Jovenes Y Adultos Quisqueya Proyecto Especial
Calle Quisqueya 207
Hato Rey, PR 00917

HUMACAO

Hogar Crea Humacao
Carretera 908 Kilometro 2 Hm 7
Barrio Tejas
Humacao, PR 00791

Proyecto Hombre
Calle Antonio Lopez 116
Humacao, PR 00792

ISABELA

Hogar Crea Isabela Adolescentes
Carretera 472 Kilometro 32
Barrio Bejucos
Isabela, PR 00662

Hogar Crea Juana Diaz Adultos
Carretera 14 Kilometro 169
Sector Tijera
Juana Diaz, PR 00795

Proyecto Especial para Adolecentes De Juana Diaz
Barrio Caoitanejo
Kilometro 115-2 Carrtera 1
Juana Diaz, PR 00795

JUNCOS

Hogar Crea Juncos
Carretera 185 Kilometro 20 Hectometro 0
Barrio Las Pinas
Juncos, PR 00777

Hogar Nuevo Pacto
Carretera 31 KM 19
Bo Caimito 1
Juncos, PR 00777

LAS MARIAS

Hogar Crea Las Marias
Carretera 119 Kilometro 261
Barrio Maravilla Norte
Las Marias, PR 00670

LOIZA

Hogar Crea Loiza
Calle San Patricio Final 16
Loiza, PR 00772

LUQUILLO

Hogar Crea Luquillo
Calle 14 Barrio Hato Viejo Fortuna
Luquillo, PR 00773

MANATI

Centro Tratamiento Ambulatorio Centro Tratamiento Adultos
Obrero 15-A Esquina Quinones
Box 583
Manati, PR 00674-0583

Hogar Crea Manati Adultos
Carretera 2 Kilometro 48
Barrio Cotto Norte
Manati, PR 00674

Hogar Crea Manati Damas
Carretera 616 Kilometro 2
Barrio Tierras Nuevas Sector Cantitos
Manati, PR 00674

MAYAGUEZ

Centro SITA Tratamiento A Sustancias (Drogas)
Avenida Hostos 11
Mayaguez Medical Center
Mayaguez, PR 00680

Centro Tratamiento A Menores Mayaguez
Avenida Eugenio Maria de Hostos
Carreterra 2 - Hospital Betances
Mayaguez, PR 00681

Hogar Crea Modulo Crea/Centro Detencion Oeste
Carretera 105 Kilometro 18
Mayaguez, PR 00680

Hogar Crea/Posada Fe Y Esperanza
Calle Comercio 242
Mayaguez, PR 00680

Mision Rescate, Inc. Drug Abuse Treatment
Road 104 Kilometro 1.7
Barrio Algarrobo
Mayaguez, PR 00680

MOROVIS

Hogar Crea Morovis Adolescentes
Carretera 159 Kilometro 16
Barrio Montellano Sector La Fabrica
Morovis, PR 00687

NAGUABO

Hogar Crea Naguabo
Carretera 3 Kilometro 63 H4
Barrio Daguao
Naguabo, PR 00718

NARANJITO

Hogar Crea Naranjito
Carretera 164 Kilometro 05
Barrio Nuevo
Naranjito, PR 00719

OROCOVIS

Hogar Crea Orocovis
Barrio Sabana Sector La Pista
Orocovis, PR 00720

PONCE

Centro de Tratamiento Para Adultos de Ponce
Carretera Num 14 Barrio Machuelo
Facilidades de Centro Medico
Ponce, PR 00731

Desintoxicacion Para Menores Cede Ponce
Carretera 14 Centro Medico
Barrio Machuelo
Ponce, PR 00731

Hogar Crea Distrito de Ponce
Calle Central 13
Barrio Machuelo
Ponce, PR 00731

Hogar Crea Ponce Adolescente
Calle 1 Numero 4
Urbanizacion Villa Flores
Ponce, PR 00733

Hogar Crea Ponce Mercedita
Carretera 1 Kilometro 119 Hm .9
Barrio Buyones
Ponce, PR 00731

Hogar Crea Ponce Playa Posada Fe Y Esperanza
Avenida Los Meros 45
Playa Ponce
Ponce, PR 00733

Institucion Regional del Sur Jovenes Adultos Tratamiento Sicosocial
Bo El Tuque Sector Las Cucharas
Ponce, PR 00731

Mision Refugio Incorporado
Bo Maraquez KM 4 HM 2
Ponce, PR 00731

Ponce Alcoholism Treatment Program
Ponce Medical Center
Ponce, PR 00731

Programa Ayuda y Consejeria Empleado PACE ASSMCA
Centro Medico Carreterra 14
Barrio Machuelo
Ponce, PR 00731

Hogar Crea Quebradillas Adultos
Carretera 478 Kilometro .5
Barrio San Antonio
Quebradillas, PR 00678

Hogar Crea Quebradillas Ninos
Carretera 113 Kilometro 141 Interior
Barrio San Antonio
Quebradillas, PR 00678

RIO PIEDRAS

Hogar Crea Rio Grande Damas
Carretera 956 Kilometro 04
Barrio Guzman
Rio Grande, PR 00745

Centro Quimioterapia San Juan Barrio Monacillos/Facilidades Centro
Rio Piedras, PR 00928

Emergency Alcoholism Detox Unit
Casa De Salud Medical Center
Rio Piedras, PR 00935

Puerto Rico Addiction Medical Services
Carretera 21
Rio Piedras, PR 00928

Hogar Crea Sabana Grande
Carretera 368 Kilometro 38
Barrio Machuchal
Sabana Grande, PR 00637

Mission Rescate Drug Abuse Treatment and Rehabilitation
Carretera 328 Kilometro 57
Intersection Barrio Rayo Guara
Sabana Grande, PR 00637

Hogar Crea San German
Carretera 318 Kilometro 08
Barrio Maresua
San German, PR 00683

SAN JUAN

ASEM
Pabellon J. Terrenos Centro Medico
Barrio Monacillos
San Juan, PR 00925-2129

ASSMCA Centro Tratmiento Drogas y Alcohol
Pabellon G Centro Medico
San Juan, PR 00918

Bayamon Quimioterapia
414 Avenida Barbosa
San Juan, PR 00928

Casa La Providencia
Calle Norzagaray Street 200
Old San Juan
San Juan, PR 00902

Cede San Juan
Pabellon B Calle Maga
Barrio Monacillos
San Juan, PR 00925

Centro de Rehabilitacion Dr Fumero
Fernadez Juncos Station
San Juan, PR 00910

Hogar Crea Districto De San Juan I
Avenida Ponce De Leon
1955 Parada 26 1/2
San Juan, PR 00915

Hogar Crea Centro Madres Con Ninos Hogar Crea San Jose
Calle Urdiales Esquina Burgos Embalse
San Jose
San Juan, PR 00928

Hogar Crea Ciudad Modelo Damas
Calle Hoare No. 716 Parada 15
San Juan, PR 00907

Hogar Crea Country Club
Calle Lola Rodriguez de Tio 794
2da Extencion Country Club RP
San Juan, PR 00928

Hogar Crea Las Americas
Calle Teniente Cesar Gonzalez
1105 Villa Nevarez
San Juan, PR 00927

Hogar Crea Parcelas Falu Proyecto Especial
Calle 36 Final Parcelas Falu
San Juan, PR 00928

Hogar Crea Park Gardens
Calle Tortosa Final P-15
Villa Andalucia
San Juan, PR 00926

Hogar Crea Puerta de Tierra
Paseo Covadonga Numero 110
Puerta de Tierra
San Juan, PR 00907

Hogar Crea Sabana Llana
Calle Lealtad Esquina Libertad 1012
Urbanizacion Victoria
San Juan, PR 00924

Hogar Crea San Jose
Calle Urdiales Esquina Burgos
Embalse San Jose
San Juan, PR 00928

Hogar Crea Taft
Calle Leon Acuna 1702
San Juan, PR 00911

Hogar Crea Tortugo
Carretera 873 Kilometro 195
Barrio Tortugo
San Juan, PR 00926

Hogar Crea Venezuela
Calle Guadacanal Final
Barrio Venezuela
San Juan, PR 00926

Hogar Crea Villa Palmeras
Calle Tapia 453
San Juan, PR 00915

Remanso De Paz Inc
Carretera 842 Km 4.2
Camino Pablo Diaz Barrio Caimito Alto
San Juan, PR 00926

Residencial Mujeres Adultas San Juan
Pabellon B. Calle Maga Centro Medico
San Juan, PR 00935

Rio Piedras Psychiatric Hospital Rio Piedras Alcoholism Program
Building G Centro Medico
San Juan, PR 00925

Veterans' Affairs Medical Center Drug Dependence Treatment Program
San Juan, PR 00927

SAN LORENZO

Hogar Crea San Lorenzo
Carretera 181 Kilometro 30.6
Barrio Quebrada
San Lorenzo, PR 00754

SAN SEBSTIAN

Hogar Crea San Sebastian
Carretera 448 Kilometro 18
Barrio Guajataca
San Sebastian

SANTA ISABEL

Hogar Crea Santa Isabel
Carretera 1 Kilometro 107
Barrio Jaucal
Santa Isabel, PR 00757

SAINT JUST

Hogar Crea Inc Posada De La Esperanza Centro De Madres Con Ninos
Carretera 848 Km 09
Esq Calle Urano Urbanizacion Wonderville
Saint Just, PR 00978

TOA ALTA

Hogar Crea Toa Alta
Barrio Galateo Centro
Carretera 804 Kilometro 17
Toa Alta, PR 00953

Hogar Posada la Victoria, Inc.
C/Principal 165 KM 4 Hect 9 Parcela
52 Barrio Galateo Hoyo
Toa Alta, PR 00953

TRUJILLO ALTO

Hogar Crea Damas El Conquistador
Carretera 175 Kilometro 90
Barrio Carraizo
Trujillo Alto, PR 00976

Hogar Crea Damas Central
Carretera 848 Kilometro 10
Avenida Saint Just
Trujillo Alto, PR 00976

Hogar Crea La Quinta Carlos Quevedo Estrada
Carretera 848 Kilometro 13
Barrio Saint Just
Trujillo Alto, PR 00976

Hogar Crea Ninas Adolescentes Hogar Crea Central Damas
Carretera 848 Kilometro 07 Saint Just
Trujillo Alto, PR 00976

Hogar Crea Trujillo Pueblo
Carretera 175 Kilometro 133
Trujillo Alto, PR 00976

VEGA ALTA

Hogar Crea Modulo Vega Alta
Kilometro 8
Barrio Sabana Hoyo
Vega Alta, PR 00692

Hogar Crea Vega Alta
Carretera 159 Kilometro .05
Vega Alta, PR 00692

VEGA BAJA

Hogar Crea Vega Baja
Carr 686 Km 37
Barrio Cabo Caribe
Vega Baja, PR 00693

Hogar El Camino Barrio Puguado Afuera
Carr 155 Km 61.5 Izquierda
Carr 673
Sector El Palmar
Vega Baja, PR 00693

Silo Mision Cristiana Inc
Carretera 2 Kilometro 426
Barrio Algarrobo
Vega Baja, PR 00693

YABUCOA

Hogar Crea Yabucoa
Kilometro 15
Barrio Aguacate Carretera 906
Yabucoa, PR 00767

YAUCO

Hogar Crea Yauco
Barrio Jacanas Carretera
127 Kilometro 1 Hectometro 5
Yauco, PR 00698

REPUBLIC OF PALAU

PALAU

Ministry of Health
Palau, PW 96940-0504

VIRGIN ISLANDS

CHRISTIANSTED

Village/Virgin Islands Partners in Recovery
1 Sion Hill
Christiansted, VI 00823

SAINT CROIX

Mental Health Alcoholism and Drug Dependency Services
3500 Richmond Christiansted Street
Saint Croix, VI 00820

SAINT THOMAS

Council on Alcoholism and Drug Dependence
4B-5-6A Norre Gade Street
Saint Thomas, VI 00802

APPENDIX IV

Bureau of Justice Statistics

INTRODUCTION

The Bureau of Justice Statistics is an agency of the U.S. Department of Justice, Washington, DC. In a 1993 report entitled *Drugs, Crime, and the Justice System,* the bureau presented an overview of how the U.S. justice system attempts to combat illegal drugs.

Many areas of society are included in the overview. Here we present summarized data in easy to review format, with new, post–1990 information provided by Mark Kleiman and Thai Ishizuka-Capp, both from the Drug Policy Analysis Program, School of Public Policy and Social Research, University of California, Los Angeles. Much of the information offered here is fully discussed throughout the alphabetical entries of the encyclopedia—in Volumes 1, 2, and 3. Consult the Index at the end of this volume for references to items of further interest.

POLICIES, STRATEGIES, AND TACTICS USED TO CONTROL THE ILLEGAL DRUG PROBLEM

POLICIES

Prohibition is the ban on the distribution, possession, and use of specified substances made illegal by legislative or administrative order and the application of criminal penalties to violators.

Regulation is control over the distribution, possession, and use of specified substances. Regulations specify the circumstances under which substances can be legally distributed and used. Prescription medications and alcohol are the substances most commonly regulated in the U.S.

STRATEGIES

Demand reduction strategies attempt to decrease individuals' tendency to use drugs. Efforts provide information and education to potential and casual users about the risks and adverse consequences of drug use, and treatment to drug users who have developed problems from using drugs.

Supply reduction focuses diplomatic, law enforcement, military, and other resources on eliminating or reducing the supply of drugs. Efforts focus on foreign countries, smuggling routes outside the country, border interdiction, and distribution within the U.S.

User accountability emphasizes that all users of illegal substances, regardless of the type of drug they use or the frequency of that use, are violating criminal laws and should be subject to penalties. It is closely associated with zero tolerance.

Zero tolerance holds that drug distributors, buyers, and users should be held fully accountable for their offenses under the law. This is an alternative to policies that focus only on some violators such as sellers of drugs or users of cocaine and heroin while ignoring other violators.

TACTICS

Criminal justice activities include enforcement, prosecution, and sentencing activities to apprehend, convict, and punish drug offenders. Although thought of primarily as having supply reduction goals, criminal sanctions also have demand reduction effects by discouraging drug use.

Prevention activities are educational efforts to inform potential drug users about the health, legal, and other risks associated with drug use. Their goal is to limit the number of new drug users and dissuade casual users from continuing drug use as part of a demand reduction strategy.

Taxation requires those who produce, distribute, or possess drugs to pay a fee based on the volume or value of the drugs. Failure to pay subjects violators to penalties for this violation, not for the drug activities themselves.

Testing individuals for the presence of drugs is a tool in drug control that is used for safety and monitoring purposes and as an adjunct to therapeutic interventions. It is in widespread use for employees in certain jobs such as those in the transportation industry and criminal justice agencies. New arrestees and convicted offenders may be tested. Individuals in treatment are often tested to monitor their progress and provide them an incentive to remain drug free.

Treatment (therapeutic interventions) focus on individuals whose drug use has caused medical, psychological, economic, and social problems for them. The interventions may include medication, counseling, and other support services delivered in an inpatient setting or on an outpatient basis. These are demand reduction activities to eliminate or reduce individuals' drug use.

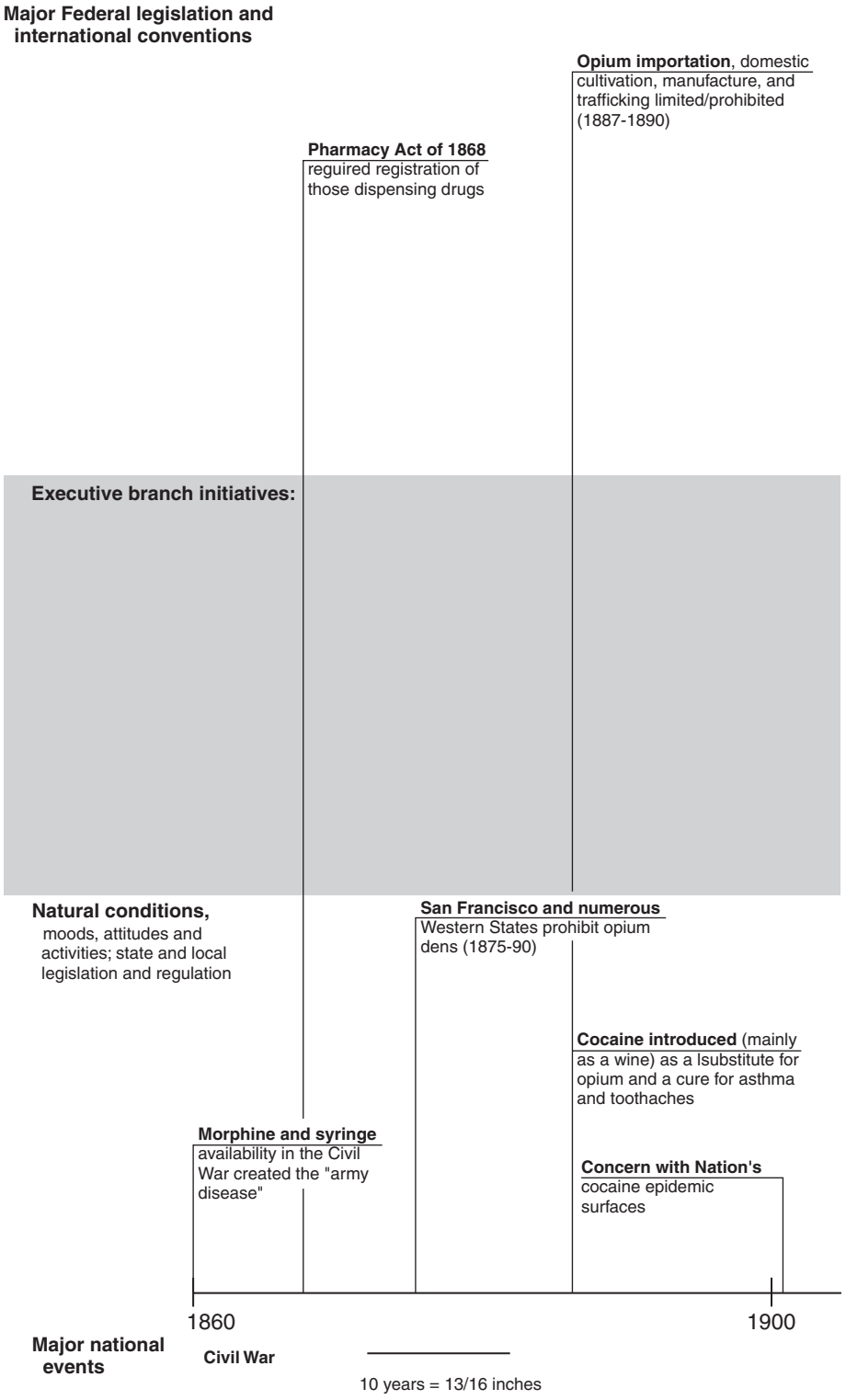
HISTORIC MILESTONES IN EARLY U.S. DRUG CONTROL EFFORTS

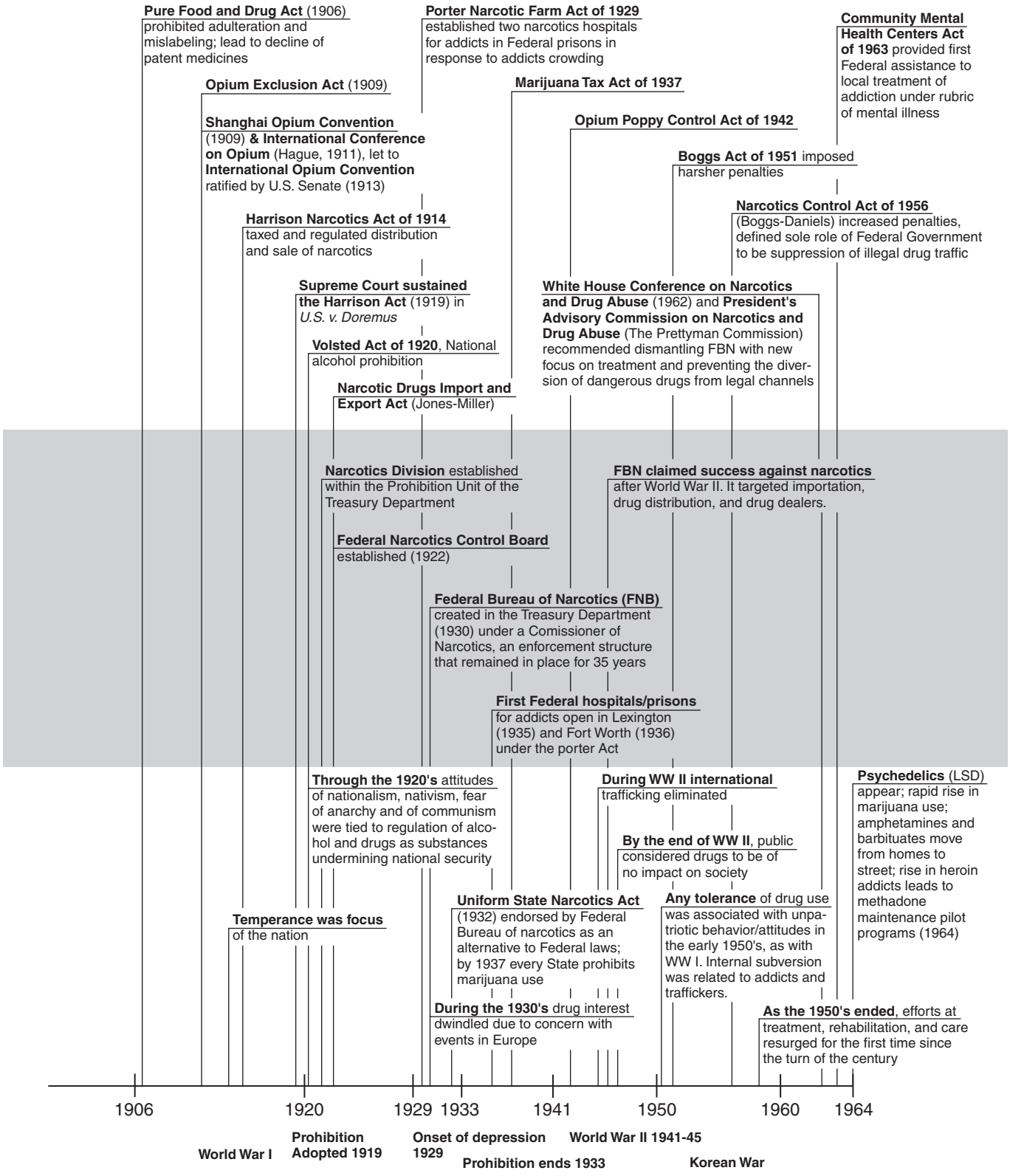
Drugs of abuse have changed since the 1800s—most rapidly over the past quarter century. Problems with opiate addiction date from widespread use of patent medicines in the 1800s. The range of drugs included opium, morphine, laudanum, cocaine, and, by the turn of the century, heroin. The tonics, nostrums, and alleged cures that contained or used such drugs were sold by itinerant peddlers, mail order houses, retail grocers, and pharmacists. There also was unrestricted access to opium in opium-smoking dens and to morphine through retailers.

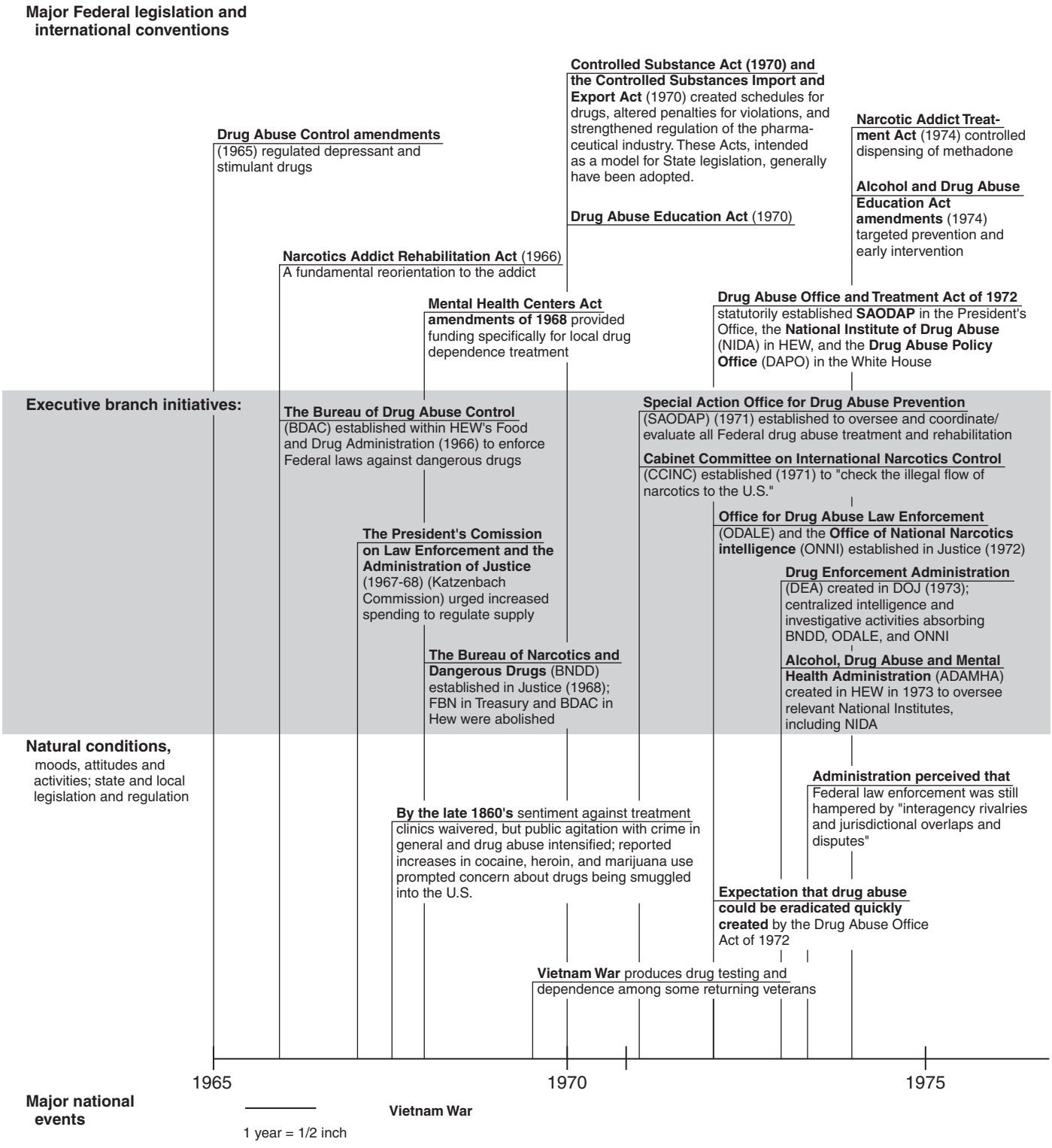
When morphine was discovered in 1806, it was thought to be a wonder drug. Its use was so extensive during the Civil War that morphine addiction was termed the "army disease." The availability of the hypodermic syringe allowed nonmedicinal use of morphine to gain popularity among veterans and other civilians. After 1898, heroin was used to treat respiratory illness and morphine addiction in the belief that it was nonaddicting.

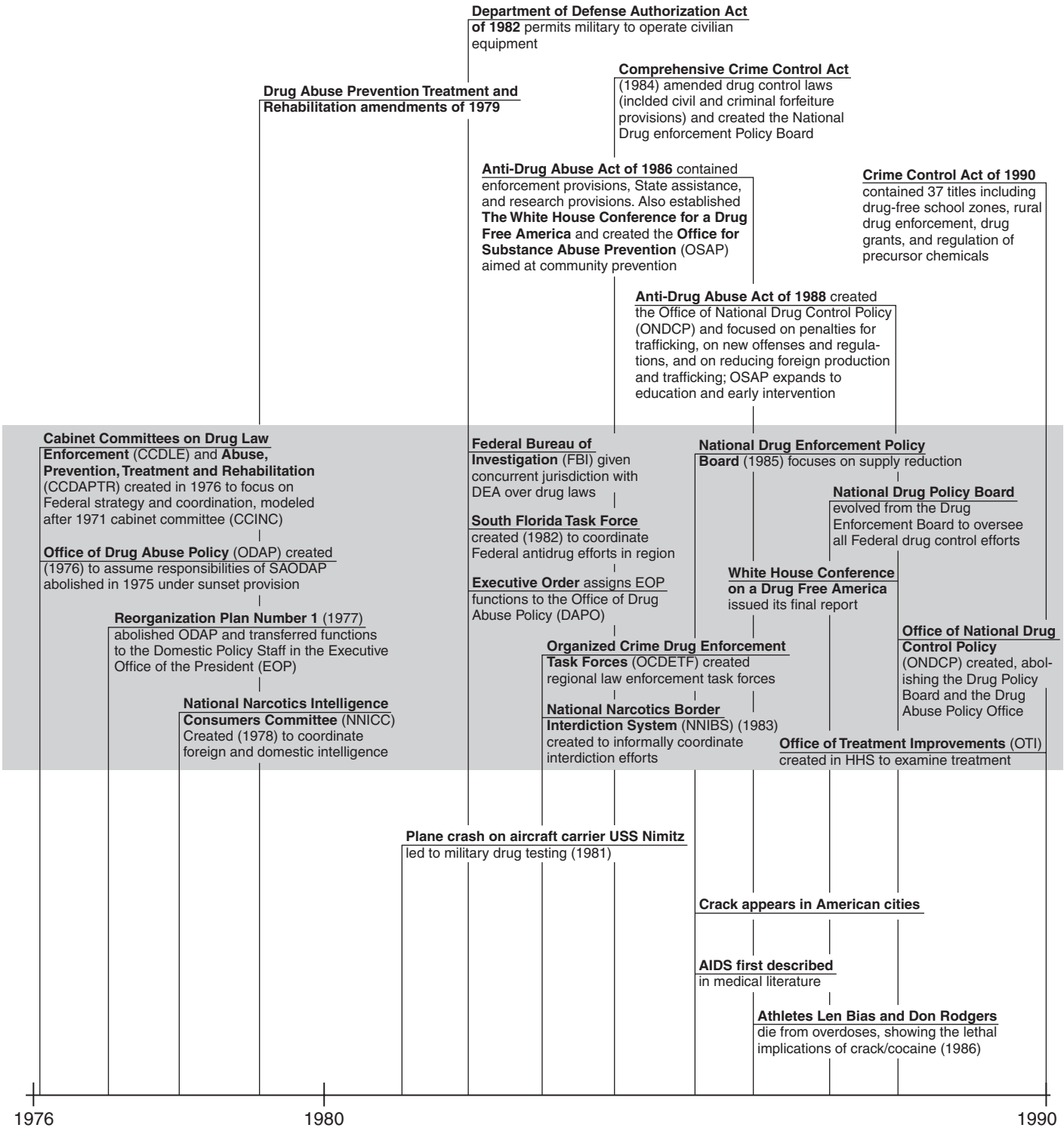
In the 1880s coca became widely available in the U.S. as a health tonic and remedy for many ills. Its use was supported first by the European medical community and later by American medical authorities. In the absence of restrictive national legislation, its use spread. Initially cocaine was offered as a cure for opiate addiction, an asthma remedy (the official remedy of the American Hay Fever Association), and an antidote for toothaches.

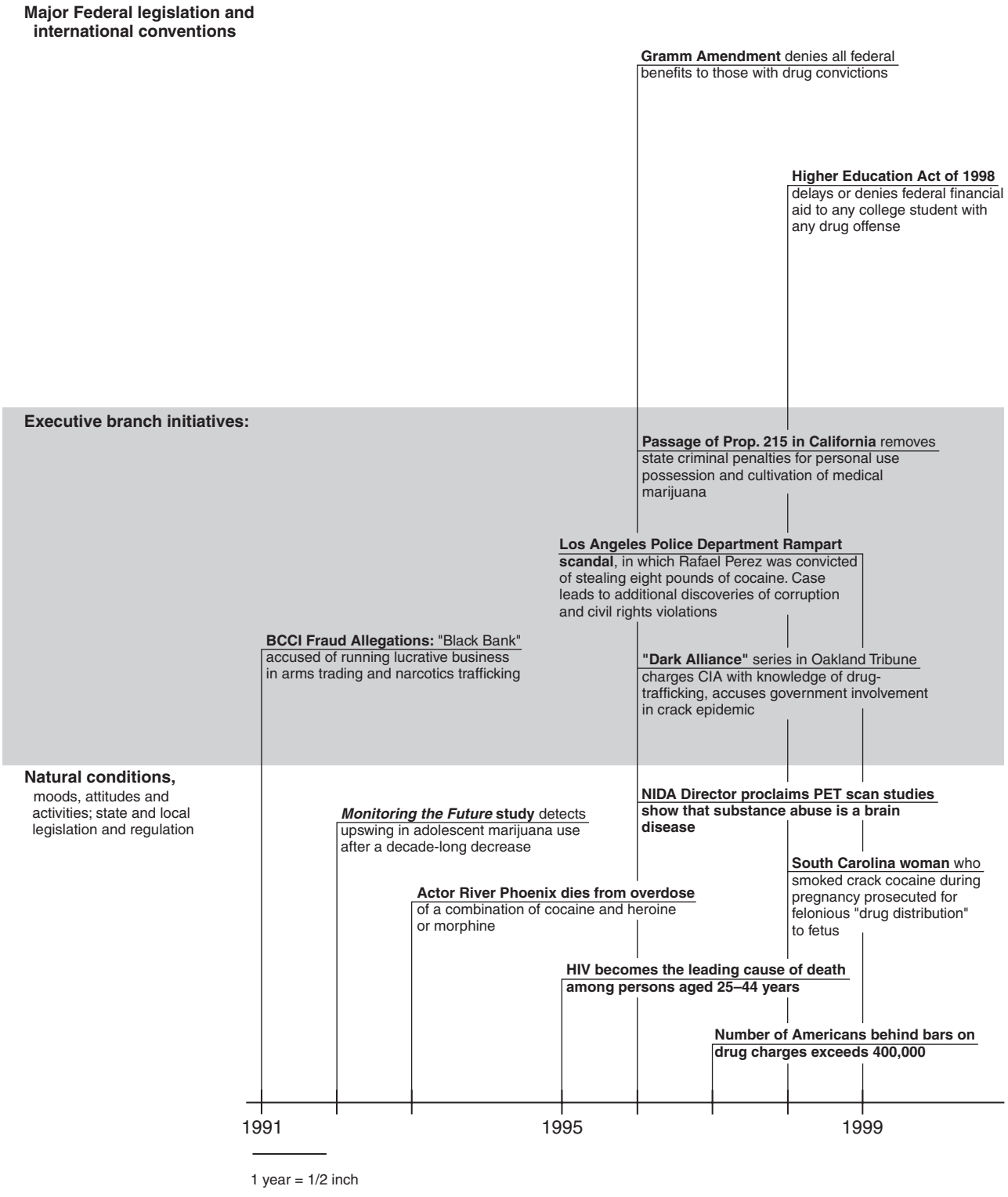
By 1900, in the face of an estimated quarter of a million addicts, State laws were enacted to curb drug addiction. The major drugs of abuse at the time were cocaine and morphine.




Pure Food and Drug Act (1906) prohibited adulteration and mislabeling; lead to decline of patent medicines
Opium Exclusion Act (1909)
Shanghai Opium Convention (1909) **& International Conference on Opium** (Hague, 1911), let to **International Opium Convention** ratified by U.S. Senate (1913)
Harrison Narcotics Act of 1914 taxed and regulated distribution and sale of narcotics
Supreme Court sustained the Harrison Act (1919) in *U.S. v. Doremus*
Volsted Act of 1920, National alcohol prohibition
Narcotic Drugs Import and Export Act (Jones-Miller)
Narcotics Division established within the Prohibition Unit of the Treasury Department
Federal Narcotics Control Board established (1922)
Federal Bureau of Narcotics (FNB) created in the Treasury Department (1930) under a Comissioner of Narcotics, an enforcement structure that remained in place for 35 years
First Federal hospitals/prisons for addicts open in Lexington (1935) and Fort Worth (1936) under the porter Act
Through the 1920's attitudes of nationalism, nativism, fear of anarchy and of communism were tied to regulation of alcohol and drugs as substances undermining national security
Temperance was focus of the nation
Uniform State Narcotics Act (1932) endorsed by Federal Bureau of narcotics as an alternative to Federal laws; by 1937 every State prohibits marijuana use
During the 1930's drug interest dwindled due to concern with events in Europe
Porter Narcotic Farm Act of 1929 established two narcotics hospitals for addicts in Federal prisons in response to addicts crowding
Marijuana Tax Act of 1937
Opium Poppy Control Act of 1942
White House Conference on Narcotics and Drug Abuse (1962) and **President's Advisory Commission on Narcotics and Drug Abuse** (The Prettyman Commission) recommended dismantling FBN with new focus on treatment and preventing the diversion of dangerous drugs from legal channels
FBN claimed success against narcotics after World War II. It targeted importation, drug distribution, and drug dealers.
During WW II international trafficking eliminated
By the end of WW II, public considered drugs to be of no impact on society
Any tolerance of drug use was associated with unpatriotic behavior/attitudes in the early 1950's, as with WW I. Internal subversion was related to addicts and traffickers.
Community Mental Health Centers Act of 1963 provided first Federal assistance to local treatment of addiction under rubric of mental illness
Boggs Act of 1951 imposed harsher penalties
Narcotics Control Act of 1956 (Boggs-Daniels) increased penalties, defined sole role of Federal Government to be suppression of illegal drug traffic
Psychedelics (LSD) appear; rapid rise in marijuana use; amphetamines and barbituates move from homes to street; rise in heroin addicts leads to methadone maintenance pilot programs (1964)
As the 1950's ended, efforts at treatment, rehabilitation, and care resurged for the first time since the turn of the century
1906
1920
1929
1933
1941
1950
1960
1964
World War I
Prohibition Adopted 1919
Onset of depression 1929
Prohibition ends 1933
World War II 1941-45
Korean War


Major Federal legislation and international conventions
Drug Abuse Control amendments (1965) regulated depressant and stimulant drugs
Controlled Substance Act (1970) and the Controlled Substances Import and Export Act (1970) created schedules for drugs, altered penalties for violations, and strengthened regulation of the pharmaceutical industry. These Acts, intended as a model for State legislation, generally have been adopted.
Drug Abuse Education Act (1970)
Narcotic Addict Treatment Act (1974) controlled dispensing of methadone
Alcohol and Drug Abuse Education Act amendments (1974) targeted prevention and early intervention
Narcotics Addict Rehabilitation Act (1966) A fundamental reorientation to the addict
Mental Health Centers Act amendments of 1968 provided funding specifically for local drug dependence treatment
Drug Abuse Office and Treatment Act of 1972 statutorily established SAODAP in the President's Office, the National Institute of Drug Abuse (NIDA) in HEW, and the Drug Abuse Policy Office (DAPO) in the White House
Executive branch initiatives:
The Bureau of Drug Abuse Control (BDAC) established within HEW's Food and Drug Administration (1966) to enforce Federal laws against dangerous drugs
Special Action Office for Drug Abuse Prevention (SAODAP) (1971) established to oversee and coordinate/evaluate all Federal drug abuse treatment and rehabilitation
Cabinet Committee on International Narcotics Control (CCINC) established (1971) to "check the illegal flow of narcotics to the U.S."
Office for Drug Abuse Law Enforcement (ODALE) and the Office of National Narcotics intelligence (ONNI) established in Justice (1972)
The President's Comission on Law Enforcement and the Administration of Justice (1967-68) (Katzenbach Commission) urged increased spending to regulate supply
Drug Enforcement Administration (DEA) created in DOJ (1973); centralized intelligence and investigative activities absorbing BNDD, ODALE, and ONNI
The Bureau of Narcotics and Dangerous Drugs (BNDD) established in Justice (1968); FBN in Treasury and BDAC in Hew were abolished
Alcohol, Drug Abuse and Mental Health Administration (ADAMHA) created in HEW in 1973 to oversee relevant National Institutes, including NIDA
Natural conditions, moods, attitudes and activities; state and local legislation and regulation
Administration perceived that Federal law enforcement was still hampered by "interagency rivalries and jurisdictional overlaps and disputes"
By the late 1860's sentiment against treatment clinics waivered, but public agitation with crime in general and drug abuse intensified; reported increases in cocaine, heroin, and marijuana use prompted concern about drugs being smuggled into the U.S.
Expectation that drug abuse could be eradicated quickly created by the Drug Abuse Office Act of 1972
Vietnam War produces drug testing and dependence among some returning veterans
1965
1970
1975
Major national events
Vietnam War
1 year = 1/2 inch


Department of Defense Authorization Act of 1982 permits military to operate civilian equipment
Comprehensive Crime Control Act (1984) amended drug control laws (incldes civil and criminal forfeiture provisions) and created the National Drug enforcement Policy Board
Drug Abuse Prevention Treatment and Rehabilitation amendments of 1979
Anti-Drug Abuse Act of 1986 contained enforcement provisions, State assistance, and research provisions. Also established **The White House Conference for a Drug Free America** and created the **Office for Substance Abuse Prevention** (OSAP) aimed at community prevention
Crime Control Act of 1990 contained 37 titles including drug-free school zones, rural drug enforcement, drug grants, and regulation of precursor chemicals
Anti-Drug Abuse Act of 1988 created the Office of National Drug Control Policy (ONDCP) and focused on penalties for trafficking, on new offenses and regulations, and on reducing foreign production and trafficking; OSAP expands to education and early intervention
Cabinet Committees on Drug Law Enforcement (CCDLE) and **Abuse, Prevention, Treatment and Rehabilitation** (CCDAPTR) created in 1976 to focus on Federal strategy and coordination, modeled after 1971 cabinet committee (CCINC)
Federal Bureau of Investigation (FBI) given concurrent jurisdiction with DEA over drug laws
National Drug Enforcement Policy Board (1985) focuses on supply reduction
National Drug Policy Board evolved from the Drug Enforcement Board to oversee all Federal drug control efforts
South Florida Task Force created (1982) to coordinate Federal antidrug efforts in region
Office of Drug Abuse Policy (ODAP) created (1976) to assume responsibilities of SAODAP abolished in 1975 under sunset provision
Executive Order assigns EOP functions to the Office of Drug Abuse Policy (DAPO)
White House Conference on a Drug Free America issued its final report
Reorganization Plan Number 1 (1977) abolished ODAP and transferred functions to the Domestic Policy Staff in the Executive Office of the President (EOP)
Organized Crime Drug Enforcement Task Forces (OCDETF) created regional law enforcement task forces
Office of National Drug Control Policy (ONDCP) created, abolishing the Drug Policy Board and the Drug Abuse Policy Office
National Narcotics Intelligence Consumers Committee (NNICC) Created (1978) to coordinate foreign and domestic intelligence
National Narcotics Border Interdiction System (NNIBS) (1983) created to informally coordinate interdiction efforts
Office of Treatment Improvements (OTI) created in HHS to examine treatment
Plane crash on aircraft carrier USS Nimitz led to military drug testing (1981)
Crack appears in American cities
AIDS first described in medical literature
Athletes Len Bias and Don Rodgers die from overdoses, showing the lethal implications of crack/cocaine (1986)
1976
1980
1990


Major Federal legislation and international conventions
Gramm Amendment denies all federal benefits to those with drug convictions
Higher Education Act of 1998 delays or denies federal financial aid to any college student with any drug offense
Executive branch initiatives:
Passage of Prop. 215 in California removes state criminal penalties for personal use possession and cultivation of medical marijuana
Los Angeles Police Department Rampart scandal, in which Rafael Perez was convicted of stealing eight pounds of cocaine. Case leads to additional discoveries of corruption and civil rights violations
BCCI Fraud Allegations: "Black Bank" accused of running lucrative business in arms trading and narcotics trafficking
"Dark Alliance" series in Oakland Tribune charges CIA with knowledge of drug-trafficking, accuses government involvement in crack epidemic
Natural conditions, moods, attitudes and activities; state and local legislation and regulation
NIDA Director proclaims PET scan studies show that substance abuse is a brain disease
Monitoring the Future study detects upswing in adolescent marijuana use after a decade-long decrease
South Carolina woman who smoked crack cocaine during pregnancy prosecuted for felonious "drug distribution" to fetus
Actor River Phoenix dies from overdose of a combination of cocaine and heroine or morphine
HIV becomes the leading cause of death among persons aged 25–44 years
Number of Americans behind bars on drug charges exceeds 400,000
1991
1995
1999
1 year = 1/2 inch

MAJOR FEDERAL ANTIDRUG BILLS, ENACTED 1984–2000

The 1984 Crime Control Act—

- expanded criminal and civil asset forfeiture laws
- amended the Bail Reform Act to target pretrial detention of defendants accused of serious drug offenses
- established a determinate sentencing system
- increased Federal criminal penalties for drug offenses

The 1986 Anti-Drug Abuse Act—

- budgeted money for prevention and treatment programs, giving the programs a larger share of Federal drug control funds than previous laws
- restored mandatory prison sentences for large-scale distribution of marijuana
- imposed new sanctions on money laundering
- added controlled substances' analogs (designer drugs) to the drug schedule
- created a drug law enforcement grant program to assist State and local efforts
- contained various provisions designed to strengthen international drug control efforts.

The 1988 Anti-Drug Abuse Act—

- increased penalties for offenses related to drug trafficking, created new Federal offenses and regulatory requirements, and changed criminal procedures
- altered the organization and coordination of Federal antidrug efforts
- increased treatment and prevention efforts aimed at reduction of drug demand
- endorsed the use of sanctions aimed at drug users to reduce the demand for drugs
- targeted for reduction drug production abroad and international trafficking in drugs

The Crime Control Act of 1990—

- doubled the appropriations authorized for drug law enforcement grants to States and localities
- expanded drug control and education programs aimed at the Nation's schools
- expanded specific drug enforcement assistance to rural States
- expanded regulation of precursor chemicals used in the manufacture of illegal drugs
- provided additional measures aimed at seizure and forfeiture of drug trafficker assets
- sanctioned anabolic steroids under the Controlled Substances Act
- included provisions on international money laundering, rural drug enforcement, drug-free school zones, drug paraphernalia, and drug enforcement grants.

"Smoke a Joint, Lose your License" Bill (passed as Public Law 101-516) of 1990—

- required that each State must either: enact laws that mandate suspending or revoking for six months the driver's license of any person convicted of controlled substance violations; or pass a resolution in both houses of the State legislature, accompanied by a written certification from the Governor acknowledging his agreement, that the State does not wish to enact the law
- failure to do one of these results in a 10% loss of the State's federal highway funds.

Gramm Amendment (Senate Amendment 4935 to the Welfare Reform package) in 1996—

- denied for life Federal assistance-based cash aid and food stamps to anyone convicted of felony drug charges
- applied to future felony drug convictions, and States have the ability to opt out of the program if they enact legislation to do so

Higher Education Act of 1998—

- delayed or denied Federal financial aid eligibility to any individual convicted of a State or Federal drug offense
- established that drug possession convictions result in ineligibility for one year (first offense), two years (second offense), or indefinitely (third offense), and that drug sale convictions result in ineligibility for two years (first offense) or indefinitely (second offense)
- provided that students may receive early restoration of benefits by completing a treatment program that fulfills yet-to-be-announced Dept. of Education regulations
- specified that ineligibility applies to all forms of Federal financial aid, including grants, student loans, and work-study

Civil Asset Forfeiture Reform Act of 1999 (effective August 23, 2000)—

- established that in order to seize assets, the government must prove that property is related to a crime, as opposed to property owners' having to prove that their property is "innocent"
- created an "innocent owner defense," whereby property owners who are either unaware of or unsuccessfully try to stop criminal activity on their property can recover the property

- eliminated the cost-bond requirement, which previously required property owners to pay $5,000 or 10 percent of the seized property's value to contest seizure in court
- provided compensation for property damage caused by federal agents and extended the time for filing a claim to contest a forfeiture

APPENDIX V

Illicit and Licit Drugs of Abuse—Schedules of Controlled Substances

INTRODUCTION

U.S. legislation called the Controlled Substances Act of 1970 has ranked and categorized drugs according to their effects, medical use, and potential for abuse. Ongoing research may reclassify drugs from one category to another, as has happened in the past.

At the federal level. Schedule I is the most strictly controlled—with the highest abuse potential; Schedule V is the least strictly controlled—drugs sold with or without prescription by mail and in shops, with instructions for use, dosages, and warnings about effects and side effects printed on the packaging of over-the-counter (OTC) medications. The schedules shown below in simplified form are followed by extensive schedules (which are discussed fully in Volume 1, in the article entitled Controls: Scheduled Drugs/Drug Schedules, U.S.). A discussion of the Controlled Substances Act of 1970 precedes it.

Drugs are scheduled under federal law according to their effects, medical use, and potential for abuse

DEA Schedule	*Abuse Potential*	*Examples of Drugs Covered*	*Some of the Effects*	*Medical Use*
I	highest	heroin, LSD, hashish, marijuana, methaqualone	unpredictable effects, severe psychological or physical dependence, or death	no accepted use; some are legal for limited research use only
II	high	morphine, PCP, cocaine, methadone, methamphetamine	may lead to severe psychological or physical dependence	accepted use with restrictions
III	medium	codeine with aspirin or Tylenol®, some barbiturates, anabolic steroids	may lead to moderate or low physical dependence or high psychological dependence	accepted use
IV	low	Darvon®, Talwin®, Equanil®, Valium®, Xanax®	may lead to limited physical or psychological dependence	accepted use
V	lowest	over-the-counter or prescription cough medicines with codeine	may lead to limited physical or psychological dependence	accepted use

SOURCE: Adapted from Drug Enforcement Administration, *Drugs of abuse (1996) and Schedules of Controlled Substances*, Revised as of April 1, 1998.

SCHEDULES OF U.S. CONTROLLED DRUGS

CRITERIA FOR U.S. DRUG SCHEDULING

Schedule	*Potential for:* *Abuse*	*Dependence*	*Medical Use & Safety*
I	+ + + +	+ + + +	No
II	+ + + +	+ + + +	Yes
III	+ + +	+ + +	Yes
IV	+ +	+ +	Yes
V	+	+	Yes

LIST OF CONTROLLED DRUGS

SCHEDULE I

Opiates

Accty-alpha-methylfentanyl
Acetylmethadol
Allylprodine
Alphameprodine
Alphamethadol
Alpha-methylfentanyl
Alpha-methylthiofentanyl
Benzethidine
Betacetylmethadol
Beta-hydroxyfentanyl
Beta-hydroxy-3-methylfentanyl
Betameprodine
Betamethadol
Betaprodine
Clonitazene
Dextromoramide
Diampromide
Diethylthiambutene
Difenoxin
Dimenoxadol
Dimepheptanol
Dimethylthiambutene
Dioxaphetyl butyrate
Dipipanone
Ethylmethylthiambutene
Etonitazene
Etoxeridine
Furethidine
Hydroxypethidine
Ketobemidone
Levomoramide
Levophenacylmorphan
3-methylfentanyl
3-methylthiofentanyl
Morpheridine
MPPP
Noracymethadol
Norlevorphanol
Normethadone
Norpipanone
Para-fluorofentanyl
PEPAP
Phenadoxone
Phenampromide
Phenomorphan
Phenoperidine
Piritramide
Proheptazine
Properidine
Propiram
Racemoramide
Thiofentanyl
Tilidine
Trimeperidine

Opium Derivatives

Acetorphine
Acetyldihydrocodeine
Benzylmorphine
Codeine methylbromide
Codeine-N-Oxide
Cyprenorphine
Desomorphine
Dihydropmorphine
Drotebanol
Etorphine (except HCI salt)
Heroin
Hydromorphinol
Methyldesorphine
Methyldihydromorphine
Morphine methylbromide
Morphine methylsulfonate
Morphine-N-Oxide
Myrophine
Nicocodeine
Nicomorphine
Normorphine
Pholcodine
Thebacon

Hallucinogens

Alpha-ethyltryptamine
4-bromo-2.5-DMA
Alpha-desmethyl DOB
2.5-DMA
DOET
PMA
5-methoxy-3,4-mdthylene-dioxyamphetamine
MMDA
DOM, STP
MDA
MDMA
MDEA
N-hydroxy MDA
3,4,5-trimethoxy amphetamine
Bufotenine
DET
DMT
Ibogaine
LSD
Marihuana
Mescaline
N-ethyl-3-peperidyl benzilate
N-methyl-3-piperidyl-benzilate
Peyote
Pheneyelidine analogs PCE, PCPy, TCP, TCPy
Psilocybin
Psilocyn
Tetrahydrocannabinols

Depressants

Mecloqualone
Methaqualone

Stimulants

Aminorex Cathinone
Fenethylline
Methcathinone
(±) cis-4-methylam-inorex
N-ethylamphetamine
N,N-dimethyl-am-phetamine

Temporary listing of substances subject to emergency scheduling:
Benzlyfentanyl
Thenylfentanyl

LIST OF CONTROLLED DRUGS

SCHEDULE II

Opiates	Opium & Derivatives	Hallucinogens	Depressants	Stimulants	Others
Alfentanil	Raw opium	Dronabinol	Amobarbital	Amphetamine	Opium poppy
Alphaprodine	Opium extracts	Nabilone	Glutethimide	Methamphetamine	Poppy straw
Anileridine	Opium fluid		Pentobarbital	Phenmetrazine	Coca leaves
Bezitramide	Powdered opium		Phencyclidine	Methylphenidate	Immediate precursors to:
Bulk dextropropophene	Granulated opium		Secobarbital		Amphetamine
Carfentanil	Tincture of opium				Methamphetamine
Dihydrocodeine	Codeine				Phencyclidine
Diphenoxylate	Ethylmorphine				
Fentanyl	Etorphine hydrochloride				
Isomethadone	Hydrodone				
Levo-alphacetylmethadol	Hydromorphone				
Levomethorphan	Metopon				
Levorphanol	Morphine				
Metazocine	Oxycodone				
Methadone	Oxymorphone				
Methadone-Intermediate	Thebaine				
Moramide-Intermediate					
Pethidine					
Pethidine-Intermediate-A					
Pethidine-Intermediate-B					
Pethidine-Intermediate-C					
Phenazocine					
Piminodine					
Racemethorphan					
Racemorphan					
Remifentanil					
Sufentanil					

LIST OF CONTROLLED DRUGS

SCHEDULE III

Narcotics	Depressants	Stimulants	Others
Limited quantities of:	Mixtures of	Limited mixtures	Nalorphine
Codeine	Amobarbital	of Schedule II	All anabolic steroids
Dihydrocodeinone,	Secobarbital	amphetamines	
Dihydrocodeine,	Pentobarbital	Benzphetamine	
Ethylmorphine,	Derivatives of	Chlorphentermine	
Opium, and	barbituric acid	Clortermine	
Morphine	Chlorhexadol	Phendimetrazine	
in combination	Lysergic acid		
with nonnarcotics.	Lysergic acid amide		
	Methyprylon		
	Sulfondiethylmethane		
	Sulfonethylmethane		
	Sulfonmethane		
	Tiletamine		
	Zolazepam		

LIST OF CONTROLLED DRUGS

SCHEDULE IV

Narcotics	Depressants		Stimulants	Others
Limited quantity of difenoxin in combination with atropine sulfate	Alprazolam	Loprasolam	Cathine	Butorphanol
	Barbital	Lorazepam	Diethylpropion	Fenfluramine
	Bromazepam	Lormetazepam	Fencamfamin	Pentazocine
Dextropropoxyphere	Camazepam	Mebutamate	Fenproporex	
	Chloral betaine	Medazepam	Mazindol	
	Chloral hydrate	Meprobamate	Mefenorex	
	Chlordiazepoxide	Methohexital	Pemoline	
	Clobazam	Methylphenobarbital	Phentermine	
	Clorazepate	Nimetazepam	Pipradrol	
	Clotiazepam	Nitrazepam	Sibutramine	
	Cloxazolam	Nordiazepam	SPA	
	Delorazepam	Oxazepam		
	Diazepam	Oxazolam		
	Estazolam	Paraldehyde		
	Ethchlorvynol	Petrichloral		
	Ethinamate	Phenobarbital		
	Ethyl loflazepate	Pinazepam		
	Fludiazepam	Prazepam		
	Flunitrazepam	Quazepam		
	Flurazepam	Temazepam		
	Halazepam	Tetrazepam		
	Haloxazolam	Triazolam		
	Ketazolam	Zolpidem		

LIST OF CONTROLLED DRUGS

SCHEDULE V

Narcotics	Stimulants
Buprenorphine	Pyrovalcrone
Limited quantities (less than Schedules III & IV) of:	
Codeine,	
Dihydrocodeine,	
Ethylmorphine,	
Diphenoxylate, Opium, and Difenoxin in combination with nonnarcotics	

Index

Numbers in **boldface** *refer to the main entry on the subject.*
Numbers in *italic* refer to illustrations.

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